The effect of speed on gait asymmetry during walking for above- and below-knee amputees.

by

P. Lee Nolan

A thesis submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy following work carried out at the Liverpool John Moores University, School of Human Sciences, Research Institute for Sport and Exercise Sciences.

May 2001
THE EFFECT OF SPEED ON GAIT ASYMMETRY DURING WALKING FOR ABOVE- AND BELOW-KNEE AMPUTEES.

P. Lee Nolan

Abstract.

The aim of the thesis is to investigate asymmetries in selected gait characteristics in above- and below-knee amputee gait, and to investigate the effects of walking speed on these gait characteristics and asymmetries. The characteristics investigated included limb loading, stance, step and swing times, impact shock, shock attenuation, hip, knee and ankle joint angles, joint moments and power output and EMG signals from the hamstrings, quadriceps and triceps surae.

With regard to asymmetries, the amputees were seen to exhibit reduced vertical ground reaction force (vGRF) loading on their prosthetic limb, spent longer stepping onto their prosthetic limb, had a reduced joint angle ROM, reduced net joint moments, power outputs and exhibited lower muscle activity on their residual/ prosthetic limb compared to their intact limb. The above-knee amputees were more asymmetrical than the below-knee amputees who were more asymmetrical than the able-bodied subjects. Although it was not possible to measure EMG activity in the residual limb of above-knee amputees, for the below-knee amputees, the results of the cross-correlations show that the intact limb functions more like that of an able-bodied person than the amputees' residual limb. Thus, the residual limb is affected by the limitations of the prosthesis.

With regard to walking speed, the vGRF, joint moments, joint power outputs and muscle activity all increased with walking speed on both the intact and prosthetic limb of the amputees, whilst all the temporal variables decreased with walking speed. For most variables, the increase was greater on the intact limb than prosthetic limb. Thus, amputee gait asymmetry was found to increase with walking speed for many variables. However, few of these variables were found to significantly increase possibly because of the low subject numbers in the studies (n=4 or n=5). Temporal asymmetry was the only variable found to decrease with increasing walking speed. This may be the mechanism in which the amputees attempt to achieve dynamic balance and co-ordination in order to attain the faster walking speeds. It appears that temporal gait asymmetry is reduced at the expense of increased loading asymmetry.

The asymmetry reflects the compensations that amputees make for their prosthesis, and this puts a greater load on the intact limb. Pain and/or joint degeneration in the intact limb is a common problem for lower limb unilateral amputees. It has previously been suggested in the literature that joint pain and/or joint degeneration is due to repeated cyclic loading. Thus increased loading on the intact limb has led amputees to report intact limb pain, and in these participants there was evidence of reduced shock attenuation capacity. This provides evidence of intact limb degeneration, the result of which is reduced capacity for locomotor activity.

The research has led to a better knowledge and understanding of how amputees cope with their prosthetic limb with regard to demanding physical activity. It is agreed that this knowledge will enable prostheses to be designed which minimise gait asymmetry which in turn will reduce the loading on the intact limb and delay the onset of joint degeneration. In this way amputees may expect an improved quality of life.
Acknowledgements.

I would like to thank my supervisors Professor Adrian Lees and Dr. Mark Lake for their help and guidance throughout this thesis. I would also especially like to thank Professor Andrzej Wit at the Academy of Physical Education, Warsaw for his help and invitation to perform two of my studies in his department. Also to his colleagues Dr. Jan Gajewski, Dr. Michal Wychowanski and mgr. Krzyztof Dudzinski for their help with data collection, subject recruitment and roles as translators.

Thanks also to Craig Armstrong from Clatterbridge Hospital, Wirral for help with recruiting amputee subjects and taking anthropometric measurements from their prostheses, and Carine van Schie also for help with recruiting amputee subjects. Also to Dr. Jai Kulkarni for permission to collect data in his department at the Disablement Services Centre, Withington Hospital, Manchester. I would also like to express my gratitude to all those subjects who gave up their valuable time to take part in my studies. Without you, there would have been no thesis.

I would like to acknowledge most of all, the help of my close friends and colleagues in the last three years - for listening to my problems, disappointments and crises and especially those who were there for me when things just didn’t look like working out. To Doris who was there from the beginning, but never got to see the end. To Nada, Nick, Mel and Ash for friendships I will always treasure, Cookie, Judith, Julie, Tanya, Tanya, Penny and Kathryn just for being there and most of all Ben and Ginny for endless discussion and hours in the biomechanics lab, trying to stay sane.

Finally, I would like to thank all those who never let me give up.
This thesis is dedicated to my granddad

Edward Nolan (1901-1972)

Lives of great men all remind us
We can make our lives sublime,
And, departing, leave behind us
Footprints in the sands of time;

Footprints, that perhaps another,
Sailing o'er life's solemn main,
A forlorn and shipwrecked brother,
Seeing, shall take heart again.

Let us, then, be up and doing,
With a heart for any fate;
Still achieving, still pursuing,
Learn to labour and to wait.

Henry Wadsworth Longfellow.
# List of contents.

Abstract
Acknowledgements  
List of Contents  
List of Figures  
List of Tables  
List of Plates

1. Introduction.  

2. Aims and Objectives.  

2.1 Definition of terms.

3. Review of Literature.  

3.1 Introduction.  

3.2 Limb loading and loading asymmetry.  

3.2.1 Able-bodied gait.  
3.2.1.1 Typical ground reaction forces.  
3.2.1.2 Effects of speed, stride length and stride frequency on ground reaction forces.  
3.2.1.3 Variation and asymmetry.  
3.2.1.4 Alternative methods of vertical ground reaction force and asymmetry measurement.  
3.2.1.5 Summary of able-bodied limb loading.  
3.2.2 Loading and loading asymmetry in amputee gait.  
3.2.2.1 Lower limb amputee ground reaction forces.  
3.2.2.2 The effect of walking speed on ground reaction forces.  
3.2.2.3 The effect of prosthetic alignment on ground reaction forces.  
3.2.2.4 The effect of different prosthesis on ground reaction forces.
3.2.2.5 Variation and asymmetry in amputee gait. 24
3.2.2.6 Summary of amputee limb loading. 25
3.2.3 Summary of limb loading and loading asymmetry. 26

3.3 Impact shock and shock attenuation. 28
3.3.1 Impact shock in able-bodied gait. 29
  3.3.1.1 Heel strike transient and acceleration. 29
  3.3.1.2 Uniaxial and multiaxial accelerometry. 32
  3.3.1.3 Accelerometer placement. 32
  3.3.1.4 The effects of footwear and surfaces on impact shock. 33
  3.3.1.5 The effect of walking speed on impact shock. 34
3.3.2 Joint degeneration. 35
  3.3.2.1 Joint degeneration mechanisms. 35
  3.3.2.2 Shock attenuation in able-bodied gait. 36
  3.3.2.3 Joint pain and shock attenuation. 37
  3.3.2.4 The effect of walking speed on shock attenuation. 40
  3.3.2.5 Summary of impact shock and shock attenuation in able-bodied gait. 40

3.3.3 Impact shock and shock attenuation in unilateral amputees. 41
  3.3.3.1 Joint degeneration in amputees. 41
  3.3.3.2 Impact shock and shock attenuation in amputee gait. 42
  3.3.3.3 Adaptation to shock absorption. 44
  3.3.3.4 Summary of impact shock and shock attenuation in amputee gait. 45
3.3.4 Summary of impact shock and shock attenuation. 46

3.4 Kinematics. 48
3.4.1 Kinematics of able-bodied gait. 48
  3.4.1.1 Temporal variables of gait. 48
  3.4.1.2 The effect of speed on temporal variables of gait. 51
  3.4.1.3 Spatial variables of gait. 51
3.4.1.4 The effect of speed on spatial variables of gait. 54
3.4.1.5 Variability and asymmetry of able-bodied gait. 55
3.4.1.6 Summary of kinematics of able-bodied gait. 55

3.4.2 Kinematics of amputee gait.
3.4.2.1 Temporal variables of gait. 57
3.4.2.2 The effect of speed on temporal variables of gait. 58
3.4.2.3 Spatial variables of gait. 59
3.4.2.4 The effect of speed on spatial variables of gait. 64
3.4.2.5 Variability and asymmetry of amputee gait. 64
3.4.2.6 Summary of kinematics of amputee gait. 64

3.4.3 Summary of kinematics. 65

3.5 Joint kinetics.
3.5.1 Description of joint kinetics for able-bodied walking.
3.5.1.1 Net joint moments for able-bodied walking. 69
3.5.1.2 Errors and variability in able-bodied joint moments. 74
3.5.1.3 Power output at the hip, knee and ankle. 76
3.5.1.4 Power output in able-bodied walking gait. 76
3.5.1.5 Variability in power output for able-bodied gait. 79
3.5.1.6 The effect of speed on joint kinetics in able-bodied gait.
3.5.1.7 Summary of joint kinetics in able-bodied gait. 79

3.5.2 Description of joint kinetics for amputee walking gait.
3.5.2.1 Net joint moments of amputee walking. 80
3.5.2.2 Variability in amputee net joint moments. 84
3.5.2.3 Errors in calculating net joint moments in amputees.
3.5.2.4 Power output in amputee gait. 85
3.5.2.5 Variability in amputee joint power output. 89
3.5.1.6 Summary of amputee joint kinetics. 89

3.5.3 Summary of joint kinetics. 90

3.6 Muscle activation patterns.
3.6.1 Methodological considerations. 92
3.6.1.1 Electrodes and electrode placement. 92
3.6.1.2 Normalisation of EMG signal. 94
3.6.2 Muscle activity during able-bodied gait. 95
   3.6.2.1 Activity of muscles in the lower limb joints. 95
   3.6.2.2 The effect of speed on muscle activity during able-bodied walking. 100
   3.6.2.3 Variability and asymmetry in able-bodied walking. 101
   3.6.2.4 Summary of muscle activity in able-bodied gait. 103
3.6.3 Muscle activity in amputee gait. 104
   3.6.3.1 Stability. 104
   3.6.3.2 The effects of different prostheses on muscle activity. 109
   3.6.3.3 Differences in muscle activity between sedentary and active amputees. 112
   3.6.3.4 Summary of muscle activity in amputee gait. 112
3.6.4 Summary of muscle activity. 113

3.7 Synthesis of literature review. 115

4. The effect of speed on gait asymmetry during walking for above- and below-knee amputees. 118
   4.1 Comparison of CDG force shoes with the force platform. 118
      4.1.1 Introduction. 118
      4.1.2 Methodology. 119
         4.1.2.1 Apparatus. 119
         4.1.2.2 Subjects and procedure. 121
         4.1.2.3 Data analysis. 121
      4.1.3 Results. 123
      4.1.4 Discussion. 126
      4.1.5 Conclusion. 127
   4.2 The effect of walking speed on loading and temporal asymmetry in above- and below-knee amputees. 129
4.2.1 Introduction. 129
4.2.2 Methodology. 131
  4.2.2.1 Apparatus. 131
  4.2.2.2 Subjects and procedure. 131
  4.2.2.3 Data analysis. 132
4.2.3 Results. 135
4.2.4 Discussion. 146
4.3 Summary. 152

5. Impact shock and shock attenuation and impact shock and shock attenuation asymmetry during walking for above- and below-knee amputees. 154
  5.1 Relationship between the heel strike transient and impact acceleration in able-bodied gait. 154
    5.1.1 Introduction. 154
    5.1.2 Methodology. 156
      5.1.2.1 Apparatus. 156
      5.1.2.2 Subjects and procedure. 157
      5.1.2.3 Data analysis. 158
    5.1.3 Results. 161
    5.1.4 Discussion. 166
    5.1.5 Conclusion. 168

  5.2 The effect of speed on impact shock and shock attenuation and impact shock and shock attenuation asymmetry. 169
    5.2.1 Introduction. 169
    5.2.2 Methodology. 171
      5.2.2.1 Apparatus. 171
      5.2.2.2 Subjects and procedure. 171
      5.2.2.3 Data analysis. 173
    5.2.3 Results. 175
      5.2.3.1 Able-bodied subjects. 175
      5.2.3.2 Amputees. 181
    5.2.4 Discussion. 189
      5.2.4.1 Able-bodied subjects. 189
5.2.4.2. Amputees. 192
5.2.5 Summary. 195

6. Kinematic and kinetic asymmetry during walking for above- and below-knee amputees. 197

6.1 Variability in kinematic measurements and joint moment and joint power output calculations in able-bodied gait. 197

6.1.1 Introduction. 197
6.1.2 Methodology. 199

6.1.2.1 Apparatus. 199
6.1.2.2 Subjects and procedure. 201
6.1.2.3 Data analysis. 201

6.1.3 Results. 210
6.1.4 Discussion. 214
6.1.5 Conclusion. 217

6.2 The effect of walking speed on kinematic and kinetic asymmetry in amputee gait. 218

6.2.1 Introduction. 218
6.2.2 Methodology. 222

6.2.2.1 Apparatus. 222
6.2.2.2 Subjects and procedure. 222
6.2.2.3 Data analysis. 223

6.2.3 Results. 227
6.2.3.1 Kinematic analysis. 227
6.2.3.2 Kinetic analysis. 235
6.2.4 Discussion. 254
6.2.5 Summary. 265

7. Muscle activation asymmetry during walking for above- and below-knee amputees. 268

7.1 Normalisation techniques of the EMG signal. 268
7.1.1 Introduction. 268
7.1.2 Methodology.

7.1.2.1 Apparatus.
7.1.2.2 Subjects and procedure.
7.1.2.3 Data analysis.

7.1.3 Results.

7.1.4 Discussion.

7.1.5 Conclusion.

7.2 The effect of walking speed on muscle activation patterns in above- and below-knee amputees.

7.2.1 Introduction.

7.2.2 Methodology.

7.2.2.1 Apparatus.
7.2.2.2 Subjects and procedure.
7.2.2.3 Data analysis.

7.2.3 Results.

7.2.4 Discussion.

7.2.5 Summary.

8. Concluding remarks.


References.

Appendix.
List of Figures.

Figure 3.1  Vertical, anterior-posterior and medio-lateral forces during able-bodied walking. The shading represents variability (Meglan and Todd, 1994, pp76).

Figure 3.2  Vertical GRF of the intact and prosthetic limb of a below-knee amputee and an able-bodied person walking (Engsberg et al., 1991, pp 659).

Figure 3.3  The vertical ground reaction force. A represents the heel strike transient peak (Collins and Whittle, 1989, pp180).

Figure 3.4  The vertical ground reaction force at heel strike, heel strike transient and peak axial acceleration during walking (Whittle et al., 1994, pp130).

Figure 3.5  Free body diagram of the mechanics of the accelerometer mounting on a) skin and b) bone.

Figure 3.6  Free body diagram of the chain of structures from ‘a’ at The heel to ‘a2’ at the jaw.

Figure 3.7  The able-bodied walking cycle (Sutherland et al., 1994, pp26).

Figure 3.8  Temporal gait parameters (Whittle, 1991, pp55).
Figure 3.9  Sagittal plane lower limb joint angles during a single gait cycle for an able-bodied subject walking. Hip (flexion positive), knee (flexion positive) and ankle (dorsiflexion positive) (Whittle, 1991, pp58).

Figure 3.10  Sagittal plane lower limb joint angles during a single gait cycle for a below-knee amputee subject walking. Hip (flexion positive), knee (flexion positive) and ankle (dorsiflexion positive) (Whittle, 1991, pp123).

Figure 3.11  Angles during a single gait cycle for an above-knee amputee subject walking. Hip, knee and ankle (flexion positive) (Whittle, 1991, pp122).

Figure 3.12  Position of the limb and ground reaction force vector during stance phase walking (adapted from Whittle, 1991, pp60-68).

Figure 3.13  a) ankle  b) knee and c) hip net joint moments of able-bodied subjects walking normalised to body weight and leg length (Meglan and Todd, 1994, pp91).

Figure 3.14  a) ankle  b) knee and c) hip joint power outputs of able-bodied subjects walking at a natural cadence (Meglan and Todd, 1994, pp94).
Figure 3.15  Ankle, knee and hip net joint moments of below-knee amputees walking at a natural cadence (Winter and Sienko, 1988, pp364).

Figure 3.16  a) ankle, b) knee and c) hip joint power output values for below-knee amputee subjects walking at a natural cadence (Winter & Sienko, 1988, pp364).

Figure 3.17  Muscles active during the gait cycle. Adapted from Gage (1990), pp294-300.

Figure 4.1  Infotronic CDG sensor shoes with 8 sensors.

Figure 4.2  An example of raw data obtained from one step of the CDG® sensor shoe.

Figure 4.3  An example of raw data obtained from one step measured by the Kistler force platform.

Figure 4.4  Comparison of mean CDG and vGRF curves for each subject walking at a natural cadence.

Figure 4.5  Scatter plot for the CDG and Kistler vGRF curve data points for all able-bodied subjects walking at a natural cadence.

Figure 4.6  An example of raw vGRF data from a) the intact and b) the Prosthetic limb of an above-knee amputee walking at 1.2 m.s⁻¹.
Figure 4.7  Vertical ground reaction force and impulse values and asymmetry during walking for able-bodied subjects (thick black line), above-knee amputees (blue square) and below-knee amputees (red diamond). Dotted line indicates prosthetic limb, solid intact limb.

Figure 4.8  Stance, swing and step time values and asymmetry during walking for able-bodied subjects (thick black line), above-knee amputees (blue square) and below-knee amputees (red diamond). Dotted line indicates prosthetic limb, solid intact limb.

Figure 5.1  The triaxial accelerometer mounting for the right leg.

Figure 5.2  The accelerometer mounting and axes of the force platform at heel strike.

Figure 5.3  Fast Fourrier Transformation analysis for the frequency content of the accelerometer signal during walking at 0.5 m.s\(^{-1}\).

Figure 5.4  Graphs showing peak tibial acceleration (unsmoothed) and the corresponding force load rate (smoothed) for the a) vertical, b) AP and c) ML directions.

Figure 5.5  Linear regression and line of best-fit for peak tibial acceleration and load rate in the axial direction for walking at 1.2 m.s\(^{-1}\).
Figure 5.6  Linear regression and line of best-fit for peak tibial acceleration and load rate in the AP direction for walking at 1.2 m.s\(^{-1}\).

Figure 5.7  Linear regression and line of best-fit for peak tibial acceleration and load rate in the ML direction for walking at 1.2 m.s\(^{-1}\).

Figure 5.8  The FFT analysis for the bite bar accelerometer signal during walking at 0.5 m.s\(^{-1}\).

Figure 5.9  Peak axial, ML, AP and bite bar data during walking at 1.2 m.s\(^{-1}\) for an able-bodied subject.

Figure 5.10  Impact shock in the ML, AP and axial directions at heel strike for able-bodied subjects walking with a) no pain, b) previously reported pain. Black line indicates dominant or pain free limb, red non-dominant or previously reported painful limb.

Figure 5.11  Shock attenuation for able-bodied subjects walking with a) no pain, b) previously reported pain. Black indicates dominant or pain free limb, red non-dominant or previously reported painful limb.

Figure 5.12  Impact shock asymmetry for able-bodied subjects walking in a) the axial direction, b) the AP direction and c) the ML direction. Black indicates pain free subjects, red subjects previously reported painful limb.
Figure 5.13  Shock attenuation asymmetry for able-bodied subjects walking. Black indicates pain free subjects, red subjects previously reported painful limb.

Figure 5.14  Peak axial, ML, AP and bite bar data for a) the intact limb, b) the prosthetic limb for a below-knee amputee walking at 1.2 m.s⁻¹.

Figure 5.15  Axial impact shock for the prosthetic (dashed line) and intact (solid line) limbs of individual below-knee and above-knee amputees. Thick black line, able-bodied subjects.

Figure 5.16  Shock attenuation for the prosthetic limb (dashed line) and intact limb (solid line) of individual below- and above-knee amputees. Thick black line, able-bodied subjects.

Figure 5.17  Impact shock asymmetry of individual above- and below-knee amputees with increasing walking speed.

Figure 5.18  Shock attenuation asymmetry of individual above- and below-knee amputees.

Figure 6.1  The six camera Proreflex (Qualysis) system.

Figure 6.2  Joint angle conventions for the kinematic data.
Figure 6.3 Location of the hip joint centre and greater trochanter.

Figure 6.4 Diagram of joint moment calculations.

Figure 6.5 Mean peak joint angles of able-bodied subjects walking at different speeds. Black line indicates dominant limb, red non-dominant limb.

Figure 6.6 Lower limb joint angle asymmetry for able-bodied subjects walking at different speeds.

Figure 6.7 Mean peak joint angles of amputees and able-bodied subjects walking at different speeds. Thick black line indicates able-bodied subjects, red line above-knee and green line below-knee amputees. Solid line indicates intact limb, dotted line prosthetic limb.

Figure 6.8 Asymmetry of kinematic variables for a) above-knee and b) below-knee amputees during walking at different speeds.

Figure 6.9 An example of the average ankle joint moment curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).

Figure 6.10 An example of the average knee joint moment curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).
Figure 6.11  An example of the average hip joint moment curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).

Figure 6.12  Mean peak net joint moments of able-bodied subjects walking at different speeds. Black line indicates dominant limb, red non-dominant limb.

Figure 6.13  Net joint moment asymmetry for able-bodied subjects walking at different speeds.

Figure 6.14  An example of the net ankle joint moment curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s\(^{-1}\).

Figure 6.15  An example of the net knee joint moment curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s\(^{-1}\).

Figure 6.16  An example of the net hip joint moment curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s\(^{-1}\).

Figure 6.17  Mean peak net joint moments of above-knee amputees and able-bodied subjects walking at different speeds. Thick black line indicates able-bodied subjects, red line above-knee amputees, green line below-knee amputees. Solid line indicates intact limb,
dotted line prosthetic limb.

Figure 6.18 Mean asymmetry of kinetic variables for a) above-knee and b) below-knee amputees during walking at different speeds.

Figure 6.19 An example of the average ankle joint power output curves for an able-bodied subject walking at 1.2 m.s⁻¹.

Figure 6.20 An example of the average knee joint power output curves for an able-bodied subject walking at 1.2 m.s⁻¹.

Figure 6.21 An example of the average hip joint power output curves for an able-bodied subject walking at 1.2 m.s⁻¹.

Figure 6.22 Mean peak power output of able-bodied subjects walking at different speeds. Black line indicates dominant limb, red non-dominant limb.

Figure 6.23 Mean peak power output asymmetry for able-bodied subjects walking at different speeds.

Figure 6.24 An example of the net ankle joint power output curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s⁻¹.
Figure 6.25 An example of the net knee joint power output curves for
a) an above-knee amputee and b) a below-knee amputee walking
at 1.2 m.s\(^{-1}\).

Figure 6.26 An example of the net hip joint power output curves for
a) an above-knee amputee and b) a below-knee amputee walking
at 1.2 m.s\(^{-1}\).

Figure 6.27 Mean peak power output for amputee and able-bodied subjects
walking at different speeds. Thick black line indicates able-bodied
subjects, red line above-knee amputees and green below-knee
amputees. Solid line indicates intact limb, dotted line prosthetic limb.

Figure 6.28 Asymmetry of joint power output variables for a) above-knee
and b) below-knee amputees during walking at different speeds.

Figure 7.1 Fast Fourrier Transformation analysis of the EMG signal during
walking at 1.2 m.s\(^{-1}\).

Figure 7.2 An example of raw EMG data from one limb of an able-bodied
subject walking at 1.2 m.s\(^{-1}\).

Figure 7.3 An example of mean (± SD) muscle activation patterns for
an able-bodied subject walking at 1.2 m.s\(^{-1}\).
Figure 7.4  An example of raw EMG data from the intact limb of an above-knee amputee walking at 1.2 m.s\(^{-1}\).

Figure 7.5  Mean EMG muscle activity for able-bodied subjects walking at increasing walking speed.

Figure 7.6  Mean tibialis anterior and medial gastrocnemius muscle activity for able-bodied subjects, the intact limb of above-knee and below-knee amputees walking at a) 0.5 m.s\(^{-1}\), b) 0.9 m.s\(^{-1}\), c) 1.2 m.s\(^{-1}\) and d) maximum walking speed. Black line indicates able-bodied subjects, red below-knee and green above-knee amputees.

Figure 7.7  Mean rectus femoris and vastus lateralis muscle activity for able-bodied subjects, the intact limb of above-knee and below-knee amputees walking at a) 0.5 m.s\(^{-1}\), b) 0.9 m.s\(^{-1}\), c) 1.2 m.s\(^{-1}\) and d) maximum walking speed. Black line indicates able-bodied subjects, red below-knee and green above-knee amputees. Solid line indicates intact limb, dotted residual limb.

Figure 7.8  Mean biceps femoris and tensor fascia latae muscle activity for able-bodied subjects, the intact limb of above-knee and below-knee amputees walking at a) 0.5 m.s\(^{-1}\), b) 0.9 m.s\(^{-1}\), c) 1.2 m.s\(^{-1}\) and d) maximum walking speed. Black line indicates able-bodied subjects, red below-knee and green above-knee amputees. Solid line indicates intact limb, dotted residual limb.
List of Tables.

Table 3.1  Vertical GRF values for a range of different feet mechanisms from different studies reporting below-knee amputees walking at a natural cadence.

Table 3.2  Temporal variables during able-bodied walking.

Table 3.3  Temporal variables for below-knee amputee gait.

Table 3.4  Temporal variables of above-knee amputees during walking.

Table 4.1  Mean (± SD) of vertical ground reaction force $F_z$ peak for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 4.2  Mean (± SD) of force asymmetry for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 4.3  Mean (± SD) of impulse for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 4.4  Mean (± SD) of impulse asymmetry for able-bodied subjects, above- and below-knee amputees with increasing walking speed.
Table 4.5  Mean (± SD) of impulse for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 4.6  Mean (± SD) of impulse asymmetry for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 4.7  Mean (± SD) of swing time for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 4.8  Mean (± SD) of swing time asymmetry for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 4.9  Mean (± SD) of step times for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 4.10 Mean (± SD) of step time asymmetry for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

Table 5.1  Mean (± S.D.) of peak acceleration and load rate for able-bodied subjects (no-pain) walking with increasing speed. D is dominant limb, ND non-dominant limb.

Table 5.2  Mean (± S.D.) of peak acceleration and load rate for able-bodied subjects (previously reported joint pain) walking with increasing speed. P indicates previously painful limb, NP non-painful limb.
Table 5.3  Correlations (r values) between force load rate and peak acceleration in the ML, AP and axial directions for walking at different speeds.

Table 5.4  Amputee subject details. RAK indicates right limb above-knee amputee, LBK left limb below-knee amputee.

Table 5.5  Mean (± SD) of shock and shock attenuation asymmetry for able-bodied subjects without back or leg pain or injury.

Table 5.6  Mean (± SD) of shock and shock attenuation asymmetry for able-bodied subjects with back or leg pain.

Table 5.7  Mean (± SD) of peak axial shock, shock attenuated and peak bite bar impact shock for able-bodied subjects. Mean of left and right limbs for both 'with no back or leg pain' and 'back or leg pain'.

Table 6.1  The error in peak joint moment (in Nm) yielded with a change in force platform origin of 1, 2 and 5 mm in the AP and ML directions. PF = planar flexor, DF= dorsi flexor, ext = extensor and flex = flexor moment.

Table 6.2  The error in peak joint power output (in Watts) yielded with a change in force platform origin of 1, 2 and 5 mm in the AP and ML directions. Gen indicates power generated, abs, power absorbed.
Table 6.3 Intra-subject variability (coefficient of variation) for able-bodied kinematics during walking at 1.2 m.s\(^{-1}\) (6 trials used).

| Table 6.4 | Intra-subject variability (coefficient of variation %) for able-bodied kinetics during walking at 1.2 m.s\(^{-1}\). PF = planar flexor, DF = dorsi flexor, ext = extensor and flex = flexor moment. Gen indicates power generated, abs, power absorbed (6 trials used). |
| Table 6.5 | Maximum and minimum mean joint angles (degrees) from both limbs of able-bodied subjects walking at different speeds. HS indicates heel strike, MS midstance and TO toe off. |
| Table 6.6 | Joint angle asymmetry (%) for able-bodied subjects walking at different speeds. HS indicates heel strike, MS midstance and TO toe off. |
| Table 6.7 | Mean peak net joint moments (Nm/kg) from both limbs of able-bodied subjects walking at different speeds. PF indicates plantar flexor moment, DF dorsi flexor moment, flex flexor moment, ext extensor moment. |
| Table 6.8 | Mean peak net joint moment asymmetry (%) for able-bodied subjects walking at different speeds. PF indicates plantar flexor moment, DF dorsi flexor moment, flex flexor moment, ext extensor moment. |
Table 6.9  Mean peak joint power output (W/kg) from both limbs of able-bodied subjects walking at different speeds. Gen indicates power generated, abs power absorbed.

Table 6.10  Mean joint power output asymmetry (%) for able-bodied subjects walking at different speeds. Gen indicates power generated, abs power absorbed.

Table 7.1  Coefficient of variation (c.v.) of the different normalisation techniques for each muscle.

Table 7.2  Cross-correlation for the mean of EMG activity for each muscle between able-bodied subjects and the intact limb of above-knee amputees.

Table 7.3  Cross-correlation for the mean of EMG activity for each muscle between able-bodied subjects and the intact limb of below-knee amputees.

Table 7.4  Cross-correlation for the mean of EMG activity for each muscle between the residual and intact limb of below-knee amputees.
List of Plates.

Plate 1. The Infotronic CDG system. 120

Plate 2. An amputee walking using the Infotronic CDG system. 131

Plate 3. Accelerometer mountings on the amputees' prosthetic and intact limbs and the bite bar. 173

Plate 4. The 14 point marker system (front and side views). 200
1.0 Introduction.
1. Introduction.

The use of prostheses for lower limb amputees has been found to date back to at least 300 B.C. and the prostheses were most commonly used for congenital lower limb deficiencies (Troup and Wood, 1982). Prostheses with artificial joints were first developed in the 17th Century as the need for artificial limbs grew due to surgical amputations performed as a result of trauma injuries on otherwise healthy individuals. In the 20th Century, the two World Wars resulted in many individuals surviving trauma as lower limb amputees. Consequently, new amputation techniques were developed and research into prosthetic design was increased.

Each year in the U.K., there are 4500 new amputees, 70% as a result of vascular problems, 10% from other illnesses, 17% trauma and 3% from congenital deficiencies (Troup and Wood, 1982). The 20% of amputees from trauma or congenital deficiencies are usually young, previously fit and healthy. They are encouraged to exercise and take up sport as part of their rehabilitation (Guttman, 1976) as the decreased use of the involved extremity following amputation leads to significant muscle atrophy and weakness (Renstrom et al., 1983) leading to more severe gait problems.

In earlier times, the perceived limit to performance of lower limb amputees has proved to be inaccurate as they are now able to compete in sport at a high level. In the last Paralympic Games in Atlanta, 1996, there were over 3500 competitors including several in amputee events. It is estimated that 20,000 amputees currently participate actively in various sports worldwide, with more than 5000 in organised competition.
With so many lower limb amputees involved in sport and exercise, demands for a prosthesis that will allow as 'near normal' a gait as possible are high. The nature of amputee locomotion needs to be fully understood to aid advances in prosthetic design for sport and exercise as it has for everyday activities. If difficulties are experienced with the prosthesis during walking, lower limb amputees may be less inclined or unable to take part in sport or exercise and this may lead to muscle weakness and further impair gait. Thus, assessing amputee gait during walking as walking speed increases may indicate the potential difficulties lower limb amputees experience as they undertake more vigorous exercise.

The adaptation to the partial loss of a lower limb consists of a compensation produced by the remaining musculature. This compensation leads to an asymmetrical gait pattern and is reflected in joint kinematics, joint kinetics and muscle activation patterns. Problems are experienced by lower limb amputees on both the prosthetic and intact limb. Studies have reported that lower limb unilateral amputees spend only a short time on their prosthetic limb compared to their intact limb as though they suffer discomfort or do not 'trust' the prosthetic limb (Breakey, 1976; Murray et al., 1983). This could partly be due to the type of prosthetic socket worn. Previously, leather corset-type sockets were used but with no close fit between residual limb and socket, allowing movement of the socket, affecting gait. Currently, moulded plastic suction-type sockets are made from casts of the residual limb, overcoming excessive movement between residual limb and socket. However, problems with these type of sockets can also arise from an inexact fit, causing abrasions and discomfort over bony prominences, also possibly affecting gait. Often,
problems with the residual part of the limb include soft tissue not suitable for load bearing (Silver-Thorn et al., 1996), osteoporosis (Burke et al., 1978), loss of musculature and atrophy of the residual limb and bone (Torres-Moreno et al., 1997) and inertial properties and weight of the prosthesis differing to the intact limb. The intact limb of unilateral amputees is reported to experience hypertrophy of musculature and bone (Torres-Moreno et al., 1997), limb pain, joint degeneration and osteoarthritis (Hungerford and Cockin, 1975; Burke et al., 1978). Lower back pain in unilateral amputees has also been reported (Burke et al., 1978).

These problems can prevent amputees from involvement in sport, exercise and performing everyday tasks, leading to a reduction in their quality of life. The causes of these problems are not yet well established, but are thought to be linked to asymmetrical loading on the lower limbs (Burke et al., 1978). It is not known whether these asymmetries contribute to problems experienced in amputee gait or vice versa (Dingwell et al., 1994). When greater demands are made on the amputee, such as in sport or exercise or through increased walking speed, limb loading, muscle activity, joint moments and power outputs may all increase. If there is a difference between the limbs for these selected gait variables and the prosthetic limb does not behave in the same way as the intact limb, this may result in increased asymmetry. Increased asymmetry may cause further problems in amputee gait leading to pain or discomfort possibly in the intact limb. This has implications not only for those involved in sport, but also for younger amputees in their later life who may suffer from the problems noted above. No study has been found which has investigated the effects that greater demands, such as increased
walking speed, make on various aspects of gait asymmetry. By increasing walking speed, amputees will demonstrate how they respond to the faster speeds, increasing or reducing gait asymmetry in order to cope with the higher demands placed on them.

Research is needed to investigate aspects of asymmetry in amputee gait and how this is affected by increasing locomotor demands. A better understanding of amputee gait is required to inform the development of prostheses that may lead to a reduction in asymmetry and reduce the occurrence and magnitude of problems such as lower back and intact limb pain or joint degeneration. In so doing, the amputee may hope for improved gait, a greater prospect for involvement in sport and exercise and a better quality of life.
2.0 Aims and Objectives.
2. Aims and Objectives.

This thesis is concerned with the measurement of aspects of asymmetry in amputee gait in comparison to able-bodied persons, and the effect of increased walking speed on these variables. The aim of this thesis is therefore:

To investigate the effects of walking speed on selected gait characteristics and asymmetries in above- and below-knee amputee gait.

Fulfilment of this aim will enhance the understanding of the mechanisms by which above- and below-knee amputees compensate for the partial loss of a limb under natural and increased walking demands.

To satisfy this aim, the following objectives need to be met:

1. To assess the effects of increased walking speed on the vertical ground reaction force on both the intact and prosthetic limbs of above- and below-knee amputees in comparison to able-bodied persons.

2. To examine impact shock and shock attenuation on both the intact and prosthetic limbs of above- and below-knee amputees during walking and to assess the effects of increased walking speed on impact shock and shock attenuation on both limbs of above- and below-knee amputees in comparison to able-bodied persons.

3. To examine joint moments and power output on the intact and prosthetic limbs of above- and below-knee amputees during walking to establish the compensation mechanisms used by the intact limb and to assess the effects of increased walking speed.
on joint moments and power output on both the intact and prosthetic limbs of above- and below-knee amputees in comparison to able-bodied persons.

4. To examine muscle activity in the intact and residual limbs of below-knee amputees and the intact limb of above-knee amputees during walking to establish the compensation mechanisms used by the intact limb, and to assess the effects of increased walking speed on muscle activity on both the intact and residual limbs of below-knee amputees and the intact limb of above-knee amputees in comparison to able-bodied persons.

Hence, the project is concerned with the measurement of kinematic and kinetic aspects of amputee gait during different walking speeds. The data will be used to evaluate the effects of walking speed on selected aspects of amputee gait asymmetry.

Approval was granted by the Liverpool John Moores University Ethics Committee for all of the experimental work in this study.
2.1 Definition of terms.

*Residual limb* – the remaining section of the limb following amputation.

*Prosthetic limb* – the residual limb and prosthesis together.

*Intact limb* – the non-amputated limb.

*Contralateral limb* – opposite limb.

*Established amputees* – those amputees who have been walking without aids for one year or more and are deemed “good to excellent” walkers by the consultant at the limb fitting centre they attend.

*Highly active amputees* – those amputees who regularly take part in sport or exercise either recreationally or competitively.

*Asymmetry* – the percentage difference between the left (L) and right (R) limbs for the variable measured, using the equation for absolute asymmetry index (ASI):

\[
ASI = \frac{L - R}{0.5 (L + R)} \times 100
\]

*vGRF* – the vertical component of the ground reaction force.

*Limb loading* – the general effect of the vGRF on the limb.

*Heel strike peak* – the initial vGRF peak corresponding to heel strike.
\( Fz1 \) – the initial loading peak (after the heel strike peak) of the vGRF corresponding to weight-acceptance.

\( Fz2 \) - the second loading peak (after the heel strike peak) of the vGRF corresponding to push-off.

Heel strike transient – the derivative of the vGRF curve from the first instance of ground contact to the heel strike peak (also termed the Fz load rate).

Impact shock – the peak acceleration in each of the anterior-posterior, medio-lateral and axial directions occurring at heel strike.

Shock attenuation – the percentage difference between the axial impact shock at heel strike and the next axial bite bar peak corresponding to that impact.

Step – from heel strike on one foot to heel strike on the contralateral foot.

Stride - from heel strike on one foot to the next heel strike on the same foot.

Stance – the time the foot is in contact with the ground i.e. from heel strike to toe-off on the same foot.

Swing - the time the foot is not in contact with the ground i.e. from toe-off to heel strike on the same foot.
3.0 Review of Literature.
3. Review of Literature.

3.1 Introduction.

In this review of literature, previously established important aspects of able-bodied gait will be identified. Further, an attempt is made to identify the areas in which amputee gait differs from able-bodied gait both for the prosthetic and intact limb.

Reference is made to the underlying biomechanical principles of movement. The review of muscle mechanics is not intended to be comprehensive but is concentrated upon the relevance of the muscle activation patterns to gait.

The project is concerned with not only identifying aspects of amputee gait asymmetry but also the effects increased demands, such as increasing walking speed, have on gait asymmetry. This section will cover aspects relating to the asymmetry of amputee gait during walking. The merits of various methodologies used to determine loading, impact shock, shock attenuation, net joint moments, power output and muscle activity will be assessed and inferences drawn as to the most appropriate means of measuring these aspects of amputee gait asymmetry.
3.2 Limb loading and loading asymmetry.

3.2.1 Able-bodied gait.

3.2.1.1 Typical ground reaction forces.

Methods of measuring components of the ground reaction force (vertical, anterior-posterior shear and medio-lateral shear) have been improving since the early 20th century (Johanson, 1994). Consequently, the force platform has enabled recent studies of locomotion to produce repeatable data for able-bodied gait. This has led not only to an understanding of the basic locomotor mechanisms, but also ground reaction force analysis has been used as a diagnostic tool to evaluate pathological gait (Nilsson and Thorstensson, 1989).

![Figure 3.1 - Vertical, anterior-posterior and medio-lateral forces during able-bodied walking. The shading represents variability (Meglan and Todd, 1994, pp76).](image)

The ground reaction force (GRF) is made up of vertical (v) and shear (anterior-posterior AP, and medio-lateral ML) components, each showing a distinctive and repeatable trace (Figure 3.1). The majority of research in walking has concentrated on the vGRF component which is characterised by a sharp initial heel-strike peak not
shown on Figure 3.1), an initial impact peak during weight-acceptance (Fz1), mid-
stance trough and a peak at push-off (Fz2). The shape of the Fz1 peak is influenced by
the intensity and timing at footstrike, walking speed and knee flexion (Claeys et al.,
1983). The shear reaction force components, AP and ML are needed to initiate and bring
to an end periods of locomotion as well as changing speed and direction of walking.
Bipedal locomotion requires shear forces to maintain body balance (Meglan and Todd,
1994). The supporting limb transmits the GRF to the ground, but the GRF is a reflection
of the acceleration of the total body. Thus the motion of the segments and their
individual accelerations contribute to the overall GRF. The linear acceleration of the
centre of gravity of the head and trunk segment accounts for slightly more than half the
total acceleration, the upper extremities 5% each and the lower extremities 18% each
(Miller, 1990). Thus, as the GRF is equal to the total body mass * total body
acceleration (Newton’s Second Law), any acceleration or deceleration of individual
segments will result in an increase or decrease in GRF. This is illustrated by the vGRF
during walking. During midstance, the supporting limb flexes at the knee during the
‘cushioning’ phase resulting in a downward acceleration, or deceleration of the centre of
gravity. This deceleration of the centre of gravity results in a vGRF of less than one
body weight (Figure 3.1). During weight-acceptance (Fz1) and push-off (Fz2), the body
is accelerating, and this a vGRF of greater than one body weight is observed. This is
also seen in the Fx and Fy directions, and accounts for the positive and negative GRF
magnitudes.

For able-bodied individuals walking at a fixed normal cadence, the vGRF peaks
at about 110% body weight (BW) (Sutherland et al., 1988; Winter, 1990) and differs
depending on footwear (Lafortune and Hennig, 1992) and walking speed (Nilsson and
Thorstensson, 1989). Changes in cadence can also affect the magnitude and duration of
the vGRF which reflects the rate of limb loading (Mann and Hagy, 1980; Skinner, 1981). Changes in the vertical force pattern have previously been reported to be a result of pain, clinical pathology or weakness (Harris et al., 1994). However, the vGRF has been found to be unreliable as a clinical measure due to the action-reaction of arm lifting which can diminish the peak component to less than body weight (BW) (Charnley and Pusso, 1968). Thus, whilst changes in vGRF can indicate pathology, the exact nature of the pathology cannot be indicated from ground reaction forces alone.

3.2.1.2. Effects of speed, stride length and stride frequency on ground reaction forces.

The speed of walking significantly affects GRF characteristics (Andriacchi et al., 1977; Nilsson and Thorstensson, 1989; Martin and Marsh, 1992; Keller et al., 1996). Keller et al. (1996) reported increases in peak vGRF from 1.15 BW to 2.11 BW during walking from 1.5 to 3 m.s\(^{-1}\). By increasing walking speed from 1 to 3 m.s\(^{-1}\), Nilsson and Thorstensson (1989) found peak vGRF to increase from 1 to 1.5 BW, and the anterior-posterior and medio-lateral peak to peak to double. Whilst the magnitude of peak vGRF at a certain walking speed has been reported to differ slightly between studies, all studies have reported increases in magnitude with walking speed for both vertical and shear force components.

Although the effect of speed on GRF has been established, the effects of stride length (SL) and stride frequency (SF) on GRF have been little documented. Meglan and Todd (1994) suggested that an increase in walking speed, which usually increases stride length, results in an increase in lateral force peaks. Martin and Marsh (1992) found contact time, AP braking force (Fy1), AP propulsive force (Fy2) and vertical impulse per step systematically increased as stride length increased. Peak vGRF, however, showed little change with increasing stride length when walking speed was controlled.
Thus, it may be the combination of SL and SF that results in increases in vGRF, and not just increases in SL. Laish et al. (1988) reported aged adults exhibited shorter stride lengths and decreased vertical and AP GRF peaks when compared to young adults. These results may have been due to the aged adults selecting a gait pattern (by altering SL and SF) in order to reduce GRF magnitude and therefore decreased stress on the musculoskeletal system. When interpreting trends for all three components of the GRF, it is necessary to take into account differences in stride length and frequency during walking, either due to disability or age.

3.2.1.3 Variation and asymmetry.

Variation.

The amount of step-to-step variation is dependent on the degree of control during gait. During walking, corrections for change in position, velocity and forces for each step is done using proprioceptive feedback (Zahedi et al., 1987). The analysis of a human motor skill involves not only the assessment of how one subject performs relative to a population norm, but also the repeatability of that subject’s performance (Smith, 1993). Given the inherent variability of gait data (Smith, 1993), it would be useful to obtain the average over a number of trials. However, the number of trials needed to produce data representative of an average stride has differed from study to study. The number of trials needed to give a stable mean value for GRFs has previously been reported. One study reported the average of three trials (Simon et al., 1981) with the majority of studies using the mean of ten trials (Hamill et al., 1983; Herzog et al., 1989; Hamill and McNiven, 1990; Grabiner et al., 1995). Grabiner et al. (1995) reported a coefficient of variation over the ten trials for a multiple step approach of ± 6.07% for the vertical, ± 41.55% for the AP and ± 84.24% for the ML GRF components. Thus,
whilst able-bodied subjects exhibit little GRF variability in the vertical direction, the AP and particularly the ML peak force during walking is extremely variable.

The problem in reproducing GRF data (obtained by the force platform) is the large number of trials needed. Stride adjustments occur when approaching a target (Lee et al., 1982; Hay, 1988; Hay and Koh, 1988; McGinas and Abendroth-Smith, 1989). Such stride adjustments on approaching the force platform may affect the GRF measurements (Abendroth-Smith, 1996). By manipulating step length or frequency, differences have been seen in support time, peak vGRF, average vGRF and AP GRF (Holt et al., 1987; Martin and Marsh, 1992). Abendroth-Smith (1996), on investigating the approach to the force platform, found that for running, significant stride alterations do occur at force platform contact. However, Grabner et al. (1995) on looking at walking, found that variability of GRFs altered by targeting or not targeting the force platform were not significant (coefficient of variation of Fz I targeting the force platform ± 6.07%, not targeting ± 6.27%). What did increase the variability in GRF was the number of strides taken to approach the force platform. Regardless of whether the platform was targeted or not, single steps produced a greater variability than multiple steps in the AP force (Grabner et al., 1995).

Asymmetry.

Able-bodied gait has been previously shown to be slightly asymmetrical. By using an asymmetry index (ASI) (Herzog et al., 1989) where

\[
ASI = \frac{(L-R)}{0.5(L+R)} \times 100
\]
illustrated a difference of less than ±4% between left and right limbs during normal walking for 34 gait variables. Hamill et al. (1983) reported no significant asymmetry between the able-bodied left and right limbs during walking and running, but Fz1, Fy1 and Fy2 values were slightly greater on the dominant compared to the non-dominant limb. Jarnett et al. (1980), on using a different symmetry index where 100% was perfect symmetry, reported able-bodied gait to be well above 90%. Therefore, able-bodied gait has previously been reported to be symmetrical with a less than 10% difference between left and right limbs, regardless of the symmetry index used.

3.2.1.4 Alternative methods of vertical ground reaction force and asymmetry measurements.

It is difficult to measure the three GRF components over continuous strides with current technology. A system of force plates will measure the GRF of 1-2 strides but two plates are expensive and do not give information of a step-to-step nature and therefore the ability to measure GRF asymmetry easily. When calculating asymmetry from selected gait variables, one has to be wary of expressing asymmetry between non-consecutive steps (Vagenas, 1989). Walking is a continuous motor skill that requires the maintenance of body balance through compensatory segmental movements. Compensatory forces and moments in the joints of the body act to counterbalance reduced or increased displacements of other body segments. Thus, expressing asymmetry between mean scores on the left and right sides of the body may mask actual bilateral dominance trends (Vagenas, 1989). Taking this into account, it would be useful if GRFs measured to express asymmetry could be obtained for consecutive right and left steps.
Within the past two decades, developments have enabled the measurement of force over continuous strides through small force transducers placed on pre-selected areas of the sole of the foot or shoe insole (Klajajic and Kranjnik, 1987). These currently only measure the vertical component of the GRF. Some systems have been developed further to measure pressure distribution, but as this thesis does not aim to investigate pressure distribution, these will not be reported here. Studies comparing the vertical force magnitude and pattern of data obtained by force shoes to force plates have reported little difference between wearing the shoes and walking over the force plate barefoot. This percentage difference was reported to be less than the difference between barefoot and shod force plate values, but was not quantified (Klajajic and Krajnik, 1987). Another study reported a difference 'far below 10%' between the vertical force curve magnitude for wearing force shoes compared to walking over force plates for many different types of gait (Krajnik et al., 1981).

The number of force transducers on each shoe sole has ranged from five (Ranu, 1987) to nine (Klajajic and Krajnik, 1987). The main disadvantage of this system is that not all of the area of the foot is covered by sensors and thus the vGRF measured may be slightly less than measured by a force plate. An eight-sensor system (CDG, Infotronic Systems, NL) has been used in a number of clinical studies to give instant feedback and look at step-to-step variation and gait asymmetry. These studies (Krajnik et al., 1981; Hermens et al., 1986; Klajajic and Krajnik, 1987; Zilvold and Baumgartner, 1988) have mainly reported on the instrumentation or a qualitative assessment of case studies. Hermens et al. (1986) showed gait asymmetry in several pathological case studies, but it was not quantified. By using the CDG shoes, the level of weight bearing during stance, level of loading at heel strike, the path of force throughout stance and the magnitude of the push-off peaks can be determined to analyse clinical gait. This
equipment was used in a case study to compare the vertical force components during walking of two different below-knee prostheses (Hermens et al., 1986). The CDG system showed ‘slight differences’ although these were not quantified.

Although the force shoes only measure the vertical component of the GRF, all the studies reported above concluded that they were advantageous in obtaining data from several continuous strides and looking at the loading under specific parts of the foot. From this amount of data, gait asymmetry in terms of the vGRF can easily be calculated in order to assess continuous step-to-step variation, and also to look at the degree of asymmetry during continuous striding in able-bodied as well as clinical gait.

3.2.1.5 Summary of able-bodied limb loading.

Limb loading and loading asymmetry have been previously well documented in able-bodied gait. Vertical GRF peaks have been shown to increase from just over 1 BW to just over 2 BW with increasing walking speed. Increases in stride length and stride frequency have also been shown to affect vertical impulse, vertical and shear forces during walking. The methodology of determining GRFs has also been discussed. Using force plates, as most studies do, alterations in GRF from targeting the force platform were not found to be significant. If multiple steps rather than single steps were taken to approach the force platform, trial-to-trial variability was found to be low. There have been comments, however, on the use of force plates to calculate loading asymmetry. As only one step per trial can be recorded by the force platform, loading from consecutive steps cannot be measured and thus the investigator needs to consider errors in calculating asymmetry. Using a system of force plates, or shoes with in-built force transducers can overcome this problem of expressing asymmetry from consecutive footfalls.
3.2.2 Loading and loading asymmetry in amputee gait.

Amputee gait is asymmetrical due to the nature of both the residual limb and the prosthesis. In order to reduce the high energy cost of walking in amputees compared to able-bodied subjects (Waters et al., 1976), the prosthetic limb is made lighter than the corresponding intact limb. This results in differences between the prosthetic and intact limbs for segment mass, centre of mass and moment of inertia properties. In addition to these differences, the prosthetic joints do not allow movement in the same way as the intact joints. Thus, as the prosthesis does not function in the same way as an intact limb, amputees are not expected to walk in the same way as able-bodied persons. Their remaining musculature, both in the residual and intact limb, will also contribute to differences in their walking patterns when compared to able-bodied subjects. Their established walking pattern, although differing from able-bodied gait, may be their most efficient method of walking.

3.2.2.1 Lower limb amputee ground reaction forces.

Studies have reported that amputees load their prosthetic limb less during locomotion than able-bodied persons load their limbs (Engsberg et al., 1991; Engsberg et al., 1993; Stefanyshyn et al., 1994). The soft tissues of the residual part of the limb are not well suited for load bearing (Silver-Thorn et al., 1996) and the load tolerance depends on the biological and physiological structure of these tissues. Thus, the amputees may be trying to protect their residual limb by loading it less.
Other reasons may be that the prosthesis is lighter than the intact limb, or that the centre of pressure (COP) lies more towards the intact limb than the prosthetic limb during stance (Clark and Zernicke, 1981) resulting in the amputee placing more of their body weight towards the intact limb whilst walking. This is implied by studies which have reported a greater load on the intact limb than for the prosthetic limb (Suzuki, 1972; Engsberg et al., 1991; Engsberg et al., 1993). The amputee’s intact limb has been shown to be loaded more than the limbs of able-bodied persons (Suzuki, 1972; Engsberg et al., 1993). Engsberg et al. (1993) reported the intact limb of below-knee amputee children was loaded by an extra 23% BW compared to able-bodied children. Impulse, illustrating the overall vertical loading characteristics of the limb in terms of force magnitude and time, was also greater on the intact limb (0.51 BW/ ratio total support time) compared to the prosthetic limb (0.46) and to able-bodied subjects (0.45). This study concluded that the below-knee amputee children had adapted to the prosthesis and that they should be taught to walk in a way that would reduce the loading differences between the limbs.
Stefanyshyn et al. (1994) reported that the average vGRF peaks for the intact leg were higher than able-bodied subjects whilst the maximum forces for the prosthetic leg were lower than able-bodied subjects, agreeing with Engsberg et al. (1993). Similarly, the peak AP force for the intact limb was higher than or equal to able-bodied subjects whilst the prosthetic limb peak was lower. Such higher forces acting on the intact limb could possibly lead to problems which are associated with joint pain and or joint degeneration in later life due to repeated cyclic loading (Voloshin, 1988).

In contrast, Nissan (1991) reported prosthetic limb vGRF values of 95 to 109% BW compared to the intact limb of 93-105% BW for below-knee and above-knee amputees. This could have been due to the subjects (whether active or sedentary was not reported) or the type of prosthesis used as Nissan (1991) reported that the knee, ankle and foot mechanisms and prosthesis alignments varied considerably. Another explanation could be that as the majority of amputees feel discomfort or do not “trust” their prosthetic limb (Murray et al., 1983), they do not place all their body weight over that limb. For those amputees who are confident to load their prosthetic limb and feel no residual limb discomfort, they may load both their limbs in a similar manner.

3.2.2.2 The effect of walking speed on ground reaction forces.

As a method of trying to reduce the high amount of loading on the intact limb, Lewallen et al. (1985) suggested that below-knee amputees walk with a slower gait than normal. Hermodsson et al. (1994) reported a below-knee amputee natural cadence speed of 0.99 m.s\(^{-1}\) compared to 1.42 m.s\(^{-1}\) for able-bodied subjects. Walking speed is reduced in lower limb amputees compared with healthy able-bodied subjects (Levine, 1984; Saleh and Murdock, 1984; Craik et al., 1985; Engsberg et al., 1991) and is significantly
reduced with increased amputation level (Skinner and Effeney, 1985). Therefore, if natural walking cadence is reported for amputees and able-bodied subjects, the differences in chosen speeds may influence the GRF values. Snyder et al. (1995) reported that amputees walked more slowly than able-bodied subjects, but still had a greater intact limb vertical GRF than able-bodied subjects. Zernicke et al. (1985) reported that speed can alter vGRF for above-knee amputee children. Increased walking speed showed an increase from 1.26 BW to 1.41 BW on their prosthetic limb and 1.39 BW to 2.09 BW on their intact limb. Thus, even though vGRF increases with walking speed for both the prosthetic and intact limb, increases on the intact limb were greater. If this is true, then loading asymmetry may increase at higher walking speeds. The greater loading of the intact limb compared to the prosthetic limb at faster walking speeds has further implications for susceptibility to joint degeneration in the intact limb in later life. Amputees have been shown to walk at a naturally slower speed than able-bodied subjects. By increasing walking speed, it follows that there will be greater loading on the intact limb and thus greater force asymmetry following the results of Zernicke et al. (1985). However, no study has been found which has looked at the effects of increasing walking speed on the vGRFs of unilateral amputees, and no study has been found which has reported the amount of asymmetry.

3.2.2.3 The effects of prosthetic alignment on ground reaction forces.

Sound prosthetic alignment has historically been stressed to improve residual limb comfort and maximise walking capabilities in lower limb amputees (Pinzur et al., 1995). Previous studies have shown that unilateral amputees transfer load to their intact limb to a relatively greater degree than to their prosthetic limb (Breakey, 1976; Hurley et al., 1990; Pinzur et al., 1991), and exhibit asymmetrical gait patterns and an altered biomechanical load when compared to able-bodied subjects. It has been suggested that
there is a single optimum alignment of a prosthesis for the best gait performance (Zahedi et al., 1986), but an amputee can be satisfied by more than one alignment fitting (Zahedi et al., 1986).

One study (Pinzur et al., 1995) looked at GRFs when the prosthetic socket alignment was altered from neutral to a) 10° varus, b) 10° valgus, c) 10° flexion and d) 10° extension. No significant effects of these alignment changes were found although when the prosthetic limb was in neutral, the intact limb exhibited a greater stance phase time (0.877 s compared to 0.824 s for the prosthetic limb), peak Fz1 (865.4 N compared to 792.8 N for the prosthetic limb) and vertical impulse (583.6 N.s compared to 468.9 N.s for the prosthetic limb). When the socket was malaligned by 10°, for the prosthetic limb, Fz1 increased by between 3 and 16 N for all conditions. Vertical impulse increased by between 2 and 18 N.s for all conditions. Stance phase time increased for all malalignments (by 0.01 to 0.04 s for all conditions), but no results were significant. The effects of these malalignments on the intact limb were not quantified. This study showed prosthetic alignment had some effect on GRF, impulse and stance time of the prosthetic limb, but none were significant. The authors concluded that the subjects in the study were sufficiently good walkers that they were able to compensate for the malalignment of their prostheses over a short period of time, but this could differ over a prolonged period. However, the study did not report the effect malalignment had on the intact limb and whether the intact limb is compensating to allow for slight differences in alignment, as this may be an influencing factor when interpreting the results.

Another study (Yang et al., 1991) reported prosthetic foot alignment changes for two above-knee amputees of 6° dorsi flexion to 6° plantar flexion in 3° increments, and socket alignment extended by 6° and 3° and then flexed by 3° and 6. The results showed...
that the AP GRF was sensitive to alignment changes at push off. However, vGRF was not assessed. Thus, from these two studies it can be concluded that whilst prosthetic alignment affects the shear components of GRF, it has not been shown to affect vertical GRF or impulse significantly.

3.2.2.4 The effects of different prostheses on ground reaction forces.

Differences in GRFs from amputees across studies may be accounted for by the different prostheses used. On the prosthetic limb, the Fz1 peak was significantly greater for the SACH foot compared to the Quantum foot with no difference in the timing of the peak (Powers et al., 1994). Winter and Sienko (1988) reported greater GRFs on the intact limb using the SACH foot compared to the FLEX foot, but this was not quantified. Menard et al. (1992) and Powers et al. (1994) reported that for the intact limb, the FLEX foot produced the lowest Fz1 values (Table 3.1). These studies supported the findings of Lewallen et al. (1985) and Hurley et al. (1990) that the FLEX foot does not develop increased joint forces in the intact limb. Snyder et al. (1994) suggested that this may be due to its large arc of dorsi flexion motion. The other prosthetic foot mechanisms, however, did increase joint forces on the intact limb. Therefore, previous studies have shown that the type of prosthetic foot mechanism has an effect on the loading characteristics of the intact limb in below-knee amputees. No studies have looked at the effect of different prosthetic foot mechanisms on above-knee amputee gait.
Table 3.1 - Vertical GRF values for a range of different feet mechanisms from different studies reporting below-knee amputees walking at a natural cadence.

<table>
<thead>
<tr>
<th>Foot mechanism</th>
<th>Powers et al. (1994)</th>
<th>Menard et al. (1992)</th>
<th>Schneider et al. (1993)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fz prosthetic limb (% BW)</td>
<td>Fz intact limb (% BW)</td>
<td>Fz prosthetic limb (% BW)</td>
</tr>
<tr>
<td>SACH</td>
<td>109.7</td>
<td>124.3</td>
<td></td>
</tr>
<tr>
<td>FLEX</td>
<td>118.9</td>
<td>110.9</td>
<td>119.9</td>
</tr>
<tr>
<td>CCH</td>
<td>106.1</td>
<td>116.3</td>
<td></td>
</tr>
<tr>
<td>Seattle</td>
<td>106.7</td>
<td>123.2</td>
<td>120.6</td>
</tr>
<tr>
<td>Quantum</td>
<td>104.9</td>
<td>131.6</td>
<td></td>
</tr>
<tr>
<td>Able-bodied</td>
<td>111.0</td>
<td>111.0</td>
<td>119.9</td>
</tr>
</tbody>
</table>

* denotes study using children.

3.2.2.5 Variation and asymmetry in amputee gait.

Asymmetry.

Whilst it has been widely reported that amputees have an asymmetrical gait, none of the studies reviewed have quantified this asymmetry. It is as yet unknown whether or not these asymmetries cause, contribute to or aggravate the physical complications associated with prosthetic use or vice versa (Dingwell et al., 1994).

Variation.

The amount of step-to-step variation is dependent of the degree of control during the gait. For amputees, the degree of control of the musculo-skeletal structure and thus proprioceptive feedback is reduced due to the partial loss of a limb. The greater amount of limb lost, the less the degree of control. Therefore, whilst it has been sometimes difficult to detect variability in active below-knee amputees, there is more variation in above-knee amputees and even more in hip disarticulation amputees (Zahedi et al., 1987). In amputee walking, due to variability, step 7 of 15 has been found to be the most
representative of a stride, and the first and last 3 steps differ from the rest (Zahedi et al., 1987).

After years of prosthetic use, below-knee amputees appear to learn to co-ordinate the timing of their prosthetic limb to resemble more closely that of their natural limb (Dingwell et al., 1994). Therefore, the length of time an individual has been an amputee could also affect intra-subject variability.

3.2.2.6 Summary of amputee limb loading.

Amputee gait, by the nature of the prosthesis, is asymmetrical. Studies have reported that generally, amputees load their intact limb more and their prosthetic limb less than able-bodied persons. One study has reported that vGRF increased with an increase in walking speed for both the prosthetic and intact limb of above-knee amputees, but the increase was greater on the intact limb. No other study has been found reporting the effect of walking speed on vertical GRF in unilateral amputees. If this study illustrates the loading trends of lower limb amputees, loading asymmetry is expected to increase with walking speed. This has implications for susceptibility to joint degeneration on the intact limb from increased repetitive cyclic loading.

Whilst prosthetic alignment has not been shown to affect vertical GRF variables significantly, the type of prosthesis worn has. No study has previously quantified loading asymmetry in lower limb amputees due to problems in calculating an asymmetry index from non-consecutive footfalls when using the force platform. Very little GRF data have been reported for above-knee amputees and no study has looked at the effects of increased demands, such as increasing walking speed, on amputee loading asymmetry.
3.2.3 Summary of limb loading and loading asymmetry.

Vertical ground reaction force patterns and magnitudes have been used to evaluate pathological gait. In able-bodied walking, vGRFs have been reported to be around 110% body weight, lower for shod than barefoot walking. An increase in walking speed will increase the vGRF to just over two times body weight. Able-bodied variation in GRFs has been well documented, the vertical Fz component being the least variable (c.v. = ± 6%) and the ML component the most (c.v. = ± 84%) over ten trials. Walking asymmetry in able-bodied subjects has been reported to be between 4 and 10%, with the ML and AP force components being slightly greater on the dominant limb. The development of shoes containing in-built force transducers has allowed vertical GRFs over continuous strides to be measured (the results being underestimated less than 10% that measured on a force platform) which is useful in looking at the degree of asymmetry during continuous striding in gait.

Amputees, by the nature of the prosthetic limb, are asymmetrical in their gait. Studies have shown that they load their prosthetic limb less (approximately 95% body weight) and their intact limb more (an extra 23% body weight) than able-bodied subjects during walking. Whilst the effect of different prostheses and prosthetic alignment on asymmetry and GRFs has previously been studied, only one study has reported the effects of increasing walking speed on amputee GRFs (Zernicke et al., 1985). If vGRFs increase with walking speed on both limbs (as with able-bodied subjects), the increase could lead to failure of the soft tissues on the residual limb that are not well-suited for load bearing, and/ or excessive forces on the intact limb. Such high forces acting on a limb could lead to pain, injury or joint degeneration in later life. This has implications
for highly active or sporting amputees who will load their limbs more, and more often than sedentary amputees.

Whether the amputees stay asymmetrical, become less asymmetrical or increase GRF asymmetry as greater demands, such as increasing walking speed, are placed on them, has not been previously reported. By first understanding how an amputee copes with greater demands placed on their limbs at speed, developments of prostheses could lead to an improvement in gait, and/ or risk of pain or injury on either limb. Further analysis of the vertical GRF and the greater forces acting on the limbs at faster walking speeds is needed to assess the likelihood of pain, injury or joint degeneration in later life.
3.3 Impact shock and shock attenuation.

The heel strike transient is the initial contact force occurring at the time of heel strike (Figure 3.3), lasts for about 20 ms in able-bodied walking (Collins and Whittle, 1989) and results from the deceleration of the moving leg (Whittle, 1993). This transient force, or impact shock, at heel strike has been implicated as a possible cause of degenerative joint disease in the lower limb (Radin, 1987), and although it can be measured by the force platform, providing it has a high enough frequency response and data are sampled at a high enough rate, it is most accurately measured using accelerometers (Light et al., 1980). The use of accelerometers also enables the measurement of shock attenuated from the lower limb to the head. This gives an insight into not only the amount of shock experienced by the body during walking, but also how much is attenuated in both clinical and able-bodied patients, providing more information into the effects of gait asymmetry.

![Figure 3.3 - The vertical ground reaction force. A represents the heel strike transient peak (Collins and Whittle, 1989, pp180).](image)
3.3.1 Impact shock in able-bodied gait.

3.3.1.1 Heel strike transient and acceleration.

During walking and running, the foot impacts the ground with a rapid change of velocity decreasing to zero in a short period of time. Measuring this acceleration and expressing it normalised to g, (acceleration due to gravity where g = 9.81 m.s\(^{-2}\)) can be used to illustrate the amount of impact shock or shock attenuated during locomotion. An extensive measure of this impact shock has been the vertical ground reaction force (Fz) load rate, or heel strike transient (Figure 3.4).

![Figure 3.4 - The vertical ground reaction force at heel strike, heel strike transient and peak axial acceleration during walking (Whittle et al., 1994, pp130).](image-url)
Average load rate (the part of the Fz curve from the first instance of force generation to the vertical force peak resulting from heel strike in BW/s) has been correlated with axial acceleration values measured at the tibia. Hennig and Lafortune (1991) reported a positive significant correlation of $r = 0.87$ between peak axial acceleration measured at the tibia, and Fz load rate. Other acceleration and force parameters did not correlate well. Peak tibial acceleration and the peak vertical force associated with the heel strike peak showed only a moderate correlation of $r = 0.76$. Thus, the heel strike transient as measured by Fz load rate can be considered to be a good indicator of peak tibial axial acceleration.

Lightweight accelerometers placed on the shank used to measure peak tibial acceleration have previously quantified impact shock for different walking surfaces, footwear and running speeds. Studies have reported that the placement and method of placement of the accelerometers affect the impact shock measured. The best way to quantify impact shock to the leg accurately would be to attach an accelerometer directly via a pin into the tibia (Valiant, 1990) although in most studies, this is not possible. Comparisons between results obtained by bone mounted and skin mounted accelerometers have been made. Light et al. (1980) used both a skin mounted and bone mounted accelerometer and found similar patterns and order of magnitude, but the skin mounted accelerometer lost some high frequency components. However, another study has reported that skin mounted accelerometers contain high frequency components compared to bone mounted (Hennig and Lafortune, 1988).

Hennig and Lafortune (1988) reported results from a bone-mounted accelerometer, skin mounted accelerometer and an accelerometer mounted on balsa wood and then attached to the skin. Axial accelerations from all three accelerometers
were recorded simultaneously. The bone mounted accelerometer exhibited the lowest axial acceleration at heel strike, the balsa wood mounted accelerometer recorded a 33% greater peak acceleration and the skin mounted accelerometer a 48% greater peak acceleration. The high accelerations for the skin mounted accelerometer were attributed to the soft viscoelastic structure of the skin. When the skin becomes close to its maximal stretching excursion, it becomes stiff very suddenly, leading to an elastic rebound that produces even higher acceleration values (Figure 3.5). By mounting the accelerometer on balsa wood, skin movement is decreased and less soft tissue vibration effects are present. The authors concluded that as the accelerometers used weighed 6 grams, an even lower mass accelerometer would improve the quality of skin accelerations even more. Gross and Nelson (1988) illustrated this with a lightweight 1 gram accelerometer glued to the skin over the distal anterior-medial tibia. The authors calculated an 8% overestimation of peak tibial acceleration compared to a bone mounted accelerometer. Therefore, by using lightweight accelerometers and a balsa wood mounting, skin mounted accelerometers can provide a reasonable, although slightly over estimation of impact shock experienced at the tibia.

Figure 3.5 – Free body diagram of the mechanics of the accelerometer mounting on a) skin and b) bone. a represents the acceleration due to impact, $a_1$ the additional acceleration measured at the bone/skin interface, and $a_2$ the additional acceleration due to skin movement.
3.3.1.2 Uniaxial and multiaxial accelerometry.

Many studies have measured shank acceleration in one direction only. The use of triaxial accelerometers has shown that the largest acceleration at impact does not occur in the axial direction. For running, a large peak tibial acceleration in the anterior-posterior (AP) direction with a slightly smaller peak in the axial direction and a much smaller peak in the medio-lateral (ML) direction was reported (Lafortune and Hennig, 1988). Similar results for shod walking have shown 1.71 g (acceleration due to gravity where $g = 9.81 \text{ m.s}^{-2}$) in the axial direction, 2.09 g in the AP direction and 0.9 g in the ML direction (Lafortune, 1991). These results show that regardless of whether the subject is walking or running, whilst shod, the greatest acceleration measured at impact is in the AP direction followed by the axial and then ML directions. With high accelerations in all three axes, particularly the AP direction, there is a need for measurements in more than one axis.

3.3.1.3 Accelerometer placement.

The placement of the accelerometer on the shank is an important consideration as locomotion will affect the magnitude of impact shock measured. Many studies have reported different skin mounted accelerometer placements such as on the anteromedial aspect of the tibia, 13.5 cm up from the medial maleollus; the proximal anterior part of the tibia, 3-4 cm under the tibial tuberosity; the tibial tuberosity; the medial tibial condyle, and the lateral tibial condyle, making results difficult to compare across studies. Lafortune (1991) stated that tibial axial acceleration as measured by accelerometers attached to the shank was the summation of tibial angular motion and gravity. A further study (Lafortune and Hennig, 1991) calculated the effects of angular motion and gravity to peak tibial accelerations using bone mounted accelerometers.
They reported an over-estimation of 18% peak acceleration for walking and an under-estimation of 43% peak acceleration for running. This study concluded that depending on the distance of the accelerometer from the tibial centre of rotation (located at the ankle joint), different axial acceleration signals should be expected during comparable locomotor activities. If the accelerometer measuring impact shock at heel strike is not located at the ankle, the corrections for the effects of angular motion and gravity are necessary. Therefore, not only accelerometer mounting, but accelerometer placement is an important factor when employing this method of measurement.

3.3.1.4 The effects of footwear and surfaces on impact shock.

Many studies have looked at the effects of different footwear and surfaces on impact shock, but the majority of these have examined subjects whilst running. As this thesis is only concerned with impact shocks during walking, studies reporting running will be omitted here. Light et al. (1980) reported the effects of different shoe soles on axial impact shock during walking at 5.3 km.h\(^{-1}\) (1.47 m.s\(^{-1}\)) using both bone mounted and skin mounted accelerometers. For all the shoes worn, the acceleration values at heel contact ranged between 2 to 8 g, and lasted for 15-25 ms. For the hard heel on a hard surface, there was a rapid deceleration of the shoe heel, a value of 5 g at foot contact which diminished to 0.5 g at the head 10 ms later thus indicating, the harder the shoe and surface, the greater the impact shock. Studies have also shown that impact shock is greater walking barefoot than walking shod. Voloshin (1988) reported axial impact accelerations of 1.2 to 1.5 g barefoot and 0.7 to 0.9 g shod. Lafortune (1991) also reported greater axial impact accelerations walking barefoot (3.3 g) compared to shod (1.71 g), but this was using a bone mounted accelerometer. These studies show that for able-bodied gait, there is a large discrepancy in axial accelerations reported. This could
be due to the accelerometer mounting (skin or bone), accelerometer placement and the type of footwear worn.

3.3.1.5 The effect of walking speed on impact shock.

Many studies have looked at the effects of increased running speed on impact shock, but few have reported the effects of increased walking speed. Shorten et al. (1992) reported an increase in peak leg acceleration as running speed increased. Clarke et al. (1985) suggested that faster running is accompanied by an increase in musculoskeletal stress and therefore it is necessary to look at the effect of increased running speed on the musculoskeletal stress incurred during each step. This study found that for an increase in running (from 3.35-5.35 m.s\(^{-1}\)), there was a 34\% increase in tibial shock for every 1.0 m.s\(^{-1}\) increase in running speed. Knee flexion angle was also found to increase and stance time decrease with speed. At all speeds, a variability of ± 9\% was reported.

Hamill et al. (1995) looked at impact shock and stride frequency relationships. Increases in impact shock can result from an increase in running speed (Hamill et al., 1983) or increasing stride length (McMahon et al., 1987). They found that for a constant running speed, but by increasing stride length (SL) and stride frequency (SF), as SL increased, impact shock increased. This study concluded that higher magnitude or more frequent shocks will place greater stress on skeletal structures increasing the risk of injury or degenerative joint disease. Impact shock was not minimised at the preferred SF, so the runners did not choose the optimum SF in order to reduce shock. Thus increases in walking speed (as an increase in SL and SF) will result in greater impact shocks. By increasing walking speed, increased and more frequent shocks will put stress on the skeletal structures. This fact will have implications for lower limb amputees who...
generally choose to walk more slowly than the able-bodied (Craik et al., 1985; Lewallen et al., 1985; Engsberg et al., 1991; Hermodsson et al., 1994) in that they will experience lower and less frequent impact shocks walking at their own natural cadence than they are likely to experience walking at the natural cadence speed of able-bodied subjects.

3.3.2 Joint degeneration.

3.3.2.1 Joint degeneration mechanisms.

Loads resulting from repeated impacts can result in overuse injuries and/or joint degeneration (Simon et al., 1972; Radin et al., 1973; Voloshin and Wosk, 1982). Animal studies have shown repetitive impact loading can lead to osteoarthritis and reduction in the shock absorbing capacity of joints (Radin et al., 1972). This has never been established in humans but investigators have suggested repetitive loading of the skeletal structure during able-bodied gait is linked with the progression of osteoarthritis (Radin et al., 1972; Radin, 1975; Radin et al., 1975). Voloshin (1988) stated that joint degeneration is due to cyclic loading rather than frictional forces. Higher shock waves experienced when increasing speed from walking to running results in more cumulative damage. The natural cause of degenerative joint disease is disputed. Radin et al. (1982) stated that cyclic loading of the joint resulted in gradual fracture of the subchondral bone trabeculae. The fractures heal causing thickening and stiffening of the subchondral bed, reducing efficiency as a shock absorber. The hitherto protected joint cartilage is now subjected to increased dynamic stresses from everyday activity, and suffers gradual fatigue-failure which ultimately leads to the destruction of the tissue. The more rigid the walking surface, the more rapid and severe the process (Radin et al., 1982). However, this study was performed on sheep and it is not known whether the theory can be transferred to humans. Freeman (1975) suggested that the articular cartilage was the first to be injured in the early stages of joint degeneration, whilst Dekel and Weissman
(1978) stated both the articular cartilage and subchondral bone are injured at the same time. Which hypothesis is correct is not known, but it is generally accepted that fatigue-failure of shock absorbing joint tissues is directly linked to the loading of the cyclic force waves on the locomotor system.

\[ a_1 = \frac{(F_a - F_b)}{m_1} \]

\[ a_2 = \frac{F_b}{m_2} \]

Figure 3.6 – Free body diagram of chain of structure for ‘a’ at the heel to ‘a2’ at the jaw

Where \( t \) is the time at peak acceleration for each point. \( a = \) acceleration.

3.3.2.2 Shock attenuation in able-bodied gait.

The amount of impact shock attenuated by the body during locomotion can be measured by accelerometers. Shock attenuation measurement can provide an insight into whether there is a clinical problem at one or more joints, such as joint degeneration. By using a lightweight accelerometer attached to the lower limb and one attached to a bite
bar, the amount of shock attenuated prior to reaching the head has been quantified. Studies have looked at accelerations at the head either by using a bite bar with accelerometer attached (Light et al., 1980) or an accelerometer placed on the forehead (Wosk and Voloshin, 1981; Voloshin and Wosk, 1982; Forner et al., 1995). To protect the head, head accelerations need to be minimal, and the head stabilised to avoid movement in the visual field (Pozzo et al., 1991). A stable head also provides a stable gravitational reference for the vestibular system during locomotion (Pozzo et al., 1989). By the time the impact shock reaches the head, the peak acceleration is reduced. This is due to the damping of the signal through the skeletal structure between the foot and the head by the structures of the body that absorb shock, including the intervertebral discs, hyaline cartilage lining of the joints, muscle action, heel pad, and pronation and flexion of the joints (Forner et al., 1995) (Figure 3.6).

3.3.2.3 Joint pain and shock attenuation.

Studies have previously investigated asymmetrical shock attenuation in patients with joint or back pain. On investigating subjects experiencing back pain, Voloshin and Wosk (1982) reported that non-injured subjects attenuated 20% more shock than those injured. They thus concluded that the reduced shock absorbing capacity of the human musculoskeletal system from the femoral condyle to the forehead correlated with the presence of low back pain. Similar amplitudes were found at the head, but those with back pain had a reduced amplitude at the femoral condyle. In order to protect the head from high accelerations in a reduced shock absorbing capacity musculoskeletal system, the initial impact shock at foot strike had to be reduced. Therefore, because of poor attenuation capacity in back pain patients, the incoming impact shocks have to be reduced, possibly by a change in the walking pattern of the patients. However, Nigg et al. (1995) commented on the results of this study, concluding that in a group such as
this, pain influences the gait pattern, and as a result, the variables measured. Consequently, conclusions about cause and effect cannot be made.

Wosk and Voloshin (1981) also investigated a group of subjects at high risk from degenerative joint disease. A number of points were found from this and the former study (Voloshin and Wosk, 1982). The walking pattern may change in order to protect the head from overloading by relatively high shock waves as a result of an insufficient musculoskeletal shock absorbing capacity (Voloshin and Wosk, 1982). Poor shock attenuation in one knee probably leads to changes in a walking pattern in order to prevent overloading of the above-knee shock absorbers by trying to reduce the absolute value of the incoming shocks (Wosk and Voloshin, 1981). These studies concluded that every patient who suffers from degenerative joint disease has an insufficient shock absorption capacity of the knee or knee-forehead system and if this is seen in a subject, then there is a strong reason for suspecting that degenerative joint diseases will develop later in the patient. This study concluded that by using accelerometers, one could distinguish with 90% confidence between healthy subjects and subjects with reduced capability of natural shock absorbers. Even under normal physiological conditions, the continuous repetitive onslaught of the incoming shock waves resulting from heel strike tends to cause a progressive weakening of the natural shock absorbers and may result in subchondral bone microfractures (Radin et al., 1980) which may then lead to articular cartilage degeneration and osteoarthritis.

Radin et al. (1991) reported that subjects with mild knee joint pain had a 37% higher loading rate of vertical GRF associated with heel strike. The study looked at a group of preosteoarthritic patients (because of mild intermittent activity related knee joint pain) and a group of healthy subjects and found that the 2 groups had similar gait
patterns including cadence, walking speed, terminal stance phase knee flexion, peak swing angular velocity and overall shape of the vertical GRF, but, there were significant differences between the 2 groups within a few ms of heel strike (transient force at heel strike). In the knee pain group, the heel hit the ground with a stronger impact of 1.97 g (1.63 g healthy subjects) thus disagreeing with the works of Voloshin and Wosk. The initial vertical ground reaction force impact peak (Fz1) was found to be 1.12 BW in the knee pain group and 1.1 BW in the healthy group. An accelerometer was placed on the side with knee pain only, and on the healthy subjects’ right leg. There was a faster downward velocity of the ankle just before HS in the knee pain group, with a larger angular velocity of the shank and greater loading rate of 67.6 BW/s compared to a loading rate of 47.9 BW/s in the healthy group. Shock attenuation was also different between the thigh and shank accelerations indicating a measure of shock attenuated at the knee. A significant difference between the two was seen, i.e. some shock was attenuated at the knee (0.31 g), but in the knee joint pain group, only 0.02 g was attenuated. Thus whilst similar kinematic and GRF patterns may be seen between groups suffering from joint pain and those with no pain, accelerometry has been shown to be a tool able to measure differences between the two, such as impact shock and shock attenuated. However, there are disagreements as to the mechanism used - whether patients suffering from joint pain or degeneration reduce impact shocks to protect the limb (Voloshin and Wosk, 1981; Wosk and Voloshin, 1982), or the increased impact shocks seen in the painful limb cause the pain and degeneration (Radin et al., 1991). One theory could be that as the knee pain group in the study of Radin et al. (1991) was preosteoarthritic and patients were chosen because of mild intermittent knee pain, the increased impact shocks contributed to the onset of knee pain. Once osteoarthritis is established, the patient may be trying to protect the painful limb that can no longer
attenuate as much shock as a healthy limb, by reducing impact shocks as in the patients seen in the studies of Voloshin and Wosk.

Studies have generally concluded that joint pain from mechanical causes can be brought about by a combination of repetitive impulsive loading and oscillatory movement (Radin et al., 1973). Studies have established there is often an impulsive foot-ground reaction at heel strike (Simon et al., 1981; Winter, 1987), but only in a third of the population (Radin et al., 1986). Those who are impulsively loading their joints and may be provoking joint damage and possibly osteoarthritis.

3.3.2.4 The effect of walking speed on shock attenuation.

As the GRF has been found to increase with increasing walking speed (Nilsson et al., 1985), and the velocity of the foot will have to reduce much more rapidly after impact, impact shock increases with increasing speed. As the body adapts to protect the head from accelerations, increased speed results in increased shock attenuation (Hamill et al., 1983). Hamill et al. (1995) investigated the relationship between stride frequency and shock. They found that shock attenuation was not minimised at preferred stride frequency. Therefore, optimal stride frequency was not chosen in order to minimise shock and thus the body must minimise the magnitude of shock at the head by other active mechanisms.

3.3.2.5 Summary of impact shock and shock attenuation in able-bodied gait.

Considerable research has been done in the area of shock and shock attenuation during walking and running. Placement and positioning of the accelerometer on the shank greatly affects results and have been varied between studies making them difficult to compare. Hard soled shoes, hard walking surfaces and increased walking speeds have
all been found to increase impact shock. An inability to attenuate the shock fully from
the shank to the head has been reported in able-bodied subjects suffering from joint
degeneration. A mechanism employed in order to protect the injured leg has been to
reduce incoming impact shocks. Whilst this has been reported for osteoarthritis
sufferers, a conflicting study has been reported for preosteoarthritic patients. The latter
study has reported that whilst shock attenuation is reduced in the affected limb,
incoming impact shocks are increased on that limb. This repeated increased impact
shock on the limb prior to osteoarthritis may be the mechanism that leads to joint
degeneration.

3.3.3 Impact shock and shock attenuation in unilateral amputees.

3.3.3.1 Joint degeneration in amputees.

Alteration of the able-bodied walking pattern over many years may result in
degenerative changes to weight-bearing joints (Burke et al., 1978). This is of particular
concern to the amputee who needs to adapt to the partial loss of a limb by altering his or
her gait pattern in terms of loading characteristics (Breakey, 1976; Hershler and Milner,
1980; Clark and Zernicke, 1981). Studies have suggested that altered gait biomechanics
resulting from amputation have been responsible for increased incidences of
osteoarthritis in the intact limb (Burke et al., 1978). It has been reported that 19 to 71% of unilateral lower limb amputees taking part in studies experience pain in their intact limb (Hungerford and Cockin, 1975; Burke et al., 1978; Skinner and Effeney, 1985;
Hurley et al., 1990). From questionnaire and X-ray investigation, Burke et al. (1978)
reported that there was a 41% incidence of osteoarthritis in the intact knee of below-
knee amputees compared to their residual knee. The residual limb exhibited
osteoporosis (due to disuse atrophy). Half the amputees questioned reported severe
backache. The reason for this was not found but it was stated that this was not due to
disc degeneration. An explanation for this backache could be unequal leg length and
thus excessive mediolateral trunk motion. The prosthetic limb is usually made slightly
shorter than the intact limb to facilitate the forward swing phase in walking. This
inequality may also contribute to increased stress on the intact limb (Dixon and
Campbell-Smith, 1969). The excessive ML trunk movement often seen in the gait of
lower limb amputees as they “hike” the hip could be a contributing factor to the low-
back pain (LBP) frequently reported by unilateral amputees. Thus, whilst lower limb
amputees report LBP as well as intact limb pain, the LBP may be due to altered gait
mechanics. Intact limb pain could possibly result from joint degeneration due to
repetitive increased loadings (Radin et al., 1991) during walking. No study has looked at
this aspect of amputee gait.

3.3.3.2 Impact shock and shock attenuation in amputee gait.

Unilateral lower limb amputees load their intact limb more and their prosthetic
limb less during walking than do able-bodied persons (Engsberg et al., 1991; 1993). If
the greater loading is responsible for incidences of joint pain in the intact limb of
unilateral amputees, and joint degeneration means less shock is attenuated, the amputees
may be trying to protect their intact limb by reducing incoming impact shocks in a
similar way to the patients in the studies of Voloshin and Wosk (1981;1982). Only one
study has looked at impact shock in unilateral below-knee amputees. Therrien and
Prince (1998) placed accelerometers at the ankles and hips of 6 below-knee amputees
and measured impact shock and shock attenuation as they ran over a force platform.
Although no significant differences were found, average impact shock at the ankle was
slightly lower for the prosthetic limb than the intact limb, with a wide variability
between subjects. These results would tend to agree with the theory supported by Radin
et al. (1991), although there was a minimal difference between the two limbs (6.21 g prosthetic limb, 6.62 g intact limb), lack of homogeneity between subjects and whether they suffered from joint pain was not reported. Shock attenuation was calculated as a difference between shock measured at the ankle and shock measured at the hip. This value was slightly higher for the intact limb than prosthetic limb. The study concluded that impact shock transmitted to the hip is attenuated in both the prosthetic and intact limbs even if the materials and mechanisms responsible are different. However, a lack of homogeneity between subjects, non-comparable calculations for shock attenuation and no individually reported data make this study difficult to interpret.

It now seems that there is a possible theory as to what may be happening in amputee gait. For amputees not reporting any intact limb pain or joint degeneration problems, the magnitude of impact shocks on each limb is expected to be asymmetrical as seen in investigations of ground reaction forces and step times (Breakey, 1976; Engsberg et al., 1991) with greater impact shock on the intact limb. More shock will thus need to be attenuated when stepping onto the intact limb compared to the prosthetic limb. If this is shown to be true, then it may lead to the high incidence of joint degeneration on the intact limb which has previously been reported for below-knee amputees. This would be in agreement with the findings of Therrien and Prince (1998) and the studies of preosteoarthritic patients by Radin et al. (1991). The mechanism of increased loading and high impact shock magnitude experienced on the intact limb of unilateral amputees may lead to joint pain and/ or joint degeneration. If this is true, from the studies of Voloshin and Wosk (1981; 1982), if joint degeneration is present in the intact limb of unilateral amputees, the intact limb will have a reduced capacity for shock attenuation. Then, in order to prevent excessive head accelerations (Pozzo et al., 1991) and protect the intact limb, incoming impact shocks will need to be reduced. Thus,
impact shock and shock attenuation asymmetry will still be present, but there will now be less impact shock experienced by the intact limb compared to prosthetic limb. Shock attenuation on the prosthetic limb will be partly dependent on the shock absorbing properties of the prosthesis and thus, with modern materials, incoming impact shocks and shock attenuation may be seen to be greater on the prosthetic than on the intact limb.

As no studies have yet looked at this aspect of joint degeneration and intact limb pain in unilateral amputees, and only one study has reported shock attenuation from ankle to hip in below-knee amputee runners, comparative data are lacking. It may be that for new amputees with no intact limb pain or joint degeneration, greater amounts of impact shock begin to overload the intact limb resulting in joint degeneration. For established amputees or highly active amputees who already suffer from intact limb pain and a reduced shock attenuation capacity, they need to reduce the incoming impact shocks. More research needs to be done in this area before greater understanding of the mechanisms of amputee intact limb pain/joint degeneration can be obtained, and steps taken to prevent it.

3.3.3.3 Adaptation to shock absorption.

The body alters (amongst other mechanisms) joint stiffness and segment kinematics and places body segments in positions that are more ready to attenuate shock (Hamill et al., 1995) such as subtalar pronation and increased knee flexion. Knee angle at foot-ground contact is believed to have a considerable influence on the body’s ability to attenuate impact loading and transmit shock during locomotion. Lafortune et al. (1996) looked at transmission between the shank and the head and found that knee flexion caused a considerable reduction in effective axial stiffness which absorbed
shock attenuation. The stiffness of the lower limb that contributes to the regulation of shock transmission has been found to reduce with knee flexion angles (Greene and McMahon, 1979). This study queried an 80% increase in shock attenuation resulting from a slight increase in knee flexion that reduced the vertical stiffness by only 18%. However, for amputees, due to the loss of an ankle joint, or ankle and knee joint, there is reduced prosthetic knee and ankle flexion. As “the action of knee flexion has a direct effect on the transmission of shock waves and hence on the magnitude of the signal at the level of the head” (Valiant, 1990, p244), this could result in less shock absorption for amputees. When greater demands are placed on amputees walking, such as an increase in speed, the amputees will either have to adapt to this increase in shock, or incur joint pain and risk of joint degeneration in later life. However, no study found has looked at this. Adaptation in order to reduced impact shock on the prosthetic or intact limb may be seen in differing joint kinematics (reviewed in section 3.4).

3.3.3.4 Summary of impact shock and shock attenuation in amputee gait.

There is a lack of previous research on shock and shock attenuation in amputee gait. Only one study measured impact shock in below-knee amputees during running and reported similarities between limbs with a slightly higher mean impact shock on the intact than on the prosthetic limb. However, there was wide variability between subjects and individual data were not reported. Results for non-amputee patients with joint pain have led to the rise of a possible theory. Those amputees not experiencing joint pain in their intact limb may have greater impact shocks on their intact limb which could be the mechanism responsible for leading to joint pain and or joint degeneration in later life. Those amputees experiencing joint pain in their intact limb will be less able to attenuate shock in that limb and as a consequence, reduce incoming impact shocks. Adaptation to this reduced shock absorption capacity may be seen in differing joint kinematics such as
increased knee flexion during mid-stance to help absorb shock. When greater demands are placed on the amputees such as increased walking speed, they will have to adapt by either altering joint kinematics or use another method to try to reduce the increasing impact shocks. As a result, any impact shock or shock attenuation asymmetry seen between the prosthetic and intact limbs is expected to increase with increased walking speed.

3.3.4 Summary of impact shock and shock attenuation.

Studies have shown that in able-bodied subjects, the heel strike transient, or axial impact shock resulting from the deceleration of the moving leg has been implicated as a possible cause of degenerative joint disease in the lower limb. Axial impact shock, typically between 0.7 and 2 g for shod walking, has been found to be affected by shoe sole, walking surface and accelerometer placement. The amount of shock attenuated before reaching the head has previously been reported as an indicator of joint problems. Individuals experiencing back or knee pain have been reported to have attenuated 20% less shock than healthy subjects and reduce incoming impact shocks. This has implications for both above- and below-knee amputees as the amount of shock absorbed through the prosthetic limb will be mainly dependent on the mechanics of the prosthesis. Amputees have previously reported greater amounts of joint degeneration on their intact limb than on their prosthetic limb and increased intact limb axial shocks at heel strike. However, those amputees experiencing joint degeneration may exhibit reduced shock attenuation and incoming impact shocks on their intact limb. As it has been previously reported that impact shock increases with increasing walking and running speeds in able-bodied subjects, it is expected that this will also be true for lower limb amputees, although no study has been found to look at this expectation. Thus, if the amputee exhibits asymmetrical impact shock and shock
attenuation during walking, it is thought that this asymmetry will also increase with walking speed. By investigating how much shock is experienced and then attenuated on both the prosthetic and intact limbs of amputees, further understanding of the mechanisms of joint degeneration and intact limb pain in unilateral amputees may be gained.
3.4 Kinematics.

A smooth passage of the centre of mass over the support foot is essential for efficient walking. For these walking movements to be smooth, the movement of the lower extremities must follow specific patterns (Sutherland et al., 1994). During the gait cycle, events can be subdivided into phases to explain how the lower limb joint angles and step variables contribute to the general walking pattern seen by able-bodied persons.

3.4.1 Kinematics of able-bodied gait.

Kinematic analysis of locomotion has previously been described in detail to explain spatial, temporal, linear and angular motion characteristics of the lower limb. These observations have concluded that able-bodied walking patterns appear constant and exhibit distinct and recognisable traces (Sutherland et al, 1994).

3.4.1.1 Temporal variables of gait.

A full gait cycle is defined as initial foot contact to contact again on the corresponding limb. Commonly, events occurring in the gait cycle are termed in percentages (e.g. for stance, heel strike is 0%, toe off 100%) rather than time elapsed, normalising the data. Any temporal gait abnormalities can thus be compared across studies. In able-bodied walking, this is divided into stance phase from heel strike (HS) to toe off (TO) (60% of the cycle) or swing phase from TO to HS (40%) (Milliron and Cavanagh, 1990). The percentages are dependent on speed of locomotion (Whittle, 1991), the stance phase becoming shorter and swing phase longer with speed (Murray, 1967). During the stance phase, there is a period of double support where both feet are in contact with the ground accounting for about 10% of the gait cycle or single support where only one foot is in contact. The main events of the gait cycle (Figure 3.7) have implications for both understanding able-bodied gait and pinpointing gait abnormalities.
From HS through to foot flat (Figure 3.7) the limb progresses through loading or ‘weight acceptance’ with the corresponding limb nearing TO, marking the end of the double support period at about 0-12% of the gait cycle. The limb is now supporting the entire body weight with the contralateral limb in swing phase (12-50% of the gait cycle). From 50-62% of the cycle, double support is again present as the limb in stance phase moves towards TO. After TO, the swing phase begins (62-75%) where the foot is being cleared and the contralateral limb is in single support. The swing phase continues for the rest of the cycle until the limb is prepared for HS again (Sutherland et al., 1994). Linear measurements of the gait cycle have been described in terms of cadence, step length and stride length. Cadence is defined as the number of steps in a certain duration i.e. steps per minute. Step length is usually defined by the distance between the heels of both feet during double support, stride length the distance between successive foot strikes of the same foot (Figure 3.8).
Figure 3.8- Temporal gait parameters (Whittle, 1991, pp55).

Other variables such as step time (the time taken from heel strike on one foot to heel strike on the next foot), swing time (time taken from toe off to heel strike on the same foot) and stride time (the duration of the stride) have been used in previous studies to quantify gait kinematics either as a percentage of the gait cycle or duration (Table 3.2).

Table 3.2 - Temporal variables during able-bodied walking.

<table>
<thead>
<tr>
<th>Author</th>
<th>Velocity (m.s⁻¹)</th>
<th>Stance time (s)</th>
<th>Swing time (s)</th>
<th>Single support time (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Andriacchi et al. (1977)</td>
<td>0.5</td>
<td>-</td>
<td>0.80</td>
<td>1.80</td>
</tr>
<tr>
<td></td>
<td>0.9</td>
<td>-</td>
<td>0.55</td>
<td>0.80</td>
</tr>
<tr>
<td></td>
<td>1.2</td>
<td>-</td>
<td>0.50</td>
<td>0.60</td>
</tr>
<tr>
<td></td>
<td>1.5</td>
<td>-</td>
<td>0.45</td>
<td>0.48</td>
</tr>
<tr>
<td>Nilsson et al. (1985)</td>
<td>0.4</td>
<td>-</td>
<td>0.51</td>
<td>1.25</td>
</tr>
<tr>
<td>Sutherland et al. (1988)</td>
<td>1.5</td>
<td>-</td>
<td>0.39</td>
<td>-</td>
</tr>
<tr>
<td>Murray et al. (1983)</td>
<td>0.78</td>
<td>0.96</td>
<td>0.52</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1.1-1.2</td>
<td>0.65</td>
<td>0.41</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1.5-1.7</td>
<td>0.49</td>
<td>0.38</td>
<td>-</td>
</tr>
</tbody>
</table>

It has been suggested that these parameters of the human gait cycle are only comparable when limited to free speed walking on level ground (Sutherland et al., 1994) and other variables such as fixed or slow walking velocities or neurological patterns can change these relationships markedly, making comparisons with able-bodied data difficult.
3.4.1.2 The effect of speed on temporal variables of gait.

A few studies have reported the effects of speed on temporal variables of able-bodied walking gait (Table 3.3). Nilsson et al. (1985) reported that the overall cycle duration and support times decreased curvilinearly with increasing velocity. Andriacchi et al. (1977) reported reductions in swing and single support times with increases in walking speed from 0.5 to 1.5 m.s\(^{-1}\). Murray et al. (1983) also found stance and swing time duration to decrease with increasing walking speed.

3.4.1.3 Spatial variables of gait.

Many studies have reported lower limb joint angles during the gait cycle, either two-dimensional (Whittle, 1991; Smith, 1993; Harris et al., 1994) or three-dimensional (Apkarian et al., 1989; Gant et al., 1991; Lafortune et al., 1992). The effects of rotation and abduction/adduction angles of the lower limb are not included in two-dimensional kinematic analysis where major effects of abnormal gait could be illustrated. Whilst a full kinematic gait analysis needs to include three-dimensional measurements of joint angles, the measurement, reconstruction and interpretation of these data are more complicated than for two-dimensional measurements. Therefore, many studies have adequately reported two-dimensional lower limb joint angles only during able-bodied walking. In terms of joint angles, the hip, knee and ankle joints of able-bodied subjects have all been reported to follow their own distinct and recognisable patterns (Figure 3.9).

The Ankle Joint.

In able-bodied walking gait, at heel strike, the ankle is near the neutral position with the heel slightly inverted and the foot slightly supinated, thus the ankle is seen to
plantarflex, reaching a maximal of 15-25° from neutral (Whittle, 1991; Harris et al., 1994; Rose & Gamble, 1994).

During the interval between heel strike and foot flat, as the foot lowers to the ground, the anterior tibial muscles then contract eccentrically and the tibia inwardly rotates causing the foot to collapse into pronation. This usually occurs at 8% of the gait cycle in walking (Whittle, 1991) and helps the body to absorb initial shock. Mid-stance occurs around 30% of the gait cycle (Whittle, 1991) and illustrates the period from “foot flat” to “heel off”. During this period, the movement of the ankle and foot is characterised by forward rotation of the tibia about the ankle joint from plantarflexion to dorsiflexion. The tibia then starts to rotate externally, moving the foot by means of the subtalar joint out of pronation. Peak ankle dorsiflexion, about 10-20°, (Whittle, 1991; Harris et al., 1994) is reached around the same time as heel off occurs. The ankle then begins to plantar flex. During the swing phase, the ankle is in a neutral position to help the toes clear the ground.
This requires a small contraction of the anterior tibial muscles, although most of the action required for clearance relies on flexing the knee which effectively shortens the leg.

The knee joint.

The pattern of the knee joint is distinct as the knee plays an important role in both absorbing shock and allowing the foot to clear the ground during the swing phase. In order to stabilise the leg before heel strike, the knee extends and the knee flexors, hamstrings, contract eccentrically at the end of extension in order to act as a braking mechanism and prevent hyperextension. At heel strike, the knee tends to be only slightly flexed. Following heel contact, the knee increases flexion in order to help absorb shock, known as the ‘cushioning’ phase where between 10-20° of flexion is seen (Whittle, 1991; Harris et al., 1994). The knee starts to flex after “heel off”, increasing flexion at “toe off”. Knee flexion continues into the swing phase as a result of the continued forward movement of the femur. The flexion of the knee during swing is passive and results largely from hip flexion. The leg acts as a double pendulum, as the hip flexes it brings the knee upwards and therefore, no muscle contraction around the knee is needed. Here, peak knee flexion usually increases to 60-70% during walking (Whittle, 1991; Harris et al., 1994). Towards the end of swing, as the speed of hip flexion decreases, the knee starts to extend. Muscle activity is needed at the end of the swing phase in order to prevent hyperextension of the knee resulting from an uncontrolled continuation of the pendulum movement. Here, the hamstrings act as knee flexors to oppose increased knee extension, and the leg is once again ready to initiate heel strike for the next stride.

The hip joint.

In addition to providing a braking force prior to heel strike, the hamstrings also initiate extension of the hip. The hip begins to extend slightly at heel strike, extending
more rapidly as the hip extensors, the hamstrings, contract. The hip is usually flexed at the
time of foot flat and continues to extend by increased contraction of the hip extensors.
Between heel off and “toe off”, the hip reaches peak extension (10-20°, Whittle, 1991) and
then starts to flex again. Peak hip extension is reached just before toe off, and thus the hip
is starting to flex again at toe off, peak flexion being around 30° (Whittle, 1991). The
hamstrings act eccentrically to help bring hip flexion to a stop at the end of the swing
phase, then initiate hip extension ready for the next heel contact.

3.4.1.4 The effect of speed on spatial variables of gait.
Previous studies have commonly reported able-bodied joint angles whilst walking
at a natural cadence. Discrepancies in “natural” walking speed may account for small
differences in joint angles across studies. Questions pertaining to the validity of recording
gait variables at specific speeds rather than the individual’s own natural walking speed
have not been fully answered (Boonstra et al., 1993). Walking speed is a basic gait
parameter which, when measured objectively, can be useful in characterising an
individual’s walking ability (Boonstra et al., 1993). However, few studies have
investigated how gait changes when increased demands such as increased walking speed
are made on the individual. Two studies have shown that able-bodied subjects adapt to
increasing walking speeds by changing knee joint function (Nilsson et al., 1985; Holden et
al., 1997). At the slower walking speeds (25 and 50% of natural walking speed), little knee
joint flexion is seen. As subjects walk faster, they increase knee flexion (Holden et al.,
1997). Nilsson et al. (1985) also reported increases in joint angles with increasing walking
speed. Hip joint angles increased by 11° for walking from 1.0 to 1.5 m.s\(^{-1}\). Knee joint
flexion increased by 10° from slow walking speeds and levelled off at walking velocities
above 1.5 m.s\(^{-1}\). Ankle dorsiflexion angle also increased with walking speed (by 27°),
becoming greater than for running at velocities above 1.5 m.s\(^{-1}\).
These studies illustrate that humans can choose to adapt to an increase in speed by altering the frequency (decreasing stance and swing time) or amplitude (increasing joint angle) of leg movement. However, if there is any asymmetry present, such as in amputee gait, increases in joint amplitude may force an increase in spatial asymmetry. This is one measure by which any deviations from able-bodied gait can be assessed.

3.4.1.5 Variability and asymmetry of able-bodied gait.

Generally, kinematic aspects of gait tend to be less variable than kinetic and EMG patterns (Winter, 1987). The movement outcomes of walking are affected to a great extent by the environment and physical parameters of bipedal gait and must be achieved within specific margins of error. This is reflected in the variability, ±15-52% hip, ±4-23% knee and ±64-72% coefficient of variation for lower limb joint angles (Winter, 1987; Smith, 1993). Smith (1993) concluded that an average of ten subjects gave similar variability as the average of 1 subject over a number of trials (±13%, 11% and 56% for the hip, knee and ankle respectively) and that any variability over 40% was too much.

On looking at three-dimensional joint angles, Apkarian et al. (1989) showed only 1-2° variation between subjects for lower limb flexion/extension and abduction/adduction angles whilst internal/external rotation provided double the variation. This illustrates the difference in kinematic variability depending on whether two-dimensional or three-dimensional angles are reported.

3.4.1.6 Summary of kinematics of able-bodied gait.

Temporal and spatial variables of able-bodied gait have previously been well documented. These established "norms" have been a measure by which pathological gait
can be assessed. Temporal aspects of kinematic analysis have shown the stride time, stance time (60% of the gait cycle) and swing time (40% of the gait cycle) during walking to decrease with increasing walking speed. Able-bodied values for two-dimensional hip, knee and ankle joint angles have been agreed on throughout studies. Whilst the majority of joint angles have been reported for natural cadence walking, a few studies have reported increases in joint angles with increasing walking speed. Those that have, reported increases in knee joint flexion and ankle dorsiflexion with speed. Thus, temporal and spatial variables of able-bodied gait have been previously quantified, and any deviations from these “norms” as seen in pathological gait can be assessed.
3.4.2 Kinematics of amputee gait.

Below-knee amputee gait differs from that of above-knee amputees largely by the fact that below-knee amputees have the ability to flex and extend the knee naturally. Therefore, below-knee amputees may exhibit little difference in movement of the centre of gravity to able-bodied gait, and having partial to full knee action, have similar gait to that of an able-bodied person (Sanders et al., 1953). Whilst there have been studies documenting aspects of amputee gait, many have been concerned with stance time, step length and velocity during the swing phase, and few have reported joint angles.

3.4.2.1 Temporal values of gait.

Robinson et al. (1977) noted an asymmetric gait pattern in below-knee amputees with the subjects taking larger steps more quickly with the prosthetic limb compared to the intact limb. Breakey (1976) found that for below-knee amputees, the stance phase in gait was significantly shorter for the prosthetic limb when compared with the intact limb, and the stance phase of able-bodied subjects (Table 3.3). The same was found for above-knee amputees (Table 3.4), with stance and swing times differing with different knee mechanisms used.

Table 3.3 - Temporal variables for below-knee amputee gait (* denotes study using children).

<table>
<thead>
<tr>
<th>Author</th>
<th>Velocity (m.s⁻¹)</th>
<th>Prosthesis type</th>
<th>Limb</th>
<th>Stride time (s)</th>
<th>Stance (% of cycle)</th>
<th>Swing (% of cycle)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colborne et al. (1992)</td>
<td>1.04</td>
<td>SACH</td>
<td>prosthetic</td>
<td>1.19</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1.10</td>
<td>SEATTLE</td>
<td>prosthetic</td>
<td>1.19</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Engsberg et al. (1990)*</td>
<td>-</td>
<td>-</td>
<td>prosthetic</td>
<td>-</td>
<td>58</td>
<td>42</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-</td>
<td>intact</td>
<td>-</td>
<td>62</td>
<td>38</td>
</tr>
<tr>
<td>Breakey (1976)</td>
<td>-</td>
<td>-</td>
<td>prosthetic</td>
<td>-</td>
<td>57</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-</td>
<td>intact</td>
<td>-</td>
<td>63</td>
<td>37</td>
</tr>
<tr>
<td>Craik et al. (1985)</td>
<td>0.42</td>
<td>-</td>
<td>intact</td>
<td>-</td>
<td>74</td>
<td>26</td>
</tr>
</tbody>
</table>
Table 3.4 - Temporal variables of above-knee amputees during walking.

<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Velocity (m.s(^{-1}))</th>
<th>Prosthesis Type</th>
<th>Single Support (s)</th>
<th>Swing (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Murray et al. (1981)</td>
<td>-</td>
<td>Prosthetic</td>
<td>0.43</td>
<td>-</td>
</tr>
<tr>
<td>Zuniga et al. (1972)</td>
<td>-</td>
<td>Intact</td>
<td>0.58</td>
<td>-</td>
</tr>
<tr>
<td>James and Oberg (1973)</td>
<td>-</td>
<td>Prosthetic</td>
<td>0.49</td>
<td>-</td>
</tr>
<tr>
<td>Suzuki et al. (1997)</td>
<td>-</td>
<td>Intact</td>
<td>0.61</td>
<td>-</td>
</tr>
<tr>
<td>Hale (1990)</td>
<td>0.98</td>
<td>-</td>
<td>0.58</td>
<td>-</td>
</tr>
<tr>
<td>Stein and Flowers (1987)</td>
<td>-</td>
<td>Conventional Prosthetic</td>
<td>0.69</td>
<td>-</td>
</tr>
<tr>
<td>Murray et al. (1983)</td>
<td>0.78</td>
<td>Prosthetic</td>
<td>0.94</td>
<td>0.61</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intact</td>
<td>1.07</td>
<td>0.49</td>
</tr>
<tr>
<td>1.1-1.2</td>
<td></td>
<td>Prosthetic</td>
<td>0.95</td>
<td>0.53</td>
</tr>
<tr>
<td>1.5-1.7</td>
<td></td>
<td>Intact</td>
<td>1.04</td>
<td>0.45</td>
</tr>
<tr>
<td>1.1-1.2</td>
<td></td>
<td>Prosthetic</td>
<td>0.78</td>
<td>0.57</td>
</tr>
<tr>
<td>1.5-1.7</td>
<td></td>
<td>Intact</td>
<td>0.90</td>
<td>0.43</td>
</tr>
<tr>
<td>1.1-1.2</td>
<td></td>
<td>Prosthetic</td>
<td>0.72</td>
<td>0.49</td>
</tr>
<tr>
<td>1.5-1.7</td>
<td></td>
<td>Intact</td>
<td>0.79</td>
<td>0.43</td>
</tr>
<tr>
<td>1.1-1.2</td>
<td></td>
<td>Prosthetic</td>
<td>0.60</td>
<td>0.55</td>
</tr>
<tr>
<td>1.5-1.7</td>
<td></td>
<td>Intact</td>
<td>0.76</td>
<td>0.40</td>
</tr>
<tr>
<td>1.1-1.2</td>
<td></td>
<td>Prosthetic</td>
<td>0.55</td>
<td>0.45</td>
</tr>
<tr>
<td>1.5-1.7</td>
<td></td>
<td>Intact</td>
<td>0.64</td>
<td>0.30</td>
</tr>
</tbody>
</table>

3.4.2.2 The effect of speed on temporal variables of gait.

Murray et al. (1983) found that both stance and swing phase duration reduced with increasing walking speed but did not comment on the level of asymmetry. Isakov et al. (1996) also reported that amputee stance and swing time duration decreased with increased walking speed, but the level of asymmetry was not significantly affected.

However, this study only reported free and fast walking speeds. Any increases or decreases in amputee temporal gait asymmetry may have been missed in this study due to either only investigating two walking speeds, or using the amputees’ natural walking speeds which may vary between subjects. Amputees are known to exhibit asymmetrical walking patterns.
in terms of stance and swing phase duration. If walking speed is found to affect stance or swing phase duration more on one limb compared to the other, lower limb amputees could either reduce or increase gait asymmetry.

3.4.2.3 Spatial values of gait.

The main difference between below-knee and above-knee amputee gait is the additional loss of a fully functional knee joint (Whittle, 1991). Whittle (1991) illustrated the walking gait pattern of a below-knee amputee wearing a multiflex foot. Graphically, the joint angle patterns look similar to those of able-bodied subjects, but there are some differences.

![Figure 3.10 - Sagittal plane lower limb joint angles during a single gait cycle for a below-knee amputee subject walking. Hip (flexion positive), knee (flexion positive) and ankle (dorsiflexion positive) (Whittle, 1991, pp123).](image)

The ankle joint.

Amputee gait is affected by the motion of the prosthetic ankle. An artificial foot
deprives the user of the ability to plantarflex and dorsiflex the ankle (Whittle, 1991). Thus, plantar flexion in amputees is passive and results from the removal of load from the artificial foot mechanism, rather than active plantar flexion seen in able-bodied subjects (Whittle, 1991). Breakey (1976) described a typical gait pattern of the below-knee amputee in that there is a delayed placing of the foot flat after “heel strike” due to the lack of ability of the prosthetic ankle to plantarflex, reduced knee flexion in the stance phase due to the rigidity of the foot and its inability to dorsiflex, early heel-off followed by early toe-off and thus reduced duration of the stance phase and a reduced knee flexion in the swing phase. Smith (1990) on comparing the movements of the prosthetic and intact limbs for below-knee amputees using the SACH foot found that the kinematics of both the prosthetic and intact ankle joints for below-knee amputees were substantially different from those of able-bodied subjects. Thus, not only does the limited ankle movement on the prosthetic limb affect ankle ROM on that limb, but a compensatory mechanism is seen on the intact limb. Whittle (1991) found some plantarflexion and dorsiflexion for the prosthetic limb of an above-knee amputee wearing a flex foot (Figure 3.10). As with below-knee amputees, the amount of ankle range of motion is dependent on the controlling prosthetic mechanism.

The knee joint.

In below-knee amputees, knee angle at heel strike has been reported to be smaller on the prosthetic limb (4°) compared to the intact limb (12°, Culham et al., 1984). The residual knee has also been reported to show slightly less flexion during the cushioning phase of stance, 15° compared to the intact limb, 25° (Smith, 1990). If knee flexion was not reduced, there would be an active raising of the centre of gravity, dissipating more energy (Breakey, 1976). Knee flexion at toe-off is also reduced in below-knee amputees in order to compensate for poor ankle plantar flexion and thus prevent the centre of mass
from excessively lowering. Maximal knee flexion during swing has also been found to be less on the residual limb compared to the intact limb (70°) depending on prosthesis worn (45° with a SACH foot, 60° with an ESCF foot, Smith, 1990). Colbome et al. (1992) also reported there was 6° more swing knee flexion on the intact limb wearing a SEATTLE foot compared to a SACH foot on the intact limb.

The amount of knee flexion/extension depends on the length of the stump. An average to long stump gives the amputee the ability to make use of the controlled flexion-extension-flexion sequence of knee action required in a smooth path of the centre of gravity and absorbing shock during stance (Radcliffe, 1994). Therefore, the below-knee amputee exhibits less knee flexion during the gait cycle than able-bodied subjects (Figure 3.10).

![Figure 3.10 - Joint angles during a single gait cycle for an above-knee amputee subject walking. Hip, knee and ankle (flexion positive) (Whittle, 1991, pp122).](image)

The main cause of above-knee amputee’s gait problems is the absence of a fully flexing knee joint. During the stance phase, the action of the knee shows greatest deviation
from able-bodied gait (Whittle, 1991). After heel strike, knee flexion during the
'cushioning' phase is absent in above knee amputee gait (Figure 3.11), thus showing that
shock usually absorbed by knee flexion must be absorbed by some other mechanism.

Due to the absence of a knee joint and depending on the type of prosthesis used,
above knee amputees could lack any ability to resist an external flexion moment at the
knee and must walk with the knee in full extension (Whittle, 1991), leading to excessive
oscillation of the pelvis, excess movement of the centre of gravity and therefore greater
energy consumption (Whittle, 1991). Another problem associated with above-knee
amputee gait is that the individual may be unable to resist knee extension in late swing
resulting in hyperextension (Radcliffe, 1994). Murray et al. (1983) reported prosthetic
knee swing flexion of 45°, 55° and 60° at slow, medium and fast walking speeds with a
hydraulic knee mechanism, and 57°, 66° and 78° walking with a constant friction knee
mechanism. Zuniga et al. (1972) reported 45° prosthetic knee flexion in the swing phase
compared to 51° on the intact limb. Thus, throughout the gait cycle, knee flexion in the
above-knee amputee prosthetic limb is determined by the prosthesis. Differences in knee
joint angles between the prosthetic limb and intact limb have been previously quantified.
The mechanisms of the prosthetic knee joint may affect the ability of above-knee
amputees to flex the knee at increasing walking speeds, thus leading to possibly greater
kinematic asymmetry when greater demands are placed on the amputee.

The hip joint.

Few differences either between limbs or compared to able-bodied subjects have
been reported for the below-knee amputee hip joint. One study has reported 30° maximal
hip flexion and 10° maximal hip extension for both the intact limb and two different
prostheses on the prosthetic limb (Smith, 1990). Therefore, little asymmetry has been noted in sagittal plane hip angles for below-knee amputees regardless of prosthesis worn.

The level of activity of the hip muscles in above-knee amputees has been found to be dependent on the length of the stump (Radcliffe, 1994). Stump length is important in providing an adequate lever arm for the transmission of force between the stump and the socket of the artificial limb. If the stump is very short, the use of the abductor muscles in stabilising the pelvis may be lost, resulting in greater movement of the hip and greater energy expenditure. In a longer stump, partial use of the hamstrings may be of additional use to the amputee. In able-bodied gait, the hamstrings are used to decelerate the leg at the end of the swing phase ready for heel contact. This decelerates both hip flexion and knee extension. After heel strike, the hamstrings act in stabilising the pelvis and initiating hip extension (Radcliffe, 1994). If the length of the hamstrings is halved, their excursion is also halved resulting in impairment of their ability to develop tension (Radcliffe, 1994) and therefore there is a loss of their function to act as knee flexors and hip extensors. This results in an obvious loss of ability to decelerate the knee and hip at the end of the swing phase, due to the loss of the hamstrings opposing hip flexion and knee extension. Another result is that the knee joint passively continues into hyperextension which, due to the reduced function of the knee flexors, this extension is incompletely opposed.

The loss of action on the hip also results in a loss of initiation of hip extension during the stance phase. This loss, or partial loss of the hamstrings also results in an inability to stabilise the pelvis, resulting in an increasing tendency to bend the lumbar spine laterally in order to carry the trunk more directly over the prosthesis (Radcliffe, 1994). Hip extension has thus been found to start early in the stance phase and increases to
a greater extension angle before toe-off, whilst hip flexion has been shown to occur late in the swing phase and is very limited for above-knee amputees (Radcliffe, 1994).

3.4.2.4 The effect of speed on spatial variables of gait.

Only one study has been found reporting the effect of walking speed on spatial variables of amputee gait. Boonstra et al. (1993) investigated “comfortable” and “fast” walking in above-knee amputee and able-bodied subjects. Variability in joint angles of amputees was lower than for the able-bodied subjects. However, values of joint angles at the two speeds and level of asymmetry were not reported. If walking speed affects spatial variables of amputee gait in the same way as in able-bodied gait, asymmetry, due to the prosthetic joints, previously seen in amputee gait at a natural walking speed, may increase. This would thus have implications for the design of the prosthesis for more active amputees.

3.4.2.5 Variability and asymmetry of amputee gait.

The amount of step-to-step variability is dependent on the degree of control during gait (Zahedi et al., 1987). With the partial loss of a lower limb, below-knee amputees should be more variable than able-bodied subjects in their gait, but not as variable as above-knee amputees. Engsberg et al. (1992) reported means and standard deviations for frontal and sagittal plane thigh angles of below-knee amputee children. Coefficient of variation was found to be up to ± 79.9% intact limb, ± 80.8% prosthetic limb for frontal plane and ± 8.7% intact limb, 8.2% prosthetic limb for sagittal plane thigh angles.

3.4.2.6 Summary of kinematics of amputee gait.

Temporal aspects of amputee gait have previously been well documented, whilst few authors have reported spatial parameters. Amputees exhibit an asymmetrical gait
pattern, having a shorter stance phase and longer swing phase on their prosthetic limb compared to their intact limb. As with able-bodied subjects, stance and swing time have been found to decrease with increasing walking speed, although the effect of walking speed on temporal asymmetry in amputee gait has not yet been concluded.

The angles of amputees at the ankle have been found to differ from those of able-bodied subjects both in terms of limited movement in the prosthetic ankle and compensatory increases in ankle ROM on the intact limb. Knee flexion angle for above-knee amputees has been found to be dependent on the prosthetic knee mechanism. Few studies have reported amputee hip angles, but it is suggested that the length of the residual stump in above-knee amputees will affect hip angle. No study has been found reporting the effect of increased walking speed on the magnitude of amputee joint angles. If joint angles are found to increase with walking speed as in able-bodied walking, this may force increased asymmetry at the higher speeds as the prosthetic ankle or ankle/knee combination offers no ability to increase ROM naturally. This has implications for amputees who are highly active and want to walk as naturally as possible. How amputees cope when greater demands are placed on them must be known before advances in prosthetic mechanisms can be made.

3.4.3 Summary of kinematics.

Temporal and spatial aspects of gait have been measured to quantify able-bodied and pathological gait. Stance, swing and single support times have been reported for able-bodied subjects at a variety of walking speeds (decreasing as walking speed increases), but with no mention of asymmetry. Amputees have been shown to spend longer in stance on their intact limb than on their prosthetic limb, and have a longer swing time on the prosthetic limb than on the intact limb. These phases have also been
found to reduce with walking speed. However, whether the amount of temporal asymmetry seen in amputee gait increases or decreases with speed has not previously been quantified.

In able-bodied walking, lower limb joint angles have been found to follow a distinct and recognisable pattern. Ankle plantarflexion and dorsiflexion angles during support have been shown to differ only within a few degrees between subjects (Whittle, 1991), thus due to anatomical movements of the foot during stance, ankle angles can be seen to vary slightly from individual to individual. There have been few studies reporting hip flexion and extension patterns. The hip has been shown to be extending at heel strike, continuing to extend to just before toe off, then flexing prior to the start of swing. Therefore, from past research, much is known about the lower limb joint patterns of able-bodied subjects during walking, and this can serve as a basis from which to compare the results of the walking gait of amputees.

Joint angles of above- and below-knee amputees have not widely been included in kinematic analysis of gait. However, from descriptive studies, it has been suggested that ankle angles will be dependent on the type and rigidity of the prosthesis used. It has also been suggested that knee flexion angle in below-knee amputees during stance is slightly less than that of able-bodied subjects due to the problems associated with a non-flexible prosthetic foot. Finally, amputee hip angle has not been documented.

Few studies have reported joint angle values for above-knee amputees during walking gait. However, previous descriptive analysis suggests that above-knee amputee gait will exhibit limited ankle movement, depending on the type and rigidity of the prosthetic foot, a lack of knee flexion during the 'cushioning' phase of stance, and a loss of
action at the hip. Therefore, lower limb joint angle data for above-knee amputees are needed in order to compare with able-bodied subjects and assess exactly how hip, knee and ankle angles differ throughout the gait cycle, and how the amputee copes with greater demands such as increased walking speed. There may be aspects of pathological gait not found by kinematic analysis alone. Therefore, a more in-depth kinetic analysis may be needed in order to assess aspects of joint function and compensation for the partial loss of a lower limb not seen in joint angle and step time patterns.
3.5 Joint kinetics.

Net joint moments, the product of a net muscle force acting at a distance about an axis of rotation, and joint power outputs, the rate of generating or absorbing energy by the muscles acting at a joint, calculated by inverse dynamics procedure, can provide additional information about hip, knee and ankle function not detected by joint kinematics during walking. While a kinematic variable such as knee angle may be relatively the same at a given point in the gait cycle both within and between subjects, the same cannot be said of the underlying kinetic variables due to force and muscle patterns acting at the joints (Winter, 1983). Therefore, kinetic assessment of joints during gait has been shown to be essential in illustrating the differences in not only gait patterns, but also the net moments acting at each joint.

3.5.1 Description of joint kinetics for able-bodied walking.

As in kinematic analysis of gait, studies have reported either two-dimensional or three-dimensional aspects of joint moments and joint power outputs. Three-dimensional kinetic analysis, whilst increasing in complexity and variability, will give insight into pathological gait in terms of frontal plane and rotation that may have been missed in two dimensional analysis. Harris et al. (1994) reported a hip flexor and extensor moment of 1 Nm.kg\(^{-1}\), abductor moment of 0.75 Nm.kg\(^{-1}\) and a zero adductor moment during able-bodied walking, indicating joint moments of similar magnitudes in a plane other than sagittal. Whilst most studies have reported joint moments in the sagittal plane only, looking at three-dimensional analysis could be helpful in analysing abnormal gait where the motion is not entirely in one plane, although variability of the results could increase.
3.5.1.1 Net joint moments for able-bodied walking.

During the stance phase of gait, shock absorption, weight bearing stability and progressional demands are made on the limb. In able-bodied gait, the appropriate intensity of muscle contraction and timing act in response to the influences of gravity and inertia to meet the demands placed upon the stance limb (Skinner et al., 1985). The patterns of moments generated at the hip, knee and ankle joints are thus a result of the ever changing alignment between centre of body mass and supporting limb progression. The resultant net joint moment is dependent on the net force moment acting at that joint and acceleration of the relative segment. Thus, the net joint moment is produced in response to the ground reaction force vector acting on the subject whilst walking (Figure 3.12).

![Figure 3.12 - Position of the limb and ground reaction force vector during stance phase walking](adapted from Whittle, 1991, pp60-68).

How these joint moment patterns occur are of interest in understanding how the lower limb joints act together in opposing the ground reaction force and propelling the body forward.

The ankle.

In able-bodied subjects, net joint moments have shown that during walking, only the ankle joint has exhibited a consistently recognisable pattern - a small dorsiflexor moment followed by a strong plantarflexion moment (Figure 3.13). For able-bodied
walking gait, at heel strike the ankle is near the neutral position with the force vector as seen in Figure 3.13. The direction of the force vector then changes within 10-20 ms of heel strike, producing an external plantarflexor moment (Figure 3.13). This moment needs to be resisted to prevent the foot from lowering too quickly and 'slapping' the ground. Here, the anterior tibial muscles contract eccentrically, lowering the foot to the ground, producing an internal dorsiflexor moment. During the interval between heel strike and "foot flat" (Figure 3.12) the ground reaction force vector increases rapidly in magnitude, showing the 'weight acceptance' phase of gait. From "foot flat" to "heel off" (Figure 3.12), the small initial external plantarflexor moment provided by the ground reaction force vector is replaced by an external dorsiflexor moment of increasing magnitude as the force vector passes in front of the ankle joint. This action is resisted by contraction of the gastrocnemius and soleus which prevents the tibia from moving forwards too quickly, producing a larger internal plantarflexor moment.

As the heel continues to leave the ground and the foot moves towards toe off (Figure 3.12), the external dorsiflexor moment caused by the ground reaction force vector increases and the gastrocnemius and soleus muscles contract to oppose it. This peak external dorsiflexor moment is reached just after heel off in conjunction with ankle plantar flexion and then decreases. A typical ankle joint moment graph can be seen in Figure 3.13.

Studies have reported net ankle joint moments of various magnitudes, some normalising to body weight, others to body weight and leg length. Winter (1980) reported net ankle flexor moments of 20 and 30 N.m and extensor moments of 120 and 170 N.m for a female and male subject, respectively. Other studies have reported between 10-36 N.m flexor and 100-150 N.m extensor moments during able-bodied walking (Bresler and Frankel, 1950; Paul, 1971; Pedotti et al., 1977). More recently, studies have been in
agreement of between 1 and 1.5 Nm.kg\(^{-1}\) net ankle extensor moments (Ounpuu et al., 1988; Winter, 1993; Novacheck, 1995; Kuster et al., 1995).

Figure 3.13 - a) ankle b) knee and c) hip net joint moments of able-bodied subjects walking normalised to body weight and leg length (Meglan and Todd, 1994, pp91).

The knee.

In all studies investigating able-bodied knee joint moments during walking, the pattern at the knee has been shown to be inconsistent and unpredictable (Morrison, 1970; Winter, 1980) (Figure 3.13). Whilst these studies showed inconsistent knee joint moment patterns, they noted no abnormal total gait patterns in any of the subjects and therefore the incidence of unpredictable knee joint moments is unexplained.

Following heel contact, the knee flexes in the 'cushioning' phase in order both to absorb shock and to prevent the vertical reaction force from building up too rapidly. Here, the ground reaction force vector passes behind the knee producing an external flexor moment (Figure 3.12). This is opposed by an internal extensor moment generated by the quadriceps which contract eccentrically to allow a small amount of controlled flexion. As knee flexion continues, the external flexor moment reaches a peak at 15-20% of the gait cycle in walking.
By mid-stance (Figure 3.12), the knee has started to extend again and the external flexor moment reduces in magnitude. Here, the quadriceps have finished contracting and therefore knee flexion is resisted by a combination of contraction of the soleus muscles and the forward momentum of the upper body. The contraction of the soleus slows down the motion of the tibia, and as the femur continues to move forwards, the knee extends towards "heel off". Just before "heel off", the ground reaction force vector moves in front of the knee, producing an external extensor moment. The gastrocnemius starts to act to oppose this motion by acting as a flexor at the knee, preventing hyperextension and producing an internal flexor moment. Prior to "toe off", the force vector moves behind the knee, subjecting it to an external flexor moment. Here, the rate of knee flexion is controlled by eccentric contraction of the rectus femoris which flexes the hip at the same time as it controls knee flexion. An internal extensor moment is thus created, reducing in magnitude as the leg reaches "toe off". The knee joint moment graph can be seen in Figure 3.13. The net knee joint moment was shown to be biphasic and mainly extensor across the stance phase with little or no flexor moment at the beginning and end of stance.

The pattern of knee joint moments in able-bodied subjects has been found to be inconsistent for most studies investigating walking (Morrison, 1970; Winter, 1980). Winter (1980) reported a variable pattern. Some subjects showed a small amount of knee flexor moment at heel strike, 10-20 Nm, followed by a varying amount of knee extensor moment, 30-42 Nm. For the rest of stance in all subjects, the knee was shown to exhibit varying flexor moments followed by a small extensor moment just prior to "toe off". Other studies reported between 25-85 Nm initial peak flexor moment, 10-65 Nm second peak knee flexor moments and 5-80 Nm peak extensor moment with a 15-50 Nm extensor moment prior to "toe off" (Bresler and Frankel, 1950; Paul, 1971; Pedotti et al., 1977) illustrating the variability of reported non-normalised net knee joint moments. Normalised.
data have shown knee flexor moments of 0.2 Nm.kg\(^{-1}\) and extensor moments of 0.3-0.5 Nm.kg\(^{-1}\) (Ounpuu et al., 1988; Winter, 1993; Novacheck, 1995) with one study reporting a net knee extensor moment of 1.2 Nm.kg\(^{-1}\) (Kuster et al., 1995).

The hip.

The hip has been shown to exhibit a less consistent pattern than the ankle, but usually shows an extensor followed by a flexor moment (Winter, 1980) (Figure 3.13). About 20 ms after heel contact, the ground reaction force vector passes in front of the hip resulting in an external flexor moment (Figure 3.12). In order to oppose this, the gluteus maximus and hamstring muscles contract to provide an internal extensor moment. The external flexor moment of the hip and the opposing contraction of the extensor muscles declines during the mid-stance phase. The external flexor moment is replaced by an external extensor moment as the ground reaction force vector passes behind the hip (Figure 3.12). This is opposed by an internal flexor moment generated by the hip flexors contracting eccentrically. The external hip extensor moment continues to act, with the hip flexors continuing to contract eccentrically in order to resist producing an internal flexor moment. A graphical representation of the hip joint moment can be seen in Figure 3.13.

Winter (1980) reported varied hip moment patterns. One female subject during walking exhibited a relatively small peak extensor moment of 55 Nm following heel strike, reducing slightly during stance and changing to a peak hip flexor moment of 40 Nm just prior to "toe off". A male subject exhibited a large peak hip extensor moment, 130 Nm, from just after heel strike until the end of the weight acceptance phase. The hip moment then became flexor, increasing to 60 Nm just prior to toe off. Other studies have reported peak extensor moments of 32-100 Nm (Bresler and Frankel, 1950; Paul, 1971; Pedotti et al., 1971), and 0.5-0.6 Nm.kg\(^{-1}\) (Ounpuu et al., 1988; Winter, 1993; Novacheck,
Peak flexor moments have been reported of between 30-170 Nm (Bresler and Frankel, 1950; Pedotti et al., 1977) or normalised to between 0.4-1 Nm.kg$^{-1}$ (Ounpuu et al., 1988; Winter, 1993; Novacheck, 1995; Kuster et al., 1995), showing the hip peak flexor moments to be more variable than extensor moments in able-bodied gait.

3.5.1.2 Errors and variability in able-bodied joint moments.

Due to the nature of the calculation of net joint moments, Davis et al. (1997) reported up to 50% error in calculation due to marker placement, errors in centre of pressure (COP) alignment, and in force and motion synchronisation. McCaw and DeVita (1995) looked at how errors in alignment of COP affected the magnitude of joint moments. By shifting the COP posteriorly by 0.5 cm and 1.0 cm, errors in the peak joint moment of magnitude 7% and 14% respectively were seen. Peak flexor moments at the hip, knee and ankle increased, whilst peak extensor moments decreased. Shifting the COP in an anterior direction yielded the opposite results. Thus, small errors in spatial alignment of force and motion data will result in considerable errors in joint moment calculations.

Errors can also arise from problems with temporally synchronising force and kinematic data (O’Connor et al., 1995). Using video, O’Connor et al. (1995) showed a one field synchronisation error (1/60 s) resulted in a 59% error in peak knee joint moment and a 70% error in the magnitude of peak power during walking. Alignment of ground reaction force vectors with the position of body segments depends on the accuracy at which they can be both spatially and temporally matched. Therefore, both synchronisation and alignment of force and joint angular data are critical issues when calculating joint kinetics (O’Connor et al., 1995).
Besides temporal and spatial alignment of force and motion data, estimates of body segment parameters can also add to errors calculated for joint kinetics. Using the inverse dynamics procedure, calculations of joint moments are dependent on previously measured anthropometric data. The majority of studies use anthropometric data from Dempster (1955) to calculate joint kinetics where errors can arise from individual differences in segment centres of mass and inertial properties. However, Miller (1987) calculating joint moments for below-knee amputees by a) taking into account individually measured segment centres of mass and inertia and b) using Dempster's (1955) data, reported only a ± 4 Nm difference in peak joint moments. For a population such as below-knee amputees where inertial properties of the prosthesis differ considerably from the intact limb as the prosthetic limb is lighter, this study showed that it is not the error in calculation of inertia that contributes greatly to errors in joint moments. This is thus true for able-bodied subjects where there is much less mass and inertial asymmetry between the limbs. It would be assumed then, that able-bodied subjects would exhibit a much less than ± 4 Nm error in joint moments due to errors from segmental data alone. The most influencing errors in kinetic calculations have been shown to stem from spatial and temporal synchronisation errors of force and motion data.

Studies have shown joint moments to be more variable than kinematic aspects of gait due to the muscle activity and forces associated with the production of joint moments. Joint moment intra-subject variability has been reported to have a coefficient of variation of ± 24-36% for the ankle, ± 62-64% for the knee and ± 62-66% for the hip (Winter, 1987; Smith, 1993). For inter-subject variability, ± 42-46% for the ankle, ± 78-135% for the knee and ± 57-140% for the hip have previously been reported (Winter, 1987; Smith, 1993). This thus illustrates that not only are able-bodied subjects variable in terms of hip
and knee joint moments, but there is a large variability between subjects, especially for knee joint moments.

3.5.1.3 Power output at the hip, knee and ankle.

Muscle mechanical power \( (P_m) \) is calculated as the product of the net muscle moment of force \( (M_m) \) and the relative joint angular velocity \( \omega \), where \( P_m = M_m \omega \) (McFadyen, 1994). If the component of the resultant net joint moment of force is in the same direction as the joint angular velocity, then the result is a positive joint power output. However, if angular motion and the net joint moment are in opposing directions, the result is a negative power output (Meglan and Todd, 1994).

Investigating muscle mechanical power output combines both kinematic and kinetic information to provide insight into the net rate of energy put into or taken from the segments by the dominant muscle group during a particular movement. This analysis thus provides an effective means of interpreting movement during gait (McFadyen, 1994).

3.5.1.4 Power output in able-bodied walking gait.

In able-bodied gait, ankle plantar flexors are the main source of power generation, and knee extensors the main source of power absorption, with the hip playing a minor role in power absorption and generation (Winter, 1983).
Studies have shown power output graphs to illustrate the action of the muscles used in gait (Figure 3.14). Positive areas reflect positive work done, a net muscle moment that is concentric in action and the muscles are generating energy. Negative parts of the graph represent an eccentric muscle action where the muscles are absorbing energy (Smith, 1990).

The ankle.

In able-bodied subjects during walking, the predominant muscle group at the ankle joint is the triceps surae which acts to both absorb power during mid-stance and generate power during push off. This muscle group thus effectively controls the motion of the body over the stationary foot throughout most of stance. Harris et al. (1994) reported a peak ankle power absorption of 1 W.kg$^{-1}$ during stance. At push-off, studies have reported 1.58-2 W.kg$^{-1}$ as the ankle is producing power (Harris et al., 1994; Kuster et al., 1995). The majority of studies have reported natural cadence walking and thus differences in power output magnitude may be accounted for by differences in walking speed.
The knee.

Meglan and Todd (1994) suggested that during walking, the knee generates power immediately after heel strike, but then switches to power absorption corresponding to the knee continuing to flex even though the net knee moment is extensor (Figure 3.14). As knee flexion slows and then moves into extension, knee power output becomes positive (0.5-1 W.kg\(^{-1}\), McFadyen, 1994; Harris et al., 1994). During weight release, knee power output becomes negative as the knee starts to flex to prepare for swing. At this point, the knee moment is in extension which helps to propel the lower limb forward during swing. Peak knee power absorption has previously been reported between 1.5 W.kg\(^{-1}\) (McFadyen, 1994; Harris et al., 1994) and 7.5 W.kg\(^{-1}\) (Kuster et al., 1995) for able-bodied walking.

The hip.

Power output curves of the hip have been shown to be triphasic (Kuster et al., 1995) and hip musculature tends to act concentrically during extension and flexion except during early push off. Here, the hip is extending to the end of its range while the hip flexors are eccentrically dominant (Smith, 1990). Meglan and Todd (1994) (Figure 3.12) described the hip as generating power early in stance as the joint is flexing and the net joint moment is in extension. During mid-stance, power is absorbed (1 W.kg\(^{-1}\), Harris et al., 1994) as hip flexion slows and switches to extension as the net hip moment moves into flexion. During weight release, power output becomes positive (1.2-1.8 W.kg\(^{-1}\), Harris et al., 1994; Kuster et al., 1995) as hip extension slows and the net hip moment remains in flexion.
3.5.1.5 Variability in power output for able-bodied gait.

Joint power outputs have been reported to be more variable than net joint moments (Winter, 1987; Smith, 1993). Studies have reported coefficients of variation for intra-subject variability of ± 51-129% for ankle power output, ± 68-110% for knee power output and ± 74-80% for hip power output. Joint power output inter-subject variability has been shown to be ± 96-100% for the ankle, ± 98-121% for the knee and ± 73-96% for the hip (Winter, 1987; Smith, 1993).

3.5.1.6 The effect of speed on joint kinetics in able-bodied gait.

Only one study has been found reporting the effect of walking speed on net joint moments and power output (Holden et al., 1997). At the slowest walking speeds (25% and 50% of natural cadence), there was a very small knee moment resulting in negligible joint power output through the first 80% of the stance phase. As the subjects walked faster and increased knee flexion, they increased knee extensor moment, power absorption and power generation (Holden et al., 1997). Thus, net joint moments and power output have been shown to increase with increasing walking speed for able-bodied subjects.

3.5.1.7 Summary of joint kinetics in able-bodied gait.

Studies have previously reported joint kinetics during walking for able-bodied subjects. However, few have normalised individual data, making it difficult to compare results. Generally, authors have been in agreement with net ankle plantar flexor moments of 1-1.5 Nm.kg⁻¹, knee flexor moments of 0.2 Nm.kg⁻¹, extensor moments of 0.3-0.5 Nm.kg⁻¹, hip flexor moments of 0.4-1 Nm.kg⁻¹ and extensor moments of 0.5-0.6 Nm.kg⁻¹. Errors in net joint moment calculations arise mainly from incorrect spatial and temporal synchronisation of force and motion data. Joint moments of able-bodied subjects have
been shown to be quite variable, particularly for the knee and hip, both within and between subjects. Joint power outputs have been shown to be even more variable.

The ankle plantar flexors in able-bodied subjects are the main source of power generation, and knee extensors the main source of power absorption. Again, few studies have normalised individual joint power output data making it difficult to compare results. Generally, authors have suggested that ankle power generation at push off is between 1.58 and 2 W.kg\(^{-1}\), knee power absorption between 1.5 and 7.5 W.kg\(^{-1}\) and hip power generation 1.2-1.8 W.kg\(^{-1}\). Both net joint moments and power outputs in able-bodied gait have been shown to increase with increasing walking speed.

3.5.2 Description of joint kinetics in amputee walking gait.

If the ankle is the major power generator at push-off, the loss of such a joint will affect both joint moments and power output of the amputee. Depending on the type of prosthesis used, the amputee will have to adapt and compensate in such a way that may show an asymmetrical kinematic pattern, generating more power at another joint during push-off.

3.5.2.1 Net joint moments of amputees walking.

On investigating joint patterns of below-knee amputees when walking, it was discovered that even though previous studies have shown certain kinematic aspects of gait between below-knee amputees and able-bodied subjects to differ only a little (Breakey, 1976; Whittle, 1991), joint moments were considered to differ immensely (Winter, 1983). Little data have been found on net joint moments of above-knee amputees walking. However, due to similar problems with the above-knee and below-knee amputee's
prosthetic ankle joint, it is expected there will be similar ankle joint moments. An example of joint moments observed for below-knee amputees are seen in Figure 3.15.

![Figure 3.15 - Ankle, knee and hip net joint moments of below-knee amputees walking at a natural cadence (Winter and Sienko, 1988, pp364).](image)

The ankle.

Winter and Sienko (1988) on investigating joint moments of below knee amputees walking, using a SACH foot (solid ankle cushioning heel), found that the ankle moment was dorsiflexor (1.6 Nm/kg) for the first 18% of stride compared with 6% for able-bodied subjects. This shows that, whilst able-bodied subjects have a rapid lowering of the foot to the ground under control of the dorsiflexors, below knee amputees using this particular prosthetic foot have rigid ankles which generate an internal dorsiflexor moment from heel contact until the foot is flat on the ground. It thus takes longer for an amputee's prosthetic leg to rotate forward until the foot is flat on the ground because of the rigid ankle than it does for able-bodied subjects, illustrating that the construction of the prosthetic foot and its ability to plantarflex will affect the resultant joint moments. Lemaire et al. (1993) reported 1.5 Nm.kg\(^{-1}\) plantar flexor moment and 0.4 Nm.kg\(^{-1}\) dorsiflexor moment for the prosthetic
limb of below-knee amputees, but did not report values on the intact limb. Schneider et al. (1993) investigated net joint moments for natural and fast walking in below-knee amputee children with a SACH and a Flex foot. The study showed that for the SACH foot, ankle joint moments were lower on the prosthetic limb than the intact limb at both speeds (1.01 Nm.N compared to 1.3 Nm.N for extensor moments), but similar for both limbs when using the Flex foot (1.45 Nm.N and 1.45 Nm.N respectively). Thus showing that the type of prosthetic foot can make a difference to the magnitude of the ankle joint moment not just on the prosthetic limb, but on the intact limb as well.

For above-knee amputees, Lewallen et al. (1985) reported a plantar flexor moment of 0.09 Nm.kg\(^{-1}\).m on the intact limb and 0.06 Nm.kg\(^{-1}\).m on the prosthetic limb. Yang et al. (1991) investigated the effects of prosthetic alignment on above-knee amputee joint moments. When the foot was set to a plantar flexion angle, the peak knee extensor moment was increased, and the dorsiflexor moment duration was extended. When the foot was set to a dorsiflexion angle, these findings were reversed. When the socket was set to an increased flexion angle, the duration of the ankle dorsiflexor moment was increased and the peak knee extensor moment was reduced.

The knee.

Knee joint moments for below-knee amputees have been found to show great deviations from able-bodied gait. Winter and Sienko (1988) reported very little knee extensor moment during natural cadence walking stance. Lemaire et al. (1993) reported a 0.5 Nm.kg\(^{-1}\) extensor moment and 0.3 Nm.kg\(^{-1}\) flexor moment. Gitter et al. (1991) reported net knee extensor moments of 10 Nm with the SEATTLE foot, 15 Nm with the SACH foot and 20 Nm with the Flex foot for below-knee amputees walking at 1.5 m.s\(^{-1}\),
again showing that the type of prosthetic foot will affect the magnitude of the knee joint moment. Hoy et al. (1982) and Schneider et al. (1993), both studies with below-knee amputee children, reported lower knee flexor and extensor moments on the prosthetic limb than the intact limb (1.5 Nm compared to 5 Nm extensor, and 6 Nm compared to 13 Nm flexor moments walking at a natural cadence). Again, for the Flex foot, the difference between the two limbs in terms of net joint moments was reduced compared to the SACH foot prosthesis. Both studies also showed that the magnitude of the joint moments increased slightly with walking speed but there was no comment on the level of asymmetry or whether walking speed had an effect on amputee kinetic asymmetry. For above-knee amputee knee joint moments, Lewallen et al. (1985) found a lower flexor moment for the prosthetic limb than the intact limb (0.005 Nm.kg$^{-1}$.m and 0.07 Nm.kg$^{-1}$.m respectively), showing similarities with below-knee amputees. A greater knee extensor moment was seen on the prosthetic limb compared to the intact limb (0.04 Nm.kg$^{-1}$.m and 0.01 Nm.kg$^{-1}$.m respectively). This contradicts the results of the studies reporting knee joint moments for below-knee amputees. This may reflect the method in which above-knee amputees compensate for the additional loss of a fully functional knee joint and the effects of the prosthetic knee mechanism.

The hip.

Hip moments for below-knee amputees walking were found to be highly variable, moreso than for able-bodied subjects (Winter and Sienko, 1988). Three subjects in the study using the SACH foot showed a completely extensor moment pattern during stance whilst two other subjects exhibited greater than able-bodied flexor moments. Below-knee amputees have therefore been found to exhibit large hip flexor and extensor moments on the prosthetic limb during the stance phase of walking, with little knee moment activity (Winter and Sienko, 1988) compared to able-bodied subjects. Lemaire et al. (1993)
reported a peak hip flexor moment of 0.8 Nm.kg\(^{-1}\), and peak hip extensor moment of 0.6 Nm.kg\(^{-1}\), both at the upper end of the range previously reported for able-bodied subjects. For above-knee amputees, the hip joint moment has been reported as a mainly flexor moment on the prosthetic limb (0.05 Nm.kg\(^{-1}\).m) with no extensor moment (Lewallen et al., 1985). For the intact limb, the flexor and extensor moments were of the same magnitude (0.05 Nm.kg\(^{-1}\).m) (Lewallen et al., 1985). From these results, it is seen that joint moment asymmetry is present in both above- and below-knee amputee gait. However, no study has been found quantifying the amount of asymmetry. Net joint moments can illustrate more fully how an amputee adapts to the partial loss of a lower limb in terms of joint movement, muscles and forces. What happens to joint moment asymmetry when increased demands such as increasing walking speed are placed on the amputee are not yet known. No studies have been found concerning the effect of speed on amputee joint moment asymmetry.

3.5.2.2 Variability in amputee net joint moments.

Little has been reported of variability in amputee joint moments. Lemaire et al. (1993) reported coefficient of variation for ankle moments of ± 24.2%, ± 81.0% for knee moments and ± 64.6% for hip moments on the prosthetic limb. Smith (1990) reported less variation on the prosthetic limb than the intact limb with ± 37% ankle, ± 70% knee and ± 30% coefficient of variation for the prosthetic limb and ± 58% ankle, ± 187% knee and ± 113% hip for the intact limb. The prosthetic limb appears to be not only less variable than the intact limb, but the prosthetic ankle is less variable than for able-bodied subjects. Thus, the construction of the prosthetic limb appears to determine partly the ankle joint moment on that limb. No data have been found for variability of above-knee amputee joint moments.
3.5.2.3 Errors in calculating net joint moments in amputees.

As in with calculations of joint moments for able-bodied subjects, errors can arise in the calculation of amputee net joint moments. In addition to errors in temporal and spatial synchronisation of force and motion data, there are differences in anthropometric measurements to take into account for amputees. Amputees have been shown to have asymmetrical gait patterns due to the nature of the prosthesis which is lighter than the contralateral limb in order to reduce energy cost whilst walking. Thus, the mass, position of centre of mass and inertial properties of the prosthetic limb differ from those of the intact limb and need to be taken into account when using anthropometric calculations. However, few researchers have done this. Whilst these anthropometric variables must be measured when calculating amputee joint moments to give a valid result, Miller (1987) reported only a ± 4 Nm difference in peak joint moments between taking these variables into account and using “normal” anthropometric data.

3.5.2.4 Power output in amputee gait.

As the major power generators at push-off, the triceps surae, are absent or partially absent in amputees, power needs to be generated elsewhere. Due to this, the hip and knee need to compensate, thus forcing amputees to exhibit different power output patterns to the able-bodied. Whilst some data have been found reporting joint power output of below-knee amputees, none have been found for above-knee amputees.

The ankle.

In below-knee amputees, the power generated from the prosthetic foot is negligible. Also, power absorption of the knee is markedly diminished and thus the major
power generating source of the stance phase is lost. The amputee compensates for this by using the hip extensors to generate the power needed (Czerniecki et al., 1991), thus showing how important the prosthetic foot is in terms of power absorption and generation. The greater the power absorption and generation provided by the foot, the less the hip extensors are needed to compensate.

On investigating the power output of below-knee amputees walking at a natural cadence, Winter and Sienko (1988) discovered that the prosthetic ankle of the SACH foot absorbed a little power after heel strike (0.3 W/kg) (Figure 3.16). As the SACH foot has a solid ankle which cannot flex, the power absorption was attributed to the foot deforming slightly on impact and the energy being dissipated in the viscous SACH foot. The study then showed negligible power was generated by the foot in the latter part of stance and push-off (0.1 W/kg). This small amount could possibly be attributed to digitisation error or error in the calculations of power output, as a SACH foot has a fixed ankle and cannot generate power. Therefore, power generation using a SACH foot should be zero. These results differed from a later study (Lemaire et al., 1993) reporting 1 W/kg ankle power absorption, and a very high 3 W.kg\(^{-1}\) generation at push off. This was due to differences in the type of prosthesis used in these studies. The SACH foot is solid with a cushioning heel and, being rigid, has no ankle movement and thus no power output at push off. Lemaire et al. (1993) investigating amputees using an “energy returning” foot would find an amount of power absorption after heel strike and power generation at push off.

Smith (1990) discovered that below knee amputees absorbed and generated little power during stance, as a lack of push off kinematically could be seen in the virtual lack of substantial amounts of positive power during push off. Here, he reported a large burst of energy absorption by the ankle early in the stride (19-20%) during weight acceptance,
indicating that the prosthesis used, here the SACH foot, was capable of absorbing power (300 W). However, the ankle did not generate much power at push off (30-40 W); it was assumed that this power was either transferred to the leg through the stump or dissipated as heat. In this particular study, again it is not possible to generate power with a SACH foot. The 30-40 W generated (or for an 80 kg individual 0.5 W/kg) could possibly have been due to errors in digitising or power output calculations.

Schneider et al. (1993) reported that both power absorption and generation of the ankle increased slightly with walking speed and less power was generated and absorbed on the prosthetic compared to the intact limb. With using the Flex foot, more power was generated at push-off on both the prosthetic and intact limbs than with the SACH foot.

The knee.

Winter and Sienko (1988) found that any power absorption by the knee during the early stance phase of walking was absent for the below-knee amputees. However, the knee was forced to absorb power during push-off (0.7 W.kg$^{-1}$) (Figure 3.16). Knee power was
shown to exhibit positive bursts during knee flexion and negative during knee extension prior to push off, unlike that of able-bodied subjects. Thus, for this particular subject, Smith (1990) suggested that there was a tendency to collapse at the knee during single support. Lemaire et al. (1993) reported 1.9 W.kg\(^{-1}\) knee power absorption at push off and 0.3 W.kg\(^{-1}\) generation during stance. Schneider et al. (1993) illustrated that below-knee amputees absorbed more power at push-off on the prosthetic limb using the Flex compared to the SACH, but did not increase power absorption on the intact limb.

The hip.

Winter and Sienko (1988) also showed hip power patterns to be extremely variable for below-knee amputees during walking with the SACH foot, the mean power trace showing an initial power generation (0.7 W.kg\(^{-1}\)) followed by absorption (0.1 W.kg\(^{-1}\)), completed with power generation (0.5 W.kg\(^{-1}\)). The initial power generation was attributed to the hip extensors shortening under tension after heel contact and generating power to propel the body forwards (Figure 3.16).

In agreement with this study, Smith (1990) reported that the hip extensors compensated for the lack of power generation at the ankle and knee by generating a considerable amount of power during the hip extension phase, with the hamstrings being the most likely source of power pattern. Thus if this was true, the length of the stump would possibly affect hip power generation for above-knee amputees. However, whilst there has been some investigation into power output characteristics of the hip, knee and ankle for below-knee amputees and able-bodied subjects, there have been no corresponding data found for above-knee amputees.
3.5.2.5 Variability in amputee joint power output.

Lemaire et al. (1993) reported coefficient of variation (c.v.) for the prosthetic limb of below-knee amputees of ± 57.5% for ankle power output, ± 88.4% for the knee and ± 94.9% for the hip. Smith (1990) again reported less variation on the prosthetic limb than intact limb with ± 108%, ± 66% and ± 107% for ankle, knee and hip power output on the prosthetic limb and ± 121%, ± 187% and ± 169% on the intact limb, respectively.

3.5.2.6 Summary of amputee joint kinetics.

Problems with calculating amputee joint moments arise from differences in mass, centre of mass and inertia properties of the prosthetic limb compared to the intact limb. Corrections for these specific anthropometric measures have been done in few studies and have been shown to yield only a ± 4 Nm error. Problems with the prosthetic ankle joint have led to a reduced ankle joint moment and power output on the prosthetic limb compared to the intact limb, affected by type of prosthesis and prosthetic alignment. Net knee joint moments for below-knee amputees have shown lower knee flexor and extensor moments on the prosthetic limb compared to the intact limb. Prosthetic knee power absorption has been shown to differ, depending on the prosthesis used. Lower limb amputees have been shown to compensate for these problems by producing greater than able-bodied net hip joint moments. Whilst below-knee amputees have also been shown to generate a considerable amount of power during hip extension, no studies have reported hip power output for above-knee amputees. It is expected that as the hamstrings may be the most likely source of power generating during hip extension, the length of the residual limb may limit hip power generation for above-knee amputees. Peak net joint moments have been shown to increase slightly with walking speed in below-knee amputees, but no study has reported the effect of walking speed on joint power output. Also, no studies have
quantified the level of asymmetry between limbs and how it is affected by increases in walking speed.

3.5.3 Summary of joint kinetics.

Net joint moments for able-bodied subjects during walking have been shown to exhibit consistent patterns for both the ankle and hip; however, net knee moment patterns have been shown to be quite variable. During running, all three joint patterns have been shown to be more consistent and have exhibited greater peak moment values. The magnitude of peak joint moments has been shown to increase with walking speed.

Few studies have reported net joint moments for above-knee amputees during walking. One problem is that joint moments are affected by the different types of prosthetic feet used across studies. However, net ankle moments in below-knee amputees have been found to differ from able-bodied subjects by having a prolonged dorsiflexor moment, with the knee and hip moments being highly variable. It is suggested that the problems encountered by below-knee amputees will be enhanced in above-knee amputees as they have to compensate for the functional loss of two joints.

For joint power output of able-bodied subjects, large differences have been noted in both ankle and knee patterns between different studies investigating walking. Peak joint power output has been found to increase slightly with walking speed. Few studies have reported joint power output for below-knee amputees during walking, and none has been found to date investigating above-knee amputees. All three joints of the below-knee amputee have been found to differ from able-bodied subjects in power output and to depend on the type of prosthesis used. Generally, in below-knee amputees, the ankle absorbs and generates little power, knee power generation is markedly diminished and the
hip is seen to compensate by generating the power needed. Whilst there have been no data on joint power output of above-knee amputees, previous research suggests that if the amount of power output is dependent on the length of the stump, above knee amputees will have diminished hip power output values. If net joint moments and power output increase on the intact limb of unilateral amputees as with able-bodied subjects, and limitations of the prosthesis prevent large joint moments or power output at the prosthetic ankle or knee from occurring, kinetic gait asymmetry may increase with walking speed for lower limb amputees. Further aspects of joint moments and power outputs and the effect of the loss of musculature have been looked at in EMG studies.
3.6 Muscle activation patterns.

There is only a partial agreement between investigators as to what is a ‘normal’ pattern of muscle activity during gait (Whittle, 1991). Problems arise from the individual variability of muscle activity and different EMG (electromyography) techniques and methodologies used. Whilst the type of electrodes used, electrode placement, sampling frequency and signal processing will affect the results, differences in walking speed and individual muscle development can also account for differences across studies. However, studies have previously illustrated the general muscle patterns during able-bodied walking, and also the problems associated with pathological gait.

3.6.1 Methodological considerations.

There are many aspects of EMG to take into account when measuring muscle activity. There is as yet no agreement between authors as to electrode placement, normalisation of EMG signal and analysis of signal. This makes results difficult to compare across studies. The choices of electrode placement and signal normalisation are difficult as there are arguments for and against the techniques previously used.

3.6.1.1 Electrodes and electrode placement.

Studies have reported EMG signals from muscles measured by either fine wire or surface electrodes. Fine wire electrodes allow signals from deeper muscles to be recorded, but only record activity from that part of the muscle where the wire is inserted. Surface electrodes can only record activity from superficial muscles, but give a general picture of activity from the whole muscle. Kadaba et al. (1985) on investigating repeatability of fine wire and surface electrodes found that reproducibility and reliability were better for surface electrodes than the needle electrodes. The study concluded that
surface electrodes represent a more consistent measure of superficial muscle activity than fine wire electrodes.

Different authors have reported different electrode placements and inter-electrode distances. Clarys and Cabri (1993) on reviewing EMG studies reported that bipolar surface electrodes are placed parallel to the muscle fibres, but placements have varied from over the motor point; equidistant from the motor point; near the motor point; the mid-point of the muscle belly, on the visual part of the muscle belly, at standard distances from anthropometric landmarks, and with no precision at all. Roy et al. (1986) suggested that for some muscles, if the electrodes are placed over the motor point (which gives the most reliable information), during concentric contractions, the muscle belly shortens in the proximal direction and the surface electrodes move over the distal tendon. Therefore, it would be better to place the electrodes over the muscle belly. The majority of authors using surface EMG have reported electrode placement over the visual part of the muscle belly.

The distance between the electrodes is also important. Zipp (1982) found that the intensity of cross-talk from non-active muscles decreased with decreasing inter-electrode distance. This study concluded that for highly detailed information, small inter-electrode distances are needed, whilst for more general information on muscle activity, greater spaced electrodes should be used. However, this was valid for static contractions and not dynamic. Studies reporting inter-electrode distances during dynamic activity have commonly reported a spacing of 2 cm (Leisman et al., 1995; Winter and Yack, 1987) or 1 cm (Yang and Winter, 1984). Thus, placing surface electrodes over the visual part of the muscle belly with electrodes 1 to 2 cm apart have
previously been shown to give valid EMG signals and reduce cross-talk from non-active muscles.

3.6.1.2 Normalisation of EMG signal.

Differences in individuals’ muscle size, level of activity and strength all contribute to differences in magnitude of the EMG signal. Thus, as in when reporting ground reaction forces and net joint moments, there is a need to normalise signals in order to compare results between subjects. As in the reporting of joint moments, normalisation of EMG signal has varied across studies. From the literature, there appears to be two main normalisation techniques - normalising to maximum voluntary contraction (MVC) and normalising to peak signal amplitude. The majority of authors in the past have normalised EMG signals to 100% maximum voluntary isometric contraction. There are a number of problems with this technique, not least that it is normalising a dynamic movement to a static maximum. Most authors have reported MVCs taken from each muscle investigated, with the hip, knee and ankle at 90°. Intra-subject variability is high when using MVCs (Yang and Winter, 1984) as the maximum signal depends on the positioning of muscles, familiarity with the test and motivation of the subject. There have been some instances of EMG signal obtained during a dynamic movement being greater than the MVC for that subject (Lewillie, 1973; Clarys et al., 1983; Jobe et al., 1984).

Another argument against normalising to MVCs is that two subjects with the same dynamic signal could have a different magnitude of EMG once normalised to MVC. Intra-subject variability has been found to decrease if the EMG signal obtained is normalised to peak or mean amplitude measured during that activity (Yang and Winter, 1984). Whilst this method is now favoured as it normalises during the same dynamic
movement, a problem using this technique arises when looking at differences in EMG activity during different walking speeds. One author (Vint, 1999) normalised all EMG data to the mean ensemble average (6 strides) of the slowest walking speed to give a baseline value. The EMG data for all subsequent walking speeds were all expressed as a percentage of the baseline value. Coefficient of variation was reported to be lowest using this normalisation technique compared to others.

3.6.2 Muscle activity during able-bodied gait.

Studies have looked at the role of the individual muscles during the gait cycle, both in their own right, and in order to explain joint moments and joint power outputs. As seen in the previous section concerning kinetics, positive work is done by the muscles when contracting concentrically, and negative work is done when contracting eccentrically. Electromyographic activity is less in eccentric contractions than concentric contractions under the same load (Rab, 1994), suggesting that an eccentric contraction (negative work) requires less motor unit activity. Able-bodied human walking is therefore an energy efficient activity, as much muscle activity is isometric or eccentric (Rab, 1994). The negative work allows the limbs to absorb energy whilst resisting gravity. Positive work allows acceleration of the limbs as at push-off.

3.6.2.1 Activity of muscles in the lower limb joints.

The ankle.

At initial foot contact, the tibialis anterior begins to lengthen, preventing the foot from ‘slapping’ on to the floor. At mid-stance, the body falls forward, the ankle plantarflexors (gastrocnemius and soleus) contract isometrically, then concentrically as the body moves into terminal stance. The ankle plantarflexors produce most of the power generation at “toe off”. During the swing phase, the ankle dorsiflexors (tibialis
anterior) contract allowing the foot to clear the floor. At terminal swing, the tibialis anterior continues activity but now contracts isometrically, or elongates contraction ready for the next foot contact (Rab, 1994) (Figure 3.17).

Capozzo et al. (1976) reported that the ankle absorbs energy whilst in dorsiflexion. During double support, the ankle continues to plantarflex, performing positive work (soleus) as the gastrocnemius ceases activity. However, Fujita et al. (1985) showed that the ankle plantar flexors do not participate in the action of push-off in able-bodied walking. During the last 10% of the stance phase, the feet are in double support and the plantar flexors are not required for propulsion but as a stabilising force. During fast walking, however, the study reported that the ankle plantarflexors are required as propulsive forces (Fujita et al., 1985), thus illustrating one way that increasing walking speed affects muscle activity in able-bodied gait.
Figure 3.17 - Muscles active (shaded) during the gait cycle. Adapted from Gage (1990), pp294-300.
Some studies have reported the magnitude of the ankle plantarflexors and dorsiflexors (normalised to 100% MVC) during able-bodied walking. The tibialis anterior was seen to be active during the whole gait cycle, but minimal for a short period at “toe off” (Ericson et al., 1986). It exhibited two peaks, one at heel strike (27% MVC) and one at the beginning of the acceleration of the swing phase (15% MVC). The peak activity of the gastrocnemius (medial and lateral heads) was at 40-45% of the gait cycle, the beginning of the push-off phase, reaching a peak of 42% (medial head) and 19% (lateral head) MVC (Ericson et al., 1986).

The knee.

At initial foot contact, the knee flexors and extensors simultaneously act to stabilise the knee as the limb reaches the floor. During weight acceptance, the knee extensors (vasti) contract, the knee flexes and then begins to extend as the knee extensors contract concentrically. The knee remains extended in mid-stance (the highest point of the centre of mass) without the need for muscle action of the quadriceps, as contraction of the soleus produces a force couple to keep the knee in extension (Rab, 1994). The knee continues to extend passively through terminal stance, the rectus femoris beginning to act to lift the limb and swing it forward. At terminal swing, the leg begins to decelerate by eccentric contraction of the hamstrings. The knee moves into extension and prepares to accept weight early by the commencement of quadriceps activity (Rab, 1994) (Figure 3.17).

Capozzo et al. (1976) reported that the knee flexes under the control of the antagonistic action of the vasti and biceps femoris, absorbing energy from the extensors (rectus femoris). This is followed by an active extension (vasti). Ericson et al. (1986) reported peak biceps femoris and medial hamstring activity of 10% and 11% MVC.
occurring at 5% and 95% of the gait cycle respectively. The rectus femoris exhibited
low activity, but had two peaks at 10% of the gait cycle (5% MVC) and at the beginning
of the swing phase (5% MVC). Vastus medialis (14% MVC at 5% of the gait cycle) and
vastus lateralis (10% MVC at 10% of the gait cycle) showed greater activity during
walking than the rectus femoris.

The hip.

At initial contact, the hip extensors contract, decelerating the thigh and aiding
knee extension and foot placement. During weight acceptance, the gluteus minimus
contracts isometrically and stabilizes the pelvis in the frontal plane. At terminal stance,
there may be a small bursts of iliopsoas activity leading into pre-swing where the hip
flexors (iliopsoas and rectus femoris) contract concentrically to lift the limb and swing it
forwards. The limb acts as a passive pendulum for much of the swing phase, hip flexion
being slowed at terminal swing by eccentric contraction of the hamstrings in preparation
for the next initial foot contact (Rab, 1994) (Figure 3.17).

Capozzo et al. (1976) on looking at muscle activity in conjunction with joint
moments and power output reported that when the hip extends, the gluteus maximus,
medius and semitendinosus act to supply a flexing moment at the knee. The tensor
fascia latae was consistently active in the first half of stance, counteracting the external
adducting moment and absorbing energy. When the hip flexes, positive work is done by
the rectus femoris and iliacus. During the last part of double stance, the hip adductors
are active, counteracting an external abducting moment, absorbing energy.

Ericson et al. (1986) compared surface EMG magnitudes, normalized to 100%
MVC, of selected muscles during the gait cycle. Mean peak activity of gluteus maximus,
medius, at heel strike was 10%, reaching a peak of 15%, at 5% of the gait cycle. The activity then decreased, the muscles becoming more active again towards the end of the swing phase. These results were lower than reported in an earlier study (Lyons et al., 1983) using fine wire electrodes and Dubo et al. (1976) using surface electrodes, but again, differences in normalising technique, electrode placement and signal analysis may account for differences in results across studies. Lyons et al. (1983) reported that the semimembranosus and biceps femoris displayed the greatest swing phase activity (beginning in mid-swing). Next highest was the adductor magnus starting in terminal swing. Both the adductor magnus and gluteus maximus were principle hip extensors active during loading response and midstance.

3.6.2.2 The effect of speed on muscle activity during able-bodied walking.

Lyons et al. (1983), using fine wire electrodes, investigated the effects of speed on muscle activity. The tensor fascia latae changed very little in magnitude and duration with an increase in walking speed, but all other muscles did. The biceps femoris increased in magnitude from less than 30% MVC walking free to almost 40% MVC walking fast. The muscle also increased in duration, starting sooner in the swing phase. The tensor fascia latae was biphasic at fast walking (loading response then towards end of single support), and monophasic at free walking (middle to end of single support). All the other muscles were active for longer during faster walking, and switched on earlier in the cycle, again illustrating that walking speed has an effect on muscle activation pattern. The study concluded that walking speed can affect muscle duration and intensity.

Shiavi et al. (1981) reported that the tibialis anterior was monophasic at slowest speed, becoming multiphasic with speed. The gastrocnemius became active earlier in the
stance phase as walking speed increased, and also active during late swing at very fast walking. The hamstrings were mainly active during late-swing, loading and initial mid-stance, and exhibited a shorter period or absence of activity during late swing at slower walking speeds. The rectus femoris was always active during late swing, loading and initial mid-stance, but did not report increased duration with speed. The vastus lateralis, also mainly active as with rectus femoris at slow and free walking, was seen as a two phase burst which became one in fast walking.

Several studies have thus shown that phasic activity does change with walking speed (Milner et al., 1971; Cavanagh and Gregory, 1975; Brandell, 1977) which can be expected as ground reaction forces are a function of walking speed.

3.6.2.3 Variability and asymmetry in able-bodied walking.

Variability.

Arsenault et al. (1986b) looked at selected lower limb muscles to see if there was a “normal” profile for EMG signals in gait, and found that within subjects, data were highly repeatable whilst EMG amplitude differed greatly between subjects with normalisation to MVC. Yang and Winter (1984) reported that the MVC calibration was seen to increase variability. Similar EMG patterns were found between subjects, but with slightly different magnitudes, again due to individual variations in MVC. Differences in techniques used, such as surface or in-dwelling wire electrodes, could affect variability. Kadaba et al. (1985) looked at differences in repeatability between surface and wire electrodes and concluded that reproducibility and reliability were better for surface electrodes, as was consistency for both within day and between days.
The position of muscles with respect to the lower limb, and whether they are one-joint or two-joint muscles has also been found to affect variability in muscle activity during able-bodied walking. Winter and Yack (1987) investigated EMG during able-bodied walking, stride-to-stride and inter-subject variability. They reported that the distal support muscles (soleus, tibialis anterior, gastrocnemius) were the most active whilst the more proximal muscles, the least active. The least variable muscles were the most distal, single joint muscles, the most variable, the proximal. The biarticulate muscles, both proximal and distal exhibit higher variability than the single-joint muscles. Intra-subject variability (coefficient of variation) was reported to be for the gluteus maximus (± 52%), biceps femoris (± 62%), rectus femoris (± 51%), vastus lateralis (± 46%), lateral gastrocnemius (± 37%), medial gastrocnemius (± 33%), and tibialis anterior (± 33%). Inter-subject variability (normalised to mean amplitude of stride) for the gluteus maximus was ± 58%, biceps femoris ± 59%, rectus femoris ± 46%, vastus lateralis ± 44%, lateral gastrocnemius ± 57%, medial gastrocnemius ± 61%, and tibialis anterior ± 28%. Previous studies have reported 20-70% stride-to-stride variability (Yang and Winter, 1984; Arsenault et al., 1986). The proximal muscles are less active than the distal muscles and the lower level of mean activity results in a higher coefficient of variation.

As there is much variability in muscle activity during able-bodied walking, Arsenault et al. (1986a) investigated the number of strides needed to give a stable mean in EMG analysis. The study found that 3 strides provides as much reliable information as twelve. Correlations of 0.958 to 0.996 were reported for 3 strides compared to correlations of 0.993 to 0.999 reported for 10 strides.
Asymmetry

Most studies have limited their EMG analysis to only one leg per subject, usually the right, thus assuming symmetry in terms of muscle activity in able-bodied walking. Arsenault et al. (1986c) correlated EMG results for the soleus and rectus femoris between the left and right limbs of able-bodied subjects walking. The results showed very high correlations for the soleus, with slightly lower, but still significant correlations for the rectus femoris. This study concluded that even though there were slight differences between the muscles of the left and right limbs, this difference was not significant and that able-bodied subjects exhibited EMG muscle symmetry regardless of lower limb dominance. Olree and Vaughan (1995) also agreed there was ‘good symmetry’ between the EMG results of eight muscles. Thus, for able-bodied walking, there appears to be good muscle activation pattern symmetry.

3.6.2.3 Summary of muscle activity in able-bodied gait.

Many studies have looked at muscle activity during able-bodied walking. However, normalisation techniques and electrode placement have differed across studies making the results difficult to compare. The majority of studies have normalised EMG signals to 100% MVC even though they reported high intra-subject variation using this method as the EMG signal during a maximum voluntary contraction is dependent on many variables. The MVC is also a static contraction and walking is a dynamic activity. More recently, studies have normalised EMG signals to their mean or peak amplitude, reporting much lower intra-subject variability.

Studies reporting different walking speeds have noted changes in able-bodied lower limb muscle activity including onset and offset, and differences in peak magnitude. However, these studies were limited to slow, free and fast walking. The
majority of studies have measured muscle activity from the right leg only, but two studies reporting symmetry in able-bodied muscle activity agreed that there was good symmetry between limbs.

3.6.3 Muscle activity in amputee gait.

Lower limb prostheses easily provide static structural supports, but not the lost dynamic functions relating to muscle activity. Consequently, good locomotion by the amputee requires adaptation in the joints of the remaining lower limb, and thus compensation by the remaining musculature of the lower limbs. As a result of the lost ankle-foot function in below-knee amputees, compensation for the prosthetic limb occurs at the residual knee joint, foot-timing of the intact limb and in the muscle activity of both lower limbs (Breakey, 1976). For above-knee amputees, and the additional problem of the loss of a knee joint and surrounding musculature, further compensation mechanisms may be seen in addition to those reported for below-knee amputees.

3.6.3.1 Stability.

Compensatory muscle activity partially masks the primary gait deficit. It is difficult to look at below-knee amputees to determine the role of the plantar flexors due to compensatory problems and differences in the weight of the prosthetic shank compared to the intact shank. The suspension of the prosthetic shank may also affect knee movements. Proprioception and sensation are absent and motion of the ankle joint is usually excluded by prosthetic design.

Due to adaptation to the partial loss of a lower limb, atrophy of the bone and soft tissue in the residual limb usually occurs. Atrophy of the thigh is common in below-
knee amputees and may cause a decrease in thigh muscle strength, influencing the function of the stump and prosthesis. Renstrom et al. (1983a) investigated thigh muscle strength and atrophy in below-knee amputees using isokinetic dynamometer and muscle biopsies. No correlation with strength and cross-sectional area of the quadriceps was found, but there was significantly reduced knee flexion and extension strength on the residual limb compared to the intact limb, which was larger than the reduction in muscle cross-sectional area. The study did not reveal whether muscle hypertrophy was found on the intact limb. Torres-Moreno et al. (1997) reported both bone and muscle atrophy in the residual thigh of above-knee amputees and bone and muscle hypertrophy on the intact limb thigh. Besides muscle atrophy, changes in motor unit recruitment patterns may be of some importance for the reduction in muscle strength. Isometric and isokinetic knee flexion and extension strength were significantly correlated with step length, maximal walking speed and thigh circumference. This study concluded that below-knee amputees with a better preserved thigh muscle strength have a good walking capacity.

A further study by Renstrom et al. (1983b) looked at thigh muscle atrophy in below-knee amputees using muscle biopsies and CT scans. They found significantly fewer fast twitch fibres in the residual limb compared to the intact limb. The mean muscle fibre area of the vastus lateralis in the residual limb was 74% that of intact limb. Cross-sectional area of the thigh on the residual limb was 80% of the intact limb, quadriceps 66% of the intact limb and hamstrings 80% of the intact limb. Thus the wasting process seems to be more localised in the quadriceps of below-knee amputees than other thigh muscles (Sargeant et al., 1977; Young et al., 1980) due to changes in the gait pattern from walking with a prosthesis. Burger et al. (1996) reported atrophy and a decrease in fast twitch fibres in the gluteus maximus in above-knee amputees. One
of the main causes for impaired gait is the imbalance of the hip muscles following the removal of the femoral ends of the major muscles such as the hamstrings, adductors and rectus femoris. The muscles left over such as the iliopsoas, gluteus maximus and medius are left to act on the hip joint as agonists, only without the antagonists. Under such conditions of imbalance, muscle function is reduced and eventually ineffective. Muscle atrophy begins during the period of inactivity that starts sometimes before and continues immediately after amputation surgery (Ryser et al., 1988).

The ankle.

One study looking at able-bodied subjects before and after tibial nerve block to assess the role of the ankle plantar-flexor muscles during walking (Sutherland et al., 1980) reported that after the block, subjects were unable to transfer weight to the front part of the foot on the blocked side. Stance phase ankle dorsiflexion and knee flexion increased, and the duration of single stance was reduced on the blocked limb. Quadriceps muscle activity was prolonged to compensate for loss of knee stability due to loss of the ankle plantar flexion muscles. Step length reduced and there was a 23% reduction in walking velocity. Therefore, reduction in walking velocity of amputees may not be in order to reduce vertical ground reaction force, but may be due to the loss of the ankle plantar flexors. An increase in horizontal and vertical velocities during part of the gait cycle resulted in an excessive fall of the centre of mass indicating excessive energy expenditure to lift the centre of mass through the intact limb. The study concluded that the role of the ankle plantar flexors was to stabilise the knee and ankle, conserving energy by minimising the vertical oscillation of the centre of mass. When the ankle plantar flexors are missing as in lower limb amputees, this method of conserving energy cannot occur and the amputee has to rely on the remaining musculature to control gait.
In able-bodied subjects, plantar flexion of the ankle in late stance initiates hip and knee flexion to begin swing phase (Gray and Basmajian, 1968) and the swinging leg receives further energy from the hip flexors (Erberhart et al., 1954; Gray and Basmajian, 1968). However, in below-knee amputees, due to the loss of the ankle plantar flexors, greater action of the hip flexors is needed to initiate swing (Breakey, 1976). The increased duration of quadriceps muscle activity in the residual limb (Breakey, 1976) was explained by a compensation for the lost ankle function as seen in able-bodied subjects after a tibial nerve block (Sutherland et al., 1984). The calf muscles are normally active from 10-25% of gait cycle (Sutherland and Hagy, 1972) and are thought to have an important stabilising effect on the knee joint in mid-stance (Saunders et al., 1953; Perry, 1967). If the amputee’s muscle activity behaved as in able-bodied subjects, the knee and hip would tend to flex in mid-stance. Thus, the hip and knee extensors showed a prolonged period of activity in order to counteract flexion moments at the hip and knee joints. Whilst studies have reported the effects of the loss of the ankle joint on the remaining musculature in below-knee amputees, few have reported whether compensatory mechanisms occur in the surrounding ankle musculature of the intact limb by comparing the intact limb to able-bodied subjects. No study has reported muscle activity for the intact limb ankle in above-knee amputees to determine whether compensatory methods occur.

The knee.

As the amputee walks, GRFs act through the foot of the prosthesis to produce moments about the knee joint. The amputee’s musculature responds by attempting to balance these moments. For the residual limb, gluteus maximus, quadriceps and hamstrings were found to be active in early stance phases with able-bodied subjects (Erberhart et al., 1947; 1954; Battye and Joseph, 1966; Sutherland and Hagy, 1972).
However, the muscle activity in the amputees' residual limb was found to be of longer duration than able-bodied subjects. For the quadriceps, (rectus femoris, vastus lateralis, vastus medialis), muscle activity occurred from HS to 27% of the cycle (compared to 18% in able-bodied subjects). Hamstrings (medial and lateral) were active from HS to 25% of the cycle compared to 8% in able-bodied subjects (Breakey, 1976). The rectus femoris showed a longer duration of activity in early swing (55-80% of the cycle) in amputees compared to able-bodied subjects (55-70%). This is probably a result of the damping effect of the quadriceps on the heel rise of the prosthesis to counteract the increased activity of the hip flexors, and to also accelerate the leg in swing after maximal knee flexion.

For the intact limb, muscle activity of below-knee amputees was found to be similar to those previously reported for able-bodied subjects (Batty and Joseph, 1966). However, the small second peak of quadriceps activity often seen in able-bodied subjects at the transition from stance to swing (Batty and Joseph, 1966) was absent, but may be related to the relatively slow walking speed of the subjects decreasing the requirement for reactive stabilising forces at the knee. The hamstrings muscle activity was again comparable with able-bodied subjects (Milner at al., 1971). On looking at the differences in muscle activation pattern between the residual and intact limbs of below-knee amputees, Batyee and Joseph (1966) and Culham et al. (1986) reported that the hamstring muscles in the residual limb were active for longer than in the intact limb. For the intact limb of above-knee amputees, no study has reported muscle activity around the knee.
The hip.

The gluteus maximus was found to be active for 25% of the cycle of the residual limb in below-knee amputees compared to 12% in able-bodied subjects (University of California, 1953). The vastus lateralis was active for 8% longer than able-bodied subjects which may be due to an increased energy requirement for forward motion as a compensation for lost energy input on the residual limb (Breakey, 1976). This again shows that for the residual limb, the remaining muscles increase activity duration in below-knee amputees compared to able-bodied subjects.

Musculature surrounding the hip on the residual limb of above-knee amputees has been well documented. The above-knee amputation surgical procedure includes detachment and removal of the lower part of the hamstrings which leaves the gluteus maximus as the sole hip extensor. The hip extensors have an important role in both standing and walking (Jaegers, 1993). In above-knee amputees, the gluteus maximus stabilises the prosthetic knee mechanism and controls the stability of the stump-prosthesis unit (Inman et al., 1981; Hale, 1990; Jaegers, 1993). It also provides the main muscular force for the forward thrust of the body during the early part of the stance phase (Inman et al., 1981). Weakness in the gluteus maximus, therefore, results in not uncommon gait disturbances after above-knee amputation (Burger et al., 1996).

3.6.3.2 The effects of different prostheses on muscle activity.

Culham et al. (1986) investigated the effect on muscle activity patterns of two below-knee prostheses, the SACH foot (no ankle joint mechanism) and single axis foot (allows 15-20° plantarflexion and 5-8° dorsiflexion.) For the intact limb, the results showed the patterns of quadriceps and hamstrings were similar to able-bodied subjects.
and unaffected by changing the prosthesis. Peak hamstrings activity for the intact limb
occurred at HS with the SACH foot and at 10% ground contact with the single axis foot,
but did not differ in magnitude. For the residual limb, the muscle activity pattern
differed to both the intact limb and to able-bodied subjects (both muscles were active for
a greater percentage of the stance phase than for able-bodied subjects) and changed
depending on the prosthesis. For the SACH foot, there was peak hamstrings activity at
30% ground contact with peaks at 10% and 60% for single axis foot. With the SACH
foot, there was also more co-contraction of the quadriceps and hamstrings during mid-
stance, thus illustrating that the type of prosthesis will affect muscle activity in the
remaining musculature of the residual limb of lower limb amputees.

For the residual limb, the delay in the occurrence of peak quadriceps activity is
probably related to the lengthened period of initial double limb support. Body weight is
not fully accepted on the residual limb until 20-25% of gait cycle. The quadriceps
muscles are active throughout this period and a peak occurred at the beginning of
residual limb single support when the limb is at its most vulnerable. The prolonged
quadriceps activity may be related to the absence of the restraining action of the calf
muscles from HS to mid-stance, necessitating a greater contribution from the quadriceps
in order to stabilise the flexed knee (Breakey, 1976; Murray, 1978).

If a prosthetic ankle mechanism is further absent, as seen when using a SACH
instead of a foot allowing limited plantarflexion and dorsiflexion, increased duration of
quadriceps activity should be seen, as prolonged quadriceps activity is needed to
actively extend the knee and contribute to forward movement of the body. In addition,
heel rise with the SACH foot must occur earlier than with able-bodied gait. Early heel
rise would tend to decrease stability and may contribute to prolonged and higher levels of quadriceps activity.

For the hamstrings, prolonged activity may be related to the longer period of double support and the absence of the stabilising function of the triceps surae. With the SACH foot, there was higher hamstring activity at mid-stance than with the single axis foot. This could have been due to the rigidity of the ankle as the same findings were present in patients who had undergone ankle fusion (Mazur et al., 1979). A second hamstrings peak occurred at 60% ground contact with the single axis foot (which weighs 1.3 kg more than the SACH foot). This could have had an effect on muscle activity, but this has not been investigated.

Peeraer et al. (1990) looked at six muscles, rectus femoris, gluteus maximus, gluteus medius, adductor longus, tensor fascia latae and hamstrings, during above-knee amputee walking, downhill and uphill (6°/9°) and level walking with two different knee mechanisms (4 bar Otto Bock and Blatchford endolite hydraulic). Electromyography electrodes for the residual stump were built into the socket. The authors found that muscle activation pattern in above-knee amputees depends on the prosthesis used. Increased tensor fascia latae activity, primarily acting as a hip flexor at the end of stance, was reported. This study concluded that more work needs to be done on a number of subjects as by controlling the knee mechanism, it is possible that muscle activity will differ at the hip and stump. However, there were no comparisons shown between two prostheses, only one subject, only 20 minutes adaptation, and no mention of effects of different prostheses on intact limb muscle activity.
3.6.3.3 Differences in muscle activity between sedentary and active amputees.

Pinzur et al. (1991) investigated EMG (quadriceps and hamstring function) in limited and active dysvascular below-knee amputees during walking. They found that the quadriceps and hamstrings were active for 60% to 64.7% of the gait cycle for the active amputees compared to 44.6% to 53.8% for the limited walkers. The quadriceps muscles were activated 10% earlier in cycle for the active walkers. For the active walkers, there was a 10% delay in quadriceps activity in the residual limb (0.9 of cycle) compared to their intact limb (0.8 of cycle) and only a 3% delay between limbs for the limited walkers. For the hamstrings, there was a 4% delay between limbs for active walkers (0.87 residual limb v 0.83 intact limb) and a 3% delay between limbs for the limited walkers (0.87 residual limb v 0.84 intact limb). The limited walkers did not increase muscle activity of the remaining motors, decreasing quadriceps activity by 21.5% and hamstrings activity by 11.8% whereas active walkers decreased quadriceps activity by 2.8% and increased hamstring activity by 30%.

The active walkers were able to maintain quadriceps and increase hamstrings activity to compensate for their absent ankle motors whilst the limited walkers showed decreased muscle activity. The study concluded that the limited walking dysvascular below-knee amputees do not use their quadriceps and hamstrings for propulsion during walking and do not appear to reap the benefits from knee joint preservation.

3.6.3.4 Summary of muscle activity in amputee gait.

There has been a lack of studies reporting muscle activity of above-knee amputees, particularly looking at compensatory mechanisms on the intact leg. For below-knee amputees, the main problem stems from the loss of the ankle plantar flexors which stabilise the ankle and knee, reducing oscillation of the centre of mass and
therefore energy consumption during walking. In order to compensate for this, studies have shown that the quadriceps on the residual limb are active for longer than on the intact limb. Whilst some studies have shown there is muscle asymmetry between the residual and intact limbs of below-knee amputees, one has reported little muscle asymmetry and none have quantified this. However, if amputees exhibit greater loading on their intact limb, greater peak net joint moments and power outputs compared to their prosthetic limb, some muscle asymmetry is expected. Few studies have reported muscle activity in the intact limb of above-knee amputees and no studies have investigated what happens to the muscle activation patterns when greater demands such as increasing walking speed are placed on the amputee.

3.6.4 Summary of muscle activity.

Studies have previously quantified muscle activity during able-bodied walking, both in magnitude and onset and offset of the EMG signal. However, normalisation techniques (100% MVC and normalisation to peak or mean amplitude) have differed across studies making the results difficult to compare. High intra-subject variations have been reported normalising to MVC which has the added problem of being a static contraction and walking is a dynamic activity. More recently, studies have normalised EMG signals to their mean or peak amplitude, reporting much lower intra-subject variability. The majority of studies have measured able-bodied muscle activity from the right leg only, but two studies reporting symmetry in able-bodied muscle activity agreed that there was good symmetry between limbs. Studies have also reported that increases in walking speed change muscle activation patterns in able-bodied subjects in both magnitude and duration.
For below-knee amputees, studies have agreed that the main problem stems from the loss of the ankle plantar flexors. In order to compensate for this, studies have shown that the quadriceps on the residual limb are active for longer than on the intact limb. Whilst some studies have suggested there is muscle asymmetry between the residual and intact limbs of below-knee amputees, none found have quantified this. There has been a lack of studies reporting muscle activity of above-knee amputees, particularly looking at compensatory mechanisms on the intact leg. Also, no study has been found investigating what happens to the muscle activation patterns when greater demands such as increasing walking speed are placed on the amputee. If increases in walking speed alter muscle activation patterns as with able-bodied subjects, muscle asymmetry in amputees may increase which has implications for further designs of the prosthesis, having been previously shown that the type of prosthesis affects muscle activity.
3.7 Synthesis of literature review.

A review of the literature reveals important aspects of able-bodied gait providing information on limb loading, impact shock and shock attenuation, kinematic, kinetic and muscle activation patterns. Some information is also available on the effect of walking speed for selected gait variables in able-bodied gait, although this is not extensive. It has also been established that able-bodied subjects yield little asymmetry when walking.

There is less information, however, for below-knee amputees, and particularly for above-knee amputees. Very few studies have looked at gait asymmetry in lower limb amputees. Asymmetry measurements can be used to investigate gait problems and possibly establish the potential for increased future joint problems. By increasing walking speed, if asymmetrical gait problems are present, these problems may increase or reduce, thus giving an insight as to how unilateral lower limb amputees cope with gait asymmetry particularly under increasing demands. Whilst aspects of amputee gait have previously been investigated and amputees have been shown to be asymmetrical in their gait, no study has attempted to obtain data across a range of walking speeds for a number of measurement methods.

Studies have shown that the established unilateral lower limb amputee places more stress on their intact limb compared to their prosthetic limb and to able-bodied subjects by increasing limb loading magnitude and stance phase duration, increasing knee flexion angle at mid-stance, possibly placing greater stress on the muscles, and exhibiting greater ankle and knee joint moments and power output. Unilateral lower limb amputees have also been reported to experience greater amounts of joint degeneration in their intact limb compared to their residual limb and to able-bodied subjects. Whilst it is not certain as to what mechanism is responsible for this, it is
thought that the high demands and repeated high cyclic loading may cause wear on the joints. If the asymmetrical walking pattern changes with walking speed, this may provide further information as to the mechanism responsible for the high incidence of intact limb pain/joint degeneration in unilateral amputees. So far, no study has been found investigating the effect of walking speed on the asymmetrical walking pattern.

One aspect of amputee gait that has been the subject of limited investigation is impact shock and shock attenuation. It is thought that wear in the joints of the intact limb in unilateral lower limb amputees results in a reduced capacity for shock attenuation, possibly leading to reduced incoming impact shocks in order to protect that leg. Previous investigations on preosteoarthritic able-bodied patients have shown that they impact their affected limb more than their healthy limb. For able-bodied patients suffering from osteoarthritis or joint degeneration, there is an inability to fully attenuate shock from the shank to the head. As a consequence, incoming impact shock needs to be reduced on the affected limb. As no study found has previously looked at impact shock and shock attenuation asymmetry in lower limb amputees during walking, findings from able-bodied studies have led to the rise of a plausible theory. Those amputees not experiencing intact limb pain or joint degeneration may experience greater impact shocks on their intact limb compared to prosthetic limb, exhibiting impact shock asymmetry. This asymmetry, along with the previously established high intact limb loading, could be the mechanism responsible for leading to joint pain and/or joint degeneration in later life. Those amputees experiencing intact limb joint pain will have a reduced capacity to attenuate shock and as a consequence, reduce incoming impact shocks. Thus, impact shock asymmetry is also exhibited, but this time greater impact shock is seen on the prosthetic limb than intact limb. The mechanism by which reduced impact shock on the intact limb is achieved may be seen in differing joint kinematics.
such as increased knee flexion. By increasing walking speed, impact shock and shock attenuation asymmetry may change as the amputees try to cope with the increasing demands placed on them. This could also be reflected in their gait kinematics.

Thus, whilst there is full and comprehensive information about able-bodied gait, there are many gaps in the literature when looking at below-knee and particularly above-knee amputee gait. Whilst it is established that unilateral lower limb amputees exhibit gait asymmetry, little is known about the effect of increased walking speed on amputee gait asymmetry. There is a need to investigate selected aspects of amputee gait, particularly limb loading, impact shock and shock attenuation, joint kinematic, kinetic and muscle activation patterns, both in the intact and prosthetic limbs of amputees, and in comparison to able-bodied subjects. There is also a need to investigate the effect of increasing walking speed on these variables in order to establish how amputees cope with increasing demands made on them during walking.
4.0 The effect of speed on gait asymmetry during walking for above- and below-knee amputees.
4.0 The effect of speed on gait asymmetry during walking for above- and below-knee amputees.

This chapter is concerned with the effects of increased walking speed on loading and temporal asymmetry over a number of strides in unilateral amputee gait and address objective 1. The first study aims to compare the measurement of vertical ground reaction force (vGRF) obtained by shoes with in-built force sensors with that measured by a force platform. Force shoes generally underestimate vGRF as the position of the force sensors do not cover the whole of the foot, but are able to provide vGRF data over a number of consecutive steps enabling relative loading and temporal asymmetry to be measured. The second study aims to investigate the effect of walking speed on loading and temporal asymmetry in above- and below-knee amputees using the force shoes. These studies were performed in the Faculty of Rehabilitation, Academy of Physical Education, Warsaw.

4.1 Comparison of CDG force shoes with force platform.

4.1.1 Introduction.

Whilst force shoes currently only measure ground reaction forces in the vertical (z) direction, their advantage over the force platform is that they can measure over numerous consecutive strides. The CDG (Infotronic, NL) force shoe system consists of 8 force sensors, sensitive to the normal compression force, in-built in to the sole of the shoes which are worn barefoot. The force sensors are capacitive sensors each of size 45 x 45 x 3 mm and have a load range of 0-100 kgf (Figure 4.1). This system has previously been used in a number of clinical studies to give instant feedback and look at step-to-step variation and loading asymmetry (Krajnik et al., 1981; Hermens et al., 1986; Zilvold and Baumgartner, 1988).
One problem with this type of system is that the force sensors do not cover the whole of the foot and therefore, not all of the vertical ground reaction force is being measured. Few studies have reported the differences in vertical force measured by the force shoe systems and a force platform. Klajajic and Krajnik (1987) reported that the vertical force magnitude and pattern obtained by the force shoes differed very little to that measured by the force platform, but were not quantified. Another study reported differences ‘far below 10%’ for force magnitude collected by the two methods (Krajnik et al., 1981). Consequently, there is a need to assess how the vertical ground reaction force magnitude and pattern measured by the CDG sensor shoes compare to that measured by the force platform. The purpose of this study was to ascertain the differences in vGRF magnitude measured between the CDG sensor shoes and force platform in order to determine whether the CDG sensor shoes give a valid measurement of vGRF.

4.1.2 Methodology.

4.1.2.1 Apparatus.

The Infotronic CDG sensor shoes contained eight sensors (each sensitive to normally applied force, with a maximum sample rate of 50 Hz) built into the sole of each shoe (Figure 4.1). The shoes were worn over bare feet and were available in different sizes to account for differences in foot size. The shoes were connected to a data logger by cable and the data logger was worn around the subject’s waist (Plate 1) leaving subjects free to walk without any other impediment.
Figure 4.1 Infotronic CDG sensor shoes with 8 sensors.

The data were stored during the trials and then downloaded to a lap top computer using the Infotronic CDG software. The software summed the vertical force from each sensor to give an overall vGRF.

Plate 1. The Infotronic CDG system.
A Kistler force platform (Type 9281B11) was used to obtain vGRFs for comparison with the CDG data. Data were obtained using Bioware® (Kistler, Switzerland) software also sampling at 50 Hz.

4.1.2.2 Subjects and procedures.

Four able-bodied male subjects took part in the study (mean ± SD age 34.5 ± 9.57 years, height 1.78 ± 0.07 m and weight 77.0 ± 6.63 kg). The CDG shoes were fitted to the subject and connected to the data logger worn around the waist. Each subject was allowed to familiarise themselves walking with the shoes prior to testing. The subject then walked over the force platform (hitting with right foot only) at their natural walking cadence whilst wearing the CDG shoes. Data were recorded simultaneously from both the force platform and CDG shoes for each right foot step. Any trial where the force platform was targeted, or the foot was not fully on the force platform was discarded and the trial repeated. Six to seven trials were obtained for each subject.

4.1.2.3 Data analysis.

For the CDG data, all eight force sensors were summed to give the overall vGRF using the Infotronic CDG software and a threshold of 8 N. All CDG and Kistler force data were then exported and analysed in Excel. To synchronise the CDG and Kistler force data, the first vGRF value above the threshold of 8 N for both measuring devices was taken as the first instance of ground contact. Firstly, the six to seven CDG steps were normalised to 100% of the stance phase and then averaged. The Kistler vGRF steps were also normalised to 100% of the stance phase and then averaged. This was done for each subject in order to compare the average force curve patterns from the two systems. Secondly, all pairs of steps (CDG and Kistler force
data) for each subject were included in the analysis to assess reproducibility. Each pair of steps measured by the Kistler force platform and corresponding CDG© sensor shoe were analysed together. Reproducibility was assessed using the method error (Thorstensson, 1976) which is similar to the RMS error method where:

\[
\text{the method error (ME)} = \frac{\text{SD} (X_1 - X_2)}{\sqrt{2}}
\]

and is expressed by the coefficient of variation V where:

\[
V = \frac{\text{ME} \times 100}{(X_1 + X_2) / 2}
\]

which gives the coefficient of variation as a percentage of the combined mean of the two methods of data collection.
4.1.3 Results

Figure 4.2 shows an example of the raw data obtained for one subject and one step from each of the individual force sensors from the CDG® shoes, which are then summed to give the vGRF for that step. Figure 4.3 shows an example of the raw vGRF data for one different subject as measured by the Kistler force platform.

Figure 4.2 – An example of raw data obtained from one step of the CDG® sensor shoe.

Figure 4.3 - An example of raw data obtained from one step measured by the Kistler force platform.
The results show consistent similarities in vGRF data between the two measurement systems for the 4 subjects (Figure 4.4). The small difference between the two, however, is not consistent across individuals. In all cases, the CDG shoes produce slightly lower force values than the Kistler force platform. The time duration of the vGRF measured by both systems was consistently similar (within ± 1 data point) for all steps.

Figure 4.4 - Comparison of mean CDG and Kistler vGRF curves for each subject walking at a natural cadence.
The results show similarities in terms of the vGRF curve for the two measurement systems.

Reproducibility calculated for all subjects and all steps (Thorstensson, 1976) showed an average variation between systems of 7.05% with the CDG shoes underestimating the vGRF measured by the force platform. The range of the error (underestimation of CGD® sensor shoes) was shown to be between 4.47% and 24.62%. The higher values were found for differences between the Fz2 peaks of the two measurement systems, with the lower values for differences between the Fz1 peaks.
4.1.4 Discussion.

The results showed a relationship between the vertical ground reaction force measured by the CDG® force shoes and the Kistler force platform. There was an average difference between the two systems of 7.05% established by the error method (Thorstensson, 1976). This, when normalising vGRF to body weight, yields an error value of less than 0.1 BW when using the CDG® force shoes compared to the force platform. This variation of less than 10% is comparable to a previous study (Krajnik et al., 1981) reporting vGRFs measured by the two systems. However, whilst the difference between the two systems was lower than 7.05% when calculating the Fz1 peak, the difference between the two systems for the Fz2 peak was greater than 20%. This could have been due to the fit of the CDG® shoe at the front of the foot, and whilst there is a good estimation of the Fz1 force using the CDG® system, there is a problem reporting the Fz2 peak. Thus, in subsequent studies, only values for the Fz1 peak will be reported.

The CDG® system gives a lower vertical GRF value than the force platform due to the position of the force sensors. As the sole of the shoe cannot be completely rigid, the force sensors do not cover the whole of the foot. Therefore, not all of the vGRF is being measured. The differences between the two systems measured was not consistent. Therefore, these differences could possibly be due to the differences in foot size and how well the CDG® sensor shoes fit the foot, or differences in foot structure. Although there are differences in sensor shoe sizes available to account for differences in foot size, small differences in the fit at the front of the shoe may show differences in the second loading peak, or Fz2, compared to the vGRF measured by the force platform. These differences for some subjects appeared to be larger than the differences between the two measuring systems for the initial loading peak, or Fz1 peak. This, in turn will
affect the magnitude of the calculated vertical impulse. As the impulse is the integral of the vGRF over time, the increasing error in vGRF as measured by the CDG® shoes at push-off will affect the impulse magnitude. This needs to be taken into account when discussing vertical impulse results.

A problem with the CDG® system is the sample rate. Due to the small sample rate of 50 Hz, the heel strike peak was absent for most trials. However, if only the weight acceptance peak (Fz1) and push off peak (Fz2) are needed, then the system is adequate. Therefore, the use of the CDG® sensor shoes provides a means of measuring vGRF over a number of continuous strides with a variation of less than 10% between this system and sampling with the force platform. Thus, whilst heel strike peak cannot be measured, the Fz1 and Fz2 peaks, often used in assessing pathological gait, can be measured from both limbs at the same time and over a continuous number of strides to calculate loading asymmetry and step-to-step variation.

4.1.5 Conclusion.

This study showed that the Infotronic CDG® sensor shoes can be used to provide an estimation of vGRF during walking. Whilst they have been shown to underestimate the vGRF measured by the force platform by an average of 7.05% (with a range of 4.47 to 24.62%), this yields an average error of less than 0.1 BW. The vertical Fz1 peak can be measured by the sensor shoes with good estimation, whilst the Fz2 peak can give up to a 20% underestimation of vertical GRF due to the fit of the foot in the front of the shoe. The advantage the CDG® shoes have over the force platform is that vGRF can be obtained from consecutive steps from both feet over a number of strides. This method of measurement has been shown to yield repeatable vGRF curves. Thus, it is deemed that the CDG® system can be used in a further study to investigate
the effect of walking speed on vGRF, temporal parameters and asymmetry in unilateral amputees.
4.2 The effect of walking speed on loading and temporal asymmetry in above- and below-knee amputees.

4.2.1 Introduction.

Many studies have previously looked at ground reaction force loading on both the intact and prosthetic limbs of unilateral amputees. The results have shown that amputees load their prosthetic limb less than able-bodied subjects load their limbs, whilst their intact limb is loaded more than able-bodied subjects (Engsberg et al., 1991; Engsberg et al., 1993) thus illustrating that unilateral amputees exhibit asymmetrical loading whilst walking. The effects of increasing walking speed on either vertical ground reaction force or loading asymmetry have not been previously reported for adult amputees. Only one study has reported increases in vGRF for both the prosthetic and intact limbs of above-knee amputee children during walking (Zernicke et al., 1985). This study showed that whilst vGRF increased with walking speed for each limb, increases were greater on the intact limb. Excessive loading on the intact limb is thought to be related to the joint pain/ degeneration previously reported in the intact limb of unilateral below-knee amputees (Hungerford and Cockin, 1975; Burke et al., 1978).

Temporal variables of gait have also indicated asymmetry. A longer intact limb stance phase and shorter swing phase compared to the prosthetic limb have previously been reported (Breakey, 1976; Murray et al., 1983). Thus, amputees not only load their intact limb more than their prosthetic limb, but also spend longer on it illustrating both loading and temporal asymmetry. Studies have investigated the effect of speed on temporal variables in lower limb amputees, finding step, swing and stance times to reduce with increasing speed (Murray et al., 1983; Isakov et al., 1996). Only one study (Isakov et al., 1996) has looked at the effect of speed on temporal asymmetry, finding no significant difference between the two walking speeds used (free and fast cadence).
As only this study has been found reporting the effect of walking speed on temporal asymmetry and only two walking speeds were used, it may be that a more detailed investigation is needed. The purpose of this study was to determine the level of temporal and loading asymmetry during walking in above- and below-knee amputees, and to investigate the effect of increasing walking speed on that asymmetry.

It is hypothesised that:

**Hypothesis 1:** Above-knee amputees experience greater loading on their intact limb compared to their prosthetic limb.

**Hypothesis 2:** Below-knee amputees experience greater loading on their intact limb compared to their prosthetic limb.

**Hypothesis 3:** Amputee and able-bodied loading asymmetry do not increase with increasing walking speed.

**Hypothesis 4:** Above-knee amputees have a longer stance time and shorter swing time on their intact limb compared to both their prosthetic limb and to able-bodied subjects.

**Hypothesis 5:** Below-knee amputees have a longer stance time and shorter swing time on their intact limb compared to both their prosthetic limb and to able-bodied subjects.

**Hypothesis 6:** Amputee and able-bodied temporal asymmetry do not increase with increasing walking speed.
4.2.2 Methodology.

4.2.2.1 Apparatus.

The same CDG® sensor shoes as used in study 4.1 were used by all subjects to measure vertical ground reaction force and temporal variables from consecutive footfalls. The shoes were fitted to the subject, a range of shoes were available to account for differences in foot size, and connected to a data logger by a cable which was worn around the subject’s waist. The subject was then free to walk without any other impedement (Plate 2).

Plate 2. An amputee walking using the Infotronic CDG system.

4.2.2.2 Subjects and Procedure

The amputees were all unilateral established highly active males and females. The below-knee amputees (n=4, 3 males and 1 female) had a mean (±S.D.) age and time as an amputee from trauma of 29.0 (±18.9) years and 22.3 (±7.6) years, respectively. The above-knee amputees (n=4, 3 males and 1 female) had a mean (±S.D.) age and time
as an amputee from trauma of 31.5 (±10.9) years and 19.0 (±5.23) years, respectively.

Able-bodied subjects (n=6) had a mean (±S.D.) age of 32.3 (±9.30) years and were also highly active. All above-knee amputees wore an Otto-Bock hinge knee prosthesis with SACH foot, and all below-knee amputees wore a SACH foot. After the CDG® sensor shoes were fitted over bare feet, all subjects were allowed to familiarise themselves with the shoes and walking at the different pre-determined walking speeds of 0.5, 0.9, 1.2 m.s⁻¹ and their maximum walking speed. Data were collected during walking (up to 20 strides) whilst wearing the CDG® sensor shoes (sampled at 50 Hz) and stored in the data logger before being downloaded to a computer. Mid-way along the walkway of 15-25 m, the floor was marked with tape at 2, 4 and 6 m intervals, and the time taken for the subject to pass these points was recorded. Any trial which was not within ±3% of the pre-determined speed was disregarded and repeated. Data were collected for both legs for between 12 and 20 steps for each subject at each walking speed.

4.2.2.3 Data Analysis

The Infotronic CDG® software was initially used to download the CDG® data to a laptop computer. The first three and last three steps for each foot and for each trial were disregarded. Using custom written software, stance time (time from heel strike to toe off), swing time (time from toe off to heel strike), step time (time from heel strike on one foot to heel strike on the contralateral foot), initial vertical ground reaction force peak (Fz1) and vertical impulse (the integral of the vertical ground reaction force over time for the duration of the stance phase) were calculated. The data were analysed over 9-17 steps for each subject at each speed. Asymmetry between the left (L) and right (R) limbs for the able-bodied subjects, and between the intact and prosthetic limbs of the amputees was calculated for each consecutive step using the absolute symmetry index (ASI)
where:

\[
ASI = \frac{(L-R)}{0.5(L+R)} \times 100
\]

(Herzog et al., 1989) and then averaged. As asymmetry between the left and right limbs of able-bodied subjects was calculated to be less than 10% for all variables, an average of the two limbs was calculated for each variable.

The data were initially tested for normality and homogeneity. Correlations were performed for each variable between the intact and prosthetic limbs of the above- and below-knee amputees to ensure they were independent, and thus did not violate the assumptions of an analysis of variance test. There was no significant correlation at the 5% level between the prosthetic and intact limbs either for above- or below-knee amputees for any variable. The intact and prosthetic limbs of the amputees were thus deemed to be independent and could therefore be included as separate groups in a two-way analysis of variance.

Each variable was then analysed using a two-way analysis of variance to investigate (i) differences between groups, (ii) the effect of walking speed and (iii) any group by speed interaction. Four walking speeds (0.5, 0.9, 1.2 m.s\(^{-1}\) and maximum. The maximum ranged from 1.5 to 1.9 m.s\(^{-1}\)) and five groups were used, the groups being: (i) the average of both limbs for able-bodied subjects, (ii) the below-knee amputee intact limb, (iii) the below-knee amputee prosthetic limb, (iv) the above-knee amputee intact limb, (v) the above-knee amputee prosthetic limb. The analysis of variance model included group (between-factor) and speed (within-factor) and was performed for each variable separately (i.e. Fz1, impulse, stance, swing and step time limb values) using the mean value of the 9-17 steps for each subject. Thus, the results of group, speed and
group by speed interaction effects will be discussed separately for each variable. The level of significance was set at 5%.

For asymmetry, a two-way analysis of variance was also performed to investigate (i) differences between groups (between-factor), (ii) the effect of walking speed on each variable (within-factor) and (iii) any group by speed interaction. Four walking speeds (0.5, 0.9, 1.2 m.s\(^{-1}\) and maximum) and three groups were used: (i) asymmetry calculated between the left and right limbs of able-bodied subjects, (ii) asymmetry calculated between the prosthetic and intact limbs of below-knee amputees and (iii) asymmetry calculated between the prosthetic and intact limbs of above-knee amputees. The two-way analysis of variance was performed for each variable (Fz1 asymmetry, impulse asymmetry and stance, swing and step time asymmetry) using the averaged asymmetry values calculated from consecutive steps for each subject. Level of significance was set at 5%. Thus the results of group, speed and group by speed effects will be discussed separately for each asymmetry variable calculated.

All statistical analyses were performed using SPSS (version 9.0) which gave the option of including calculations of the statistical power of the test and allowed for different numbers of subjects in each group. The coefficient of variation was used to report intra- and inter-subject variability. Level of significance was again set at 5%. For all two-way analysis variance tests, a post-hoc Tukey HSD test was performed.
4.2.3 Results.

The results for the maximum walking speed which ranged from 1.5 m.s\(^{-1}\) in the amputees to 1.9 m.s\(^{-1}\) in the able-bodied subjects were compared directly, even though the speeds differed, because the effect of their maximum possible walking speed on the chosen variables for each individual needed to be seen. Whilst it is appreciated that these speeds differed from individual to individual, the aim was to investigate the effect of their absolute maximum walking speed as an indication of the maximum demands placed on that individual during walking. Thus, all maximum values will be compared across individuals, marked separately on the graphs by a discontinued line //.

For all the amputees, a greater Fz1 force was produced on their intact limb compared to their prosthetic limb (Figure 4.6a). The Fz1 peak was seen to increase with walking speed for able-bodied subjects and the intact limb of above- and below-knee amputees. There was an increase in Fz1 asymmetry with speed for the amputees, with both the amputee groups observed to be similar and more asymmetrical than the able-bodied subjects (Figure 4.7b). The data for these graphs is referred to in tables 4.1 to 4.4.

Figure 4.6 – An example of raw vGRF data from a) the intact and b) prosthetic limb of an above-knee amputee walking at 1.2 m.s\(^{-1}\).
Figure 4.7 - vGRF and vertical impulse values and respective asymmetries during walking for able-bodied subjects (thick black line), above-knee amputees (blue square) and below-knee amputees (red diamond). Dotted line indicates prosthetic limb, solid intact limb.

Greater impulse was observed to be produced on the amputees’ intact limb compared to their prosthetic limb and to the able-bodied subjects (Figure 4.6c). Impulse reduced as walking speed increased. For impulse asymmetry (Figure 4.6d), above-knee amputees were more asymmetrical than below-knee amputees and able-bodied subjects. However, there appeared to be no effect of speed on impulse asymmetry (Figure 4.6d).
Table 4.1 - Mean (± SD) of vertical ground reaction force Fz1 peak for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Below-knee amputees</th>
<th>Above-knee amputees</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prosthetic limb</td>
<td>Intact limb</td>
<td>Prosthetic limb</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Intact limb</td>
</tr>
<tr>
<td>0.5</td>
<td>1.34</td>
<td>1.16</td>
<td>1.40</td>
</tr>
<tr>
<td>[0.08]</td>
<td>[0.07]</td>
<td>[0.23]</td>
<td>[0.95]</td>
</tr>
<tr>
<td>0.9</td>
<td>1.32</td>
<td>1.17</td>
<td>1.40</td>
</tr>
<tr>
<td>[0.08]</td>
<td>[0.05]</td>
<td>[0.23]</td>
<td>[0.98]</td>
</tr>
<tr>
<td>1.2</td>
<td>1.34</td>
<td>1.18</td>
<td>1.47</td>
</tr>
<tr>
<td>[0.08]</td>
<td>[0.08]</td>
<td>[0.2]</td>
<td>[0.99]</td>
</tr>
<tr>
<td>max</td>
<td>1.65</td>
<td>1.23</td>
<td>1.68</td>
</tr>
<tr>
<td>[0.08]</td>
<td>[0.08]</td>
<td>[0.32]</td>
<td>[0.14]</td>
</tr>
</tbody>
</table>

Table 4.2 - Mean (± SD) of force asymmetry as indicated by the ASI for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Below-knee amputees</th>
<th>Above-knee amputees</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Prosthetic limb</td>
<td>Intact limb</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Prosthetic limb</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Intact limb</td>
</tr>
<tr>
<td>0.5</td>
<td>6.95</td>
<td>17.54</td>
<td>18.72</td>
</tr>
<tr>
<td>[3.72]</td>
<td>[19.4]</td>
<td>[16.66]</td>
<td></td>
</tr>
<tr>
<td>0.9</td>
<td>7.29</td>
<td>16.96</td>
<td>16.88</td>
</tr>
<tr>
<td>[3.62]</td>
<td>[18.49]</td>
<td>[15.40]</td>
<td></td>
</tr>
<tr>
<td>1.2</td>
<td>8.00</td>
<td>22.59</td>
<td>20.19</td>
</tr>
<tr>
<td>[4.19]</td>
<td>[18.44]</td>
<td>[14.90]</td>
<td></td>
</tr>
<tr>
<td>max</td>
<td>10.82</td>
<td>29.34</td>
<td>28.89</td>
</tr>
<tr>
<td>[4.74]</td>
<td>[19.70]</td>
<td>[23.23]</td>
<td></td>
</tr>
</tbody>
</table>

Table 4.3 - Mean (± SD) of impulse for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Below-knee amputees</th>
<th>Above-knee amputees</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prosthetic limb</td>
<td>Intact limb</td>
<td>Prosthetic limb</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Intact limb</td>
</tr>
<tr>
<td>0.5</td>
<td>1.10</td>
<td>0.78</td>
<td>1.11</td>
</tr>
<tr>
<td>[0.14]</td>
<td>[0.09]</td>
<td>[0.12]</td>
<td>[0.67]</td>
</tr>
<tr>
<td>0.9</td>
<td>0.92</td>
<td>0.70</td>
<td>0.95</td>
</tr>
<tr>
<td>[0.08]</td>
<td>[0.07]</td>
<td>[0.19]</td>
<td>[0.59]</td>
</tr>
<tr>
<td>1.2</td>
<td>0.70</td>
<td>0.61</td>
<td>0.84</td>
</tr>
<tr>
<td>[0.05]</td>
<td>[0.01]</td>
<td>[0.15]</td>
<td>[0.55]</td>
</tr>
<tr>
<td>max</td>
<td>0.52</td>
<td>0.49</td>
<td>0.67</td>
</tr>
<tr>
<td>[0.05]</td>
<td>[0.07]</td>
<td>[0.13]</td>
<td>[0.49]</td>
</tr>
</tbody>
</table>
Table 4.4 - Mean (± SD) of impulse asymmetry as indicated by the ASI for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Below-knee amputees</th>
<th>Above-knee amputees</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>8.91 [4.96]</td>
<td>31.73 [10.39]</td>
<td>53.94 [36.64]</td>
</tr>
<tr>
<td>1.2</td>
<td>7.67 [4.1]</td>
<td>30.50 [15.39]</td>
<td>51.48 [23.97]</td>
</tr>
<tr>
<td>max</td>
<td>11.86 [4.79]</td>
<td>30.65 [15.62]</td>
<td>51.98 [20.03]</td>
</tr>
</tbody>
</table>
For the stance phase, all amputees spent a greater time on their intact limb than their prosthetic limb. Stance time duration was found to reduce with speed (Figure 4.7a).

![Graphs showing stance, swing, and step time values and absolute symmetry index during walking for able-bodied subjects (thick black line), above-knee amputees (blue square) and below-knee amputees (red diamond). Dotted line indicates prosthetic limb, solid intact limb.]

Figure 4.8 - Stance, swing and step time values and absolute symmetry index during walking for able-bodied subjects (thick black line), above-knee amputees (blue square) and below-knee amputees (red diamond). Dotted line indicates prosthetic limb, solid intact limb.
For stance time, above-knee amputees were seen to be more asymmetrical than below-knee amputees and able-bodied subjects. Stance time asymmetry decreased slightly with walking speed (Figure 4.7b). The data for these graphs are referred to in tables 4.5 to 4.10.

Table 4.5 - Mean (± SD) of impulse for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Below-knee amputees</th>
<th>Above-knee amputees</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stance time (s)</td>
<td>Prosthetic limb</td>
<td>Intact limb</td>
</tr>
<tr>
<td>0.5</td>
<td>1.09</td>
<td>1.01</td>
<td>1.10</td>
</tr>
<tr>
<td></td>
<td>[0.08]</td>
<td>[0.07]</td>
<td>[0.12]</td>
</tr>
<tr>
<td>0.9</td>
<td>0.93</td>
<td>0.89</td>
<td>0.95</td>
</tr>
<tr>
<td></td>
<td>[0.06]</td>
<td>[0.08]</td>
<td>[0.12]</td>
</tr>
<tr>
<td>1.2</td>
<td>0.75</td>
<td>0.75</td>
<td>0.79</td>
</tr>
<tr>
<td></td>
<td>[0.03]</td>
<td>[0.04]</td>
<td>[0.02]</td>
</tr>
<tr>
<td>max</td>
<td>0.53</td>
<td>0.62</td>
<td>0.64</td>
</tr>
<tr>
<td></td>
<td>[0.03]</td>
<td>[0.08]</td>
<td>[0.09]</td>
</tr>
</tbody>
</table>

Table 4.6 - Mean (± SD) of impulse asymmetry as indicated by the ASI for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Below-knee amputees</th>
<th>Above-knee amputees</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stance time ASI (%)</td>
<td>Prosthetic limb</td>
<td>Intact limb</td>
</tr>
<tr>
<td>0.5</td>
<td>2.98</td>
<td>5.47</td>
<td>33.49</td>
</tr>
<tr>
<td></td>
<td>[2.29]</td>
<td>[4.68]</td>
<td>[14.50]</td>
</tr>
<tr>
<td>0.9</td>
<td>2.60</td>
<td>6.27</td>
<td>25.80</td>
</tr>
<tr>
<td></td>
<td>[1.84]</td>
<td>[4.50]</td>
<td>[9.1]</td>
</tr>
<tr>
<td>1.2</td>
<td>2.78</td>
<td>5.15</td>
<td>23.92</td>
</tr>
<tr>
<td></td>
<td>[2.11]</td>
<td>[3.54]</td>
<td>[5.8]</td>
</tr>
<tr>
<td>max</td>
<td>3.09</td>
<td>3.52</td>
<td>23.42</td>
</tr>
<tr>
<td></td>
<td>[1.35]</td>
<td>[3.72]</td>
<td>[3.68]</td>
</tr>
</tbody>
</table>

All amputees exhibited a reduced intact limb swing phase compared to both their
prosthetic limb and to able-bodied subjects (Figure 4.7c). Swing phase duration decreased with increasing walking speed. For swing time asymmetry, again the above-knee amputees were seen to be more asymmetrical than below-knee amputees and able-bodied subjects (Figure 4.7d). Swing phase asymmetry was also seen to reduce slightly with increasing walking speed.

Table 4.7 - Mean (± SD) of swing time for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Swing time (s)</th>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Below-knee amputees</th>
<th>Above-knee amputees</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Prosthetic limb</td>
<td>Intact limb</td>
<td>Prosthetic limb</td>
</tr>
<tr>
<td>0.5</td>
<td></td>
<td>0.53</td>
<td>0.54</td>
<td>0.48</td>
</tr>
<tr>
<td></td>
<td></td>
<td>[0.03]</td>
<td>[0.08]</td>
<td>[0.04]</td>
</tr>
<tr>
<td>0.9</td>
<td></td>
<td>0.48</td>
<td>0.48</td>
<td>0.42</td>
</tr>
<tr>
<td></td>
<td></td>
<td>[0.04]</td>
<td>[0.10]</td>
<td>[0.05]</td>
</tr>
<tr>
<td>1.2</td>
<td></td>
<td>0.42</td>
<td>0.44</td>
<td>0.40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>[0.04]</td>
<td>[0.04]</td>
<td>[0.03]</td>
</tr>
<tr>
<td>max</td>
<td></td>
<td>0.37</td>
<td>0.37</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td></td>
<td>[0.04]</td>
<td>[0.02]</td>
<td>[0.04]</td>
</tr>
</tbody>
</table>

Table 4.8 - Mean (± SD) of swing time asymmetry as indicated by the ASI for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Swing time ASI (%)</th>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Below-knee amputees</th>
<th>Above-knee amputees</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td></td>
<td>3.72</td>
<td>10.30</td>
<td>64.56</td>
</tr>
<tr>
<td></td>
<td></td>
<td>[2.02]</td>
<td>[9.20]</td>
<td>[30.27]</td>
</tr>
<tr>
<td>0.9</td>
<td></td>
<td>3.62</td>
<td>12.27</td>
<td>50.39</td>
</tr>
<tr>
<td></td>
<td></td>
<td>[3.30]</td>
<td>[8.23]</td>
<td>[19.23]</td>
</tr>
<tr>
<td>1.2</td>
<td></td>
<td>4.36</td>
<td>10.74</td>
<td>42.17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>[4.23]</td>
<td>[4.07]</td>
<td>[10.93]</td>
</tr>
<tr>
<td>max</td>
<td></td>
<td>5.47</td>
<td>8.06</td>
<td>37.38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>[2.26]</td>
<td>[11.10]</td>
<td>[3.37]</td>
</tr>
</tbody>
</table>

For step times, the amputees spent longer stepping onto their prosthetic limb
than intact limb. Step time was seen to reduce slightly with walking speed (Figure 4.7e).

For step time asymmetry, again the above-knee amputees were seen to be more asymmetrical than below-knee amputees and able-bodied subjects but speed did not appear to have an effect on step time asymmetry (Figure 4.7f).

Table 4.9 - Mean (± SD) of step times for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Step time (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prosthetic limb</td>
<td>Intact limb</td>
</tr>
<tr>
<td>0.5</td>
<td>0.80</td>
<td>0.82</td>
</tr>
<tr>
<td></td>
<td>[0.03]</td>
<td>[0.08]</td>
</tr>
<tr>
<td>0.9</td>
<td>0.71</td>
<td>0.72</td>
</tr>
<tr>
<td></td>
<td>[0.03]</td>
<td>[0.06]</td>
</tr>
<tr>
<td>1.2</td>
<td>0.60</td>
<td>0.61</td>
</tr>
<tr>
<td></td>
<td>[0.02]</td>
<td>[0.03]</td>
</tr>
<tr>
<td>max</td>
<td>0.45</td>
<td>0.51</td>
</tr>
<tr>
<td></td>
<td>[0.03]</td>
<td>[0.05]</td>
</tr>
</tbody>
</table>

Table 4.10 - Mean (± SD) of step time asymmetry as indicated by the ASI for able-bodied subjects, above- and below-knee amputees with increasing walking speed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Able-bodied Subjects</th>
<th>Step time ASI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prosthetic limb</td>
<td>Below-knee amputees</td>
</tr>
<tr>
<td>0.5</td>
<td>3.33</td>
<td>5.20</td>
</tr>
<tr>
<td></td>
<td>[1.73]</td>
<td>[5.42]</td>
</tr>
<tr>
<td>0.9</td>
<td>2.74</td>
<td>5.20</td>
</tr>
<tr>
<td></td>
<td>[3.13]</td>
<td>[3.61]</td>
</tr>
<tr>
<td>1.2</td>
<td>1.74</td>
<td>2.48</td>
</tr>
<tr>
<td></td>
<td>[1.57]</td>
<td>[3.93]</td>
</tr>
<tr>
<td>max</td>
<td>2.25</td>
<td>1.98</td>
</tr>
<tr>
<td></td>
<td>[3.60]</td>
<td>[4.94]</td>
</tr>
</tbody>
</table>

The results of the two-way analysis of variance will now be presented. The exact p values for each significant test will also be presented. A p value of p = 0.000 does not represent p = 0, rather that p is less than 0.0005. As all other p values are given exactly,
this convention of reporting p to three decimal places was undertaken for consistency.
Results of the two-way analysis of variance showed significant differences between the
groups for the Fz1 peak (F_{5, 22} = 5.36, p= 0.002). The post-hoc test showed group
differences between the below-knee amputee intact and prosthetic limbs (p=0.050),
between the below-knee amputee prosthetic limb and able-bodied subjects (p=0.007)
and between the above-knee amputee prosthetic limb and able-bodied subjects
(p=0.008). There was also a significant effect of speed on the Fz1 peak (F_{3, 66} = 57.31,
p= 0.000) and a significant group by speed interaction (F_{15, 66} = 4.10, p= 0.000). Thus,
the intact limb of the amputees was more affected by walking speed than the prosthetic
limb and the limbs of able-bodied subjects. For Fz1 asymmetry, the power of the test
between the groups was low (0.26) and this could explain why there was no significant
difference (F_{2, 11} = 1.55, p= 0.256) between groups. There was a significant effect of
walking speed on Fz1 asymmetry (F_{3, 33} = 5.45, p= 0.004), asymmetry increasing with
walking speed for all groups, but no significant interaction.

For vertical impulse, there was a significant difference between the groups (F_{5, 22}
= 8.75, p= 0.000). The post-hoc test revealed a difference between the below-knee
amputee intact and prosthetic limbs (p=0.049), between the above-knee amputee intact
and prosthetic limbs (p=0.000), between the below-knee amputee prosthetic limb and
able-bodied subjects (p=0.017), and between the above-knee amputee prosthetic limb
and able-bodied subjects (p=0.002). There was also a significant effect of speed (F_{3, 66} =
158.18, p= 0.000) and a significant interaction (F_{15, 66} = 7.01, p= 0.000). Again, vertical
impulse on the intact limb of the amputees and the limbs of the able-bodied subjects
was more affected by speed than on the amputees’ prosthetic limb. For impulse
asymmetry, there was again a significant difference between groups (F_{2, 11} = 8.86, p=
0.005), the post-hoc test revealing that the above-knee amputees were more
asymmetrical than the able-bodied subjects (p=0.004). However, there was no effect of walking speed on vertical impulse (F_{3,33} = 0.58, p= 0.63) and no significant interaction.

For the temporal variables, there was a significant difference between the groups in terms of stance time duration (F_{5,22} = 17.10, p= 0.000). From the post-hoc analysis, the above-knee amputee intact limb was found to differ from both the prosthetic limb (p=0.000) and to able-bodied subjects (p=0.000). Speed was found to have a significant effect, decreasing stance time with increasing walking speed (F_{3,66} = 155.94, p= 0.000). There was also a significant interaction of group by speed (F_{15,66} = 2.74, p= 0.002), the amputees’ intact limb and able-bodied stance phase being more affected by speed than the amputees’ prosthetic limb. There was also a significant difference between groups for stance time asymmetry (F_{2,11} = 49.81, p= 0.000), the post-hoc test showing that the above-knee amputees were more asymmetrical than the below-knee amputees (p=0.000) and able-bodied subjects (p=0.000). There was no significant effect of speed on stance time asymmetry (F_{3,33} = 1.24, p= 0.311), but the power of the test was low (0.30) and this may account for a non-significant effect of speed. There was no significant group by speed interaction.

For swing time duration, there was a significant difference between the groups (F_{5,22} = 7.91, p= 0.000), the post-hoc test showing differences between the above-knee amputee intact and prosthetic limbs (p=0.000) and between the above-knee amputee prosthetic limb and able-bodied subjects (p=0.003). There was also a significant effect of speed (F_{3,66} = 46.65, p= 0.000) and a significant interaction (F_{15,66} = 2.16, p= 0.017). This time, the amputees’ prosthetic limb was more affected by speed than their intact limb and able-bodied subjects. For swing time asymmetry, again there was a significant difference between groups (F_{2,11} = 32.31, p= 0.000), the post-hoc test revealing
differences between the below- and above-knee amputees (p=0.000) and between the above-knee amputees and able-bodied subjects (p=0.000). There was a significant effect of speed ($F_{3,33} = 3.94$, $p = 0.017$). There was also an interaction between group and speed ($F_{6,33} = 3.83$, $p = 0.005$) showing that swing phase asymmetry was more affected by speed for above-knee amputees than for the below-knee amputees and able-bodied subjects.

For step time duration, there was a significant difference between groups ($F_{5,22} = 15.00$, $p = 0.000$), the post-hoc test showing differences between the above-knee amputee prosthetic and intact limbs ($p=0.000$), and between the above-knee amputee prosthetic limb and able-bodied subjects ($p=0.000$). There was also a significant effect of speed ($F_{3,66} = 235.14$, $p = 0.000$) and a significant interaction ($F_{15,66} = 4.27$, $p = 0.000$), again the amputees’ prosthetic limb being more affected by speed than their intact limb or for able-bodied subjects. Step time asymmetry, whilst significant between groups ($F_{2,11} = 31.56$, $p = 0.000$), above-knee amputees were shown to be more asymmetrical than below-knee amputees ($p=0.000$) and able-bodied subjects ($p=0.000$) from the results of the post-hoc test, was not found to be affected by speed. However, the power of the test was low (0.23) and this could be a possible reason as to why no significant effect of speed was found.
4.2.4 Discussion.

This study aimed to investigate the level of temporal and loading asymmetry during walking in above- and below-knee amputees, and to investigate the effect of increased walking speed on that asymmetry. Whilst there were low subject numbers in the amputee groups due to lack of availability of healthy, highly active trauma amputees wearing similar prostheses, inter-subject variability was low for walking at maximum speed (Fz1 peak c.v. = ± 6.5% for below-knee, ± 11.4 % for above-knee amputees; stance time c.v. = ±12.9% for below-knee, ± 11.1% for above-knee amputees). All amputees in each group wore a similar prosthesis (SACH foot for below-knee amputees, Otto-Bock hinge knee and SACH foot for above-knee amputees), thus it was deemed that the below-knee and above-knee amputee groups used in this study were homogenous.

All the amputees were seen to experience a greater Fz1 peak on their intact limb compared to their prosthetic limb which was in agreement with previous studies (Suzuki, 1972; Engsberg et al., 1991; Engsberg et al., 1993) and this was observed at all speeds. The post-hoc test revealed that there was a difference between the below-knee amputee prosthetic and intact limbs, and between the below-knee amputee prosthetic limb and able-bodied subjects, whilst for the above-knee amputees a difference was found only between their prosthetic limb and able-bodied subjects. Thus hypothesis 1 was rejected and hypothesis 2 accepted. The below-knee amputees experienced greater loading on their intact limb compared to their prosthetic limb. Whilst this was also observed for the above-knee amputees, a significant difference was not found. This could have been due to the small number of subjects in the group. The soft tissues of the residual limb are not suitable for load-bearing (Silver-Thorn et al., 1996), and so higher forces through the residual limb may lead to pain and soft tissue breakdown as
experienced by highly active amputees during sport. One theory is that the amputees may be trying to protect their residual limb by loading it less and consequently loading their intact limb more. Another is that it may be due to the position of the centre of pressure (COP). Amputees' centre of gravity is closer to their intact limb than to their prosthetic limb whilst standing (Clark and Zernicke, 1981) and thus they are placing more body weight over their intact limb than their prosthetic limb during walking, leading to elevated loading. The position of the body segments may also be affecting the asymmetrical loading during amputee gait. With a non-fully functioning ankle or ankle and knee joint, the amputee may not be able to accelerate their prosthetic limb during weight-acceptance and push-off as much as their intact limb, thus exhibiting reduced Fz1 and Fz2 peaks on their prosthetic limb.

From the observations, with the exception of the maximum walking speed, where able-bodied subjects were walking much faster than the amputees, all amputees exhibited a greater intact limb peak Fz1 force than able-bodied subjects. Again, this was in agreement with previous studies (Suzuki, 1972; Engsberg et al., 1991; Engsberg et al., 1993). This finding indicates that the intact limb of unilateral amputees was subjected to greater stress compared to able-bodied subjects, regardless of comparable walking speed.

The Fz1 peak was observed to increase with increasing walking speed for all subjects. An increase in Fz1 on both limbs with walking speed is in agreement with previous studies of able-bodied subjects (Nilsson and Thorstensson, 1989). The Fz1 peak was increased by a greater amount on the intact limb compared with the prosthetic limb for all amputees as shown by the group by speed interaction. This is comparable with the findings of Zernicke et al. (1985) who reported greater increases on the intact
limb than on the prosthetic limb of above-knee amputee children when changing from a natural to a 'fast' walking cadence. Increases with speed for Fz1 from 1.15 BW to 1.23 BW were seen on the prosthetic limb and 1.34 BW to 1.68 BW on the intact limb for all amputees. If there is a limit to the acceleration of the prosthetic limb with walking speed, then this could account for the limited increase in the prosthetic limb Fz1 peak with walking speed compared to the intact limb. This could have implications for the design of the prosthetic limb in reducing ground reaction force asymmetry.

The Fz1 asymmetry increased with speed (from 17.5% to 29.3% asymmetry) for all the amputees. Natural walking speed is lower in lower limb amputees compared to able-bodied subjects (Lewallen et al., 1985; Hermodsson et al., 1994). Placing greater demands on the amputee such as increasing walking speed, has produced increases in asymmetry. To walk at faster speeds, it is necessary to accelerate the body over the limb they are stepping onto, and accelerate at push-off providing increasing vGRF peaks with increasing walking speed (from Newton's second law $F = m \cdot a$). It may be that in order for the amputees to keep their stability at the higher walking speeds, being unable to accelerate their body over their prosthetic limb during weight-acceptance due to a lack of fully functioning limb, they accelerate their intact limb more, or whole body whilst stepping onto their intact limb more. Thus, due to this compensation, they load their intact limb to an even greater extent than in natural cadence walking. Hypothesis 3 was thus rejected. Whilst allowing amputees to become stable at higher speeds, increases in intact limb loading could lead to intact limb joint pain or joint degeneration in later life. Radin et al. (1973) reported that those who are constantly excessively loading their joints may be provoking joint damage and possibly arthritis. Greater amounts of joint degeneration in the unilateral amputee's intact limb have been previously reported compared with both their residual limbs and the limbs of the able-bodied (Hungerford...
and Cockin, 1975; Burke et al., 1978) and joint degeneration is due to repeated cyclic loading (Radin et al., 1973). This has implications not only for highly active amputees who load their limb more than sedentary amputees, but also for younger amputees in later life.

For the temporal variables, all amputees were observed to have a longer stance time and shorter swing time on their intact limb compared to their prosthetic limb, but was only significantly different for above-knee amputees. The low numbers of subjects used in this study may be the reason that a significant difference was not found for the below-knee amputees. Thus, whilst hypothesis 4 was accepted, hypothesis 5 was rejected. The longer stance and shorter swing phase on the intact limb is in agreement with previous studies (Breakey, 1976; Murray et al., 1983) which indicated that less time was spent on the prosthetic limb either due to discomfort or pain from socket fit, or as if the amputees did not ‘trust’ the artificial limb. The shorter intact limb swing time with longer prosthetic limb swing time, possibly due to the inertial properties of the prosthesis (Torres-Moreno et al., 1997) has previously been seen as a mechanism to keep the gait cycle time similar for both limbs. All amputees were observed to spend longer stepping onto their prosthetic limb than on their intact limb, taking longer to transfer the weight onto the prosthetic limb. Again, significant differences were only found for the above-knee amputees, This step time asymmetry was again in agreement with previous studies (Breakey, 1976; Robinson et al., 1977).

All temporal values were observed to decrease with walking speed in agreement with previous studies (Andriacchi et al., 1977; Murray et al., 1983). There was a group by speed interaction for all temporal variables, showing that one limb was more affected by walking speed than the other for all amputees. For swing and step time duration, the
prosthetic limb appeared to be affected more by walking speed, whilst for stance time duration the intact limb appeared to be most affected by walking speed. Thus, to achieve the faster walking speeds, it appeared that the amputees altered their temporal gait patterns for both limbs.

The above-knee amputees were the most asymmetrical of the three groups for all temporal variables and differences were found between above- and below-knee amputees, and between above-knee amputees and able-bodied subjects. This was as expected due to the additional problems of being unable to flex the prosthetic knee properly, and loss of surrounding musculature. Temporal asymmetry was found to reduce, but not significantly, with walking speed (33.5% to 23.4% asymmetry for stance time, and 29.3% to 24.5% step time for above-knee amputees). Below-knee amputees were initially more symmetrical, but temporal asymmetry was also found to reduce slightly, but not significantly (5.5% to 3.5% stance time, and 5.2% to 2.0% step time asymmetry). For swing time asymmetry, there was a significant reduction of asymmetry with walking speed (above-knee amputees reduced from 64.6% to 37.4% swing time asymmetry, and below-knee amputees from 10.3% to 8.1% swing time asymmetry). There was also a significant interaction of group by speed, thus whilst swing time asymmetry decreased with walking speed for all three groups, the above-knee amputees appeared to be more affected by walking speed than below-knee amputees and able-bodied subjects. The reason no other significant effects of walking speed were found for temporal asymmetry may be due to the small number of subjects in this study, or that amputees consistently increase swing time symmetry in order to achieve faster walking speeds. Swing time asymmetry was greater than other temporal variables for both above- and below-knee amputees. Unless this is reduced significantly, the amputees may be unable to increase walking speed. These results are in disagreement with an
earlier study by Isakov et al. (1996) who reported that faster gait significantly affected all temporal parameters of gait, but not the level of symmetry between limbs in below-knee amputees. Isakov et al. (1996) only reported natural cadence walking and a faster speed; therefore, any changes in levels of symmetry may not have been noted with only a one-stage increase in walking speed. The study also only included below-knee amputees. In the present study, the greatest reduction in temporal asymmetry was found in above-knee amputees. Thus, hypothesis 6 was accepted.

From these results, amputees, particularly above-knee amputees, reduce temporal asymmetry, most notably swing time asymmetry, with increasing walking speed. This may be the mechanism in which they are able to achieve the faster walking speeds. Therefore, in order to walk at maximal speed, the amputee is observed to reduce stance, swing and step time asymmetry.

On analysing the vertical impulse data, differences were noted between the intact and prosthetic limbs of the amputees, and the prosthetic limbs and able-bodied subjects. Greater vertical impulse was observed on the intact limb of all amputees compared to their prosthetic limb and compared to able-bodied subjects. Vertical impulse was found to be affected by walking speed (reducing from 1.16 to 0.67 BW.s for the intact limb and 0.67 to 0.49 BW.s for the prosthetic limb) where there was a group by speed interaction. Thus, the intact limb of amputees appeared to be more affected by walking speed than the prosthetic limb for vertical impulse. Impulse asymmetry was greater for above-knee amputees than below-knee amputees and able-bodied subjects. There was no effect of walking speed on impulse asymmetry. As Fz1 asymmetry increased with speed and stance time asymmetry decreased with speed, impulse asymmetry stayed constant.
This study showed that when greater demands are placed on lower limb amputees such as increased walking speed, loading asymmetry increases and temporal asymmetry decreases. The data suggest that the amputees decrease their swing time asymmetry most, in order to make their stride times more symmetrical. This then allows them to achieve the faster walking speeds. If this co-ordination was not achieved, the amputees would be unable to increase walking speed as an increase in swing and stance time asymmetry would not allow an increase in stride frequency. If this was the only mechanism they used to achieve the faster walking speeds, they would become more symmetrical when walking faster. However, this co-ordination appears to be achieved at the expense of increased loading asymmetry to obtain stability at the higher walking speeds. This is seen in the results of the impulse asymmetry, the link between stance time and vertical loading. Whilst greater asymmetry occurs with increasing level of amputation, as previously noted (Zahedi et al., 1987), impulse asymmetry was not found to be affected by speed. Thus, lower limb amputees appear to achieve greater temporal symmetry, enabling them to achieve faster walking speeds, by increasing vertical loading asymmetry (by loading their intact limb more than their prosthetic limb).

4.3 Summary.

The decrease in temporal asymmetry with walking speed may be the means in which amputees achieve fast walking speeds. If swing time asymmetry was not reduced at higher speeds, it may not be possible for amputees to achieve these speeds. The increase in loading asymmetry with walking speed in unilateral amputees may be the method in which they achieve stability in order to walk at higher speeds, or may be a result of limitations of the prosthetic limb in that amputees cannot accelerate that limb or their body over that limb when stepping onto the prosthetic limb or pushing off.
Consequently, the body is accelerated more when stepping onto the intact limb by way of compensation resulting in a greater vGRF produced. Thus, unilateral amputees appear to be willing to accept higher loading levels on the intact limb to achieve greater control and stability walking at the faster speeds. It is not known whether this is a conscious or naturally occurring mechanism. As a result of increased loading asymmetry and decreased temporal asymmetry with speed, walking speed had no effect on impulse. Further research is needed to see how such high forces are absorbed by the intact limb, relating to possible indications of joint degeneration in the intact limb. Hypotheses 1, 3 and 5 were rejected and hypotheses 2 and 6 accepted.
5.0 Impact shock and shock attenuation asymmetry during walking for above- and below-knee amputees.
5.0 Shock and shock attenuation asymmetry during walking for above- and below-knee amputees.

This chapter is concerned with the measurement of impact shock and shock attenuation in unilateral lower limb amputees. Lower limb amputees have previously reported greater amounts of joint pain and joint degeneration in their intact limb compared to both their prosthetic limb and to able-bodied subjects. As the results of chapter four have shown that amputees load their intact limb consistently more than their prosthetic limb during walking, and that this asymmetry increases with walking speed, this increased asymmetry may partly explain the increased incidence of joint degeneration in the intact limb. However, it may not be the loading per se, but the impact shock and how this shock is attenuated that is responsible for the increased incidence of joint degeneration in the intact limb. The first study aims to establish whether the use of the accelerometer is a valid method of measuring peak acceleration at heel strike. The second study aims to investigate impact shock and shock attenuation asymmetry during walking for unilateral lower limb amputees.

5.1 Relationship between the heel-strike transient and impact accelerations in able-bodied gait.

5.1.1 Introduction.

It has previously been suggested that an accelerometer can be used to measure the peak axial acceleration at heel strike. While this can be measured by a force platform as the Fz load rate or heel strike transient, a measure of shock on the system, a force platform cannot measure impact shock from consecutive steps or the amount of shock attenuated as the shock travels through the body. Accelerometers are able to measure acceleration from consecutive foot contacts, and also impact shock attenuation through the body. Thus, if the accelerometers used are an accurate method of measuring peak
acceleration at heel strike, they can be used to provide more information measuring impact shock and shock attenuation during walking. The relationship between the heel strike transient and peak axial acceleration measured by accelerometers has been the subject of previous studies. The slope of the vertical ground reaction force (Fz) from the first instance of force to the heel strike peak, calculated as load rate, has been shown to correlate highly with peak tibial axial acceleration using bone mounted accelerometers (Hennig and Lafortune, 1991). Time to peak axial acceleration and time to peak heel strike was also highly correlated. Other aspects of acceleration have been shown to correlate only moderately or not at all. Magnitude of peak force and peak acceleration correlated moderately whilst peak axial acceleration was found not to correlate with the Fz1 force peak. The authors concluded that close relationships can be found between some of the vertical ground reaction force and acceleration parameters.

With the introduction of lightweight three-dimensional accelerometers, studies have shown that it is necessary to measure impact shock at heel strike in more than one direction. For studies of both walking and running, the greatest impact shock has been consistently measured in the AP direction. The axial direction exhibits a slightly lower acceleration whilst there is a relatively small acceleration in the ML direction (Lafortune and Hennig, 1988; Lafortune, 1991). Thus, there is a need to report impact accelerations in all three directions. The purpose of this study was to assess whether the use of the accelerometer was a suitable method of measuring the heel strike transient in the axial, ML and AP directions for able-bodied subjects regardless of whether they suffer from joint pain/ degeneration or not.
5.1.2 Methodology.

5.1.2.1 Apparatus.

Two triaxial accelerometers (Entran, model EGXT3-M-50 with a sensitivity of ± 50 g) were used to record impact shock in the axial, ML (medio-lateral) and AP (anterior-posterior) directions. A balsa wood shelf was first attached to the tibia. The balsa wood shelf was stuck to the skin of the subject’s leg with superglue (Loctite™), 5 cm above the malleolus, on the anterior-medial aspect of the tibia. Tape was then used to attach the balsa wood more firmly and reduce skin movement. The triaxial accelerometer was then placed on the balsa wood shelf, then visually aligned and glued to the balsa wood so that the three axes corresponded to the axial, anterior-posterior and medio-lateral directions (Figure 5.1). This was done with the subject standing, and whilst there may be some error associated with visually aligning the accelerometer, this study was performed to determine whether this type of accelerometer mounting gave valid results.

![Diagram showing triaxial accelerometer mounting](image_url)

Figure 5.1 - The triaxial accelerometer mounting for the right leg.
Both accelerometers were synchronised with data collected from a Kistler force platform (type 9281B11). The accelerometers and force platform were sampled together at 1000 Hz using Biopac Acknowledge software.

![Diagram](image)

Figure 5.2 – The accelerometer mounting and axes of the force platform at heel strike.

Whilst there is a difference in the alignment of the accelerometer and force platform axes, this study aims to investigate the impact shock and shock attenuation along the longitudinal axis of the shank and not the vertical acceleration, thus the placement of the accelerometer with its axes relative to the tibia are valid.

### 5.1.2.2 Subjects and procedure

Ten able-bodied subjects (all males) took part in the study. Each subject was initially questioned as to whether he had sustained a previous injury to their lower limbs or back, or whether they suffer or have suffered from intermittent lower limb or lower
back pain. Those that reported no pain or previous injury were then questioned as to their lower limb dominance. The subjects were then split into two groups. Five had no previous history of lower back/leg pain or injury (mean ± S.D.) age 25.0 ± 1.58 years, height 1.78 ± 0.05 m and mass 75.2 ± 9.57 kg, and five had previous lower back/leg pain or injury (mean ± S.D.) age 27.2 ± 2.17 years, height 1.77 ± 0.05 m and mass 75.0 ± 10.8 kg. All subjects wore the same footwear whilst walking (Umbro astroturf elite shoes).

A triaxial accelerometer was attached to the tibia in the manner described above. The same procedure was followed to attach a triaxial accelerometer to the contralateral leg. Each subject was allowed familiarisation with the accelerometers, and at each walking speed. The subjects walked over a force platform at four different speeds of 0.5, 0.9, 1.2 m.s\(^{-1}\) and their maximum walking speed. Any trial that was not within ± 3% of the pre-determined walking speed was disregarded and repeated. Force platform and accelerometer data were sampled together. Each subject walked over the force platform for 3 trials at each speed, then repeated the trials hitting the force platform with their contralateral leg. The synchronised peak tibial ML, AP and axial accelerations and ground reaction forces in the Fx, Fy and Fz directions were recorded for each limb.

5.1.2.3 Data analysis.

For the accelerometry data, a Fast Fourier Transformation (Figure 5.3) was used to determine the cut-off frequency of the filter used. As 99% of the signal was between 8 and 100 HZ, a Hanning band pass filter of 8-100Hz was used. Tibial acceleration peaks from the accelerometers were taken corresponding to the first instance of heel strike as measured by the force platform for the axial, AP and ML accelerations. The accelerometers had initially been calibrated using a specially written Labview (Scientific
Instruments) program. Here, the accelerometer was rotated through 180° (2 g) in each of the three planes it was to measure. The output value (measured by the program) was then used to multiply the data collected in that plane. All the data peaks were thus transformed to units of g (acceleration due to gravity, 9.81 m.s\(^{-2}\)).

![Graph](image)

Figure 5.3 – FFT analysis for the frequency content of the accelerometer signal during walking at 0.5 m.s\(^{-1}\).

For the force platform data, the heel strike transient was smoothed using a Hanning window with cut-off frequency of 62.5 Hz and calculated as the derivative of the Fz slope from a threshold value of 5 N to the heel strike peak (Lafortune and Hennig, 1988). The load rates of the Fy and Fx force components were calculated in the same way and for the same time period as the heel strike transient. Any trial where no heel strike was present was not included in this analysis. A two-way analysis of variance was performed to identify differences in peak acceleration or load rate between the pain and no-pain groups. Four groups (between-factor) (i) no-pain group dominant limb, (ii) no-pain group non dominant limb, (iii) pain group painful limb and (iv) pain group non-painful limb and four speeds (0.5, 0.9, 1.2 m.s\(^{-1}\) and maximum) were used in the analysis of variance model. Separate two-way analysis of variance calculations were performed for each of the 6 variables – axial acceleration, AP acceleration, ML
acceleration, Fz load rate, Fy load rate and Fx load rate, where mean values of the three trials were used for each individual. Significance was set at the 5% level. Correlations were performed on the mean data between the peak acceleration at the tibia and the comparative heel strike transient at each speed and in each of the three directions. Significance was set at the 5% level. As the accelerometers were placed at the ankle, the effects of angular motion and gravity during the stance phase were assumed to be negligible.
5.1.3 Results.

Figure 5.4 illustrates the peak tibial acceleration (g) and force derivative (N.s\(^{-1}\)) in each of the three directions.

![Graphs showing peak tibial acceleration (unsmoothed) and the corresponding force load rate (smoothed) for the a) vertical, b) AP and c) ML directions.](image)

The peak acceleration and corresponding load rate for able-bodied subjects with no limb pain are given in Table 5.1. These results show that for the no-pain group, there was little difference between the limbs for acceleration or load rate in either of the axial, ML or AP directions. This was seen to be true regardless of walking speed. Load rate and acceleration increased with increasing walking speed in each of the three directions.
Table 5.1 - Mean (± S.D.) of peak acceleration and corresponding load rate for able-bodied subjects (no-pain) walking with increasing speed. D is dominant limb, ND non-dominant limb.

<table>
<thead>
<tr>
<th>Walking Speed (m.s⁻¹)</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
<th>ND D</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>0.37</td>
<td>0.40</td>
<td>4212</td>
<td>3872</td>
<td>0.70</td>
<td>1.24</td>
<td>2513</td>
<td>2770</td>
<td>0.29</td>
<td>0.37</td>
<td>458</td>
</tr>
<tr>
<td></td>
<td>[0.33</td>
<td>[0.30</td>
<td>[2434]</td>
<td>[1777]</td>
<td>[0.51]</td>
<td>[0.72]</td>
<td>[1071]</td>
<td>[1476]</td>
<td>[0.10]</td>
<td>[0.13]</td>
<td>[175]</td>
</tr>
<tr>
<td>0.9</td>
<td>0.55</td>
<td>0.64</td>
<td>9136</td>
<td>8077</td>
<td>1.90</td>
<td>1.60</td>
<td>4184</td>
<td>4192</td>
<td>0.61</td>
<td>0.45</td>
<td>1209</td>
</tr>
<tr>
<td></td>
<td>[0.40]</td>
<td>[0.43]</td>
<td>[7923]</td>
<td>[5063]</td>
<td>[0.84]</td>
<td>[1.07]</td>
<td>[2801]</td>
<td>[3751]</td>
<td>[0.33]</td>
<td>[0.29]</td>
<td>[702]</td>
</tr>
<tr>
<td>1.2</td>
<td>0.75</td>
<td>1.01</td>
<td>11587</td>
<td>12058</td>
<td>1.76</td>
<td>2.60</td>
<td>5054</td>
<td>4740</td>
<td>0.88</td>
<td>0.44</td>
<td>1035</td>
</tr>
<tr>
<td></td>
<td>[0.34]</td>
<td>[0.55]</td>
<td>[4877]</td>
<td>[4317]</td>
<td>[0.96]</td>
<td>[1.13]</td>
<td>[1479]</td>
<td>[1670]</td>
<td>[0.63]</td>
<td>[0.36]</td>
<td>[504]</td>
</tr>
<tr>
<td>Max</td>
<td>1.07</td>
<td>1.70</td>
<td>21689</td>
<td>38011</td>
<td>2.83</td>
<td>3.39</td>
<td>16452</td>
<td>12973</td>
<td>1.26</td>
<td>0.96</td>
<td>1947</td>
</tr>
<tr>
<td></td>
<td>[0.43]</td>
<td>[0.52]</td>
<td>[17557]</td>
<td>[15032]</td>
<td>[1.61]</td>
<td>[0.62]</td>
<td>[4939]</td>
<td>[6131]</td>
<td>[0.25]</td>
<td>[0.43]</td>
<td>[1143]</td>
</tr>
</tbody>
</table>

For the subjects previously reporting joint pain, there was a slightly higher axial acceleration on the non-painful limb compared with the painful limb. Both load rate and acceleration appeared to increase with increasing walking speed.

Table 5.2 - Mean (± S.D.) of peak acceleration and corresponding load rate for able-bodied subjects (previously reported joint pain) walking with increasing speed. P indicates previously painful limb, NP non-painful limb.

<table>
<thead>
<tr>
<th>Walking Speed (m.s⁻¹)</th>
<th>P P</th>
<th>NP NP</th>
<th>P P</th>
<th>NP NP</th>
<th>P P</th>
<th>NP NP</th>
<th>P P</th>
<th>NP NP</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>0.27</td>
<td>0.32</td>
<td>4055</td>
<td>2714</td>
<td>0.62</td>
<td>0.73</td>
<td>2562</td>
<td>1462</td>
</tr>
<tr>
<td></td>
<td>[0.02]</td>
<td>[0.08]</td>
<td>[2901]</td>
<td>[1800]</td>
<td>[0.37]</td>
<td>[0.28]</td>
<td>[525]</td>
<td>[594]</td>
</tr>
<tr>
<td>0.9</td>
<td>0.35</td>
<td>0.52</td>
<td>5571</td>
<td>3498</td>
<td>1.47</td>
<td>0.73</td>
<td>5121</td>
<td>1762</td>
</tr>
<tr>
<td></td>
<td>[0.19]</td>
<td>[0.43]</td>
<td>[2679]</td>
<td>[2263]</td>
<td>[1.12]</td>
<td>[0.23]</td>
<td>[2892]</td>
<td>[647]</td>
</tr>
<tr>
<td>1.2</td>
<td>0.58</td>
<td>0.81</td>
<td>10571</td>
<td>6682</td>
<td>2.20</td>
<td>1.37</td>
<td>7151</td>
<td>3258</td>
</tr>
<tr>
<td></td>
<td>[0.20]</td>
<td>[0.27]</td>
<td>[1438]</td>
<td>[3699]</td>
<td>[0.15]</td>
<td>[0.68]</td>
<td>[2260]</td>
<td>[1094]</td>
</tr>
<tr>
<td>Max</td>
<td>1.40</td>
<td>1.66</td>
<td>37213</td>
<td>38124</td>
<td>2.88</td>
<td>2.89</td>
<td>15724</td>
<td>12741</td>
</tr>
<tr>
<td></td>
<td>[0.71]</td>
<td>[0.26]</td>
<td>[25394]</td>
<td>[13221]</td>
<td>[0.91]</td>
<td>[0.61]</td>
<td>[1644]</td>
<td>[6450]</td>
</tr>
</tbody>
</table>
The results of the two-way analysis of variance will now be presented. The exact p values for each significant test will also be presented. A p value of p = 0.000 does not represent p = 0, rather that p is less that 0.0005. As all other p values are given exactly, this convention of reporting p to three decimal places was undertaken for consistency. The results from the analysis of variance show no significant differences between the limbs for load rate in the axial, ML or AP directions for either the pain or no-pain group. There was also no significant difference between the limbs for acceleration in the ML or AP directions. In the axial direction, there was a difference between the limbs (F3,16 = 3.07, p=0.058), which failed to reach significance. However, the power of the test was low (0.605) and this could account for the non-significant result. As no significant differences were noted between limbs, all subjects were grouped in order to perform the correlations. A significant effect of speed was found for all variables in the ML direction, load rate F3,36 = 25.35, p=0.000, acceleration F3,36 = 25.35, p=0.000; AP direction, load rate F3,36 = 7.54, p=0.000, acceleration F3,48 = 59.11, p=0.000 and axial direction, load rate F3,36 = 38.89, p=0.000, acceleration F3,48 = 84.14, p=0.000. All variables were found to increase with increasing walking speed. No significant interaction was found between group and speed.

Table 5.3 shows correlations between the mean force rate of loading and mean accelerations measured at the tibia in the corresponding directions whilst walking at different speeds. The results show high correlations of peak tibial acceleration with ground reaction force load rate in each of the ML, AP and axial directions. There was a positive significant correlation (p<0.05) between mean peak acceleration and mean rate of loading for the X, Y and Z directions at all walking speeds.
Table 5.3 - Correlations (r values) between force load rate and peak acceleration in the ML, AP and axial directions for walking at different speeds.

<table>
<thead>
<tr>
<th>Walking speed (m.s(^{-1}))</th>
<th>ML</th>
<th>AP</th>
<th>Axial</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>0.69*</td>
<td>0.66*</td>
<td>0.57*</td>
</tr>
<tr>
<td>0.9</td>
<td>0.65*</td>
<td>0.81*</td>
<td>0.58*</td>
</tr>
<tr>
<td>1.2</td>
<td>0.61*</td>
<td>0.60*</td>
<td>0.60*</td>
</tr>
<tr>
<td>maximum</td>
<td>0.69*</td>
<td>0.57*</td>
<td>0.57*</td>
</tr>
</tbody>
</table>

* denotes significant correlation at 5% level.

Figure 5.5 - Linear regression and line of best-fit for peak tibial acceleration and load rate in the axial direction for walking at 1.2 m.s\(^{-1}\).

Figure 5.6 - Linear regression and line of best-fit for peak tibial acceleration and load rate in the AP direction for walking at 1.2 m.s\(^{-1}\).
Figure 5.7 - Linear regression and line of best-fit for peak tibial acceleration and load rate in the ML direction for walking at 1.2 m.s$^{-1}$. 

The results indicate that the load rate in the peak load acceleration correlated highly with peak tibial load rate. Herring and Loeb (1995) reported a higher correlation of $r = 0.81$ between these two variables using a bone-conduction accelerometer. In the present study, this relationship was not as strong as reported by Herring and Loeb (1995) primarily because of the differences in accelerometer calibrations. While a bone-conduction accelerometer with peak a given range of accelerations would have been more sensitive, however, the accelerometer notoriously is not sensitive to the bone used and lightweight accelerometer subject to the load rate was not transduced to the ML, AP, and axial walking directions. The second study, as well, implemented some of the measurements of the bone load rate. This study has shown that the ML and AP load rate correlate significantly with the corresponding load rate and the present study has measured and reported this.
5.1.4 Discussion.

This methodological study aimed to determine whether the use of accelerometers was a suitable method of measuring peak acceleration at heel strike. This was done by correlating the acceleration peaks in each of the ML, AP and axial directions with the load rate in the corresponding direction as measured by the force platform. The results showed significant correlations of peak acceleration with load rate for each of the ML, AP and axial directions. This relationship was true for all walking speeds. Whilst significant correlations between the accelerometer and force platform axes were found, differences will arise between the measured accelerations because of the axes orientations. This study aims to investigate the longitudinal impact shock experienced by the body and thus, as the axes of the accelerometer are more directly able to measure accelerations in the longitudinal (axial), ML and AP directions, relative to the tibia, the accelerometers may be a better method of measuring these accelerations.

The results are comparable with the literature in that peak axial acceleration correlated highly with vertical force load rate. Hennig and Lafortune (1991) reported a higher correlation \( r = 0.87 \) between these two variables using a bone mounted accelerometer. In the present study, this relationship, although significant, was not as strong as reported by Hennig and Lafortune (1991) possibly because of the differences in accelerometer mountings. Whilst a bone-mounted accelerometer will give a more accurate acceleration value, this technique is rarely possible. However, the accelerometer mounting in the present study, mounted on balsa wood and light-weight accelerometer angled so that each of the axes corresponded to the ML, AP and axial walking directions, has been shown to be an acceptable representation of the force load rate. This study has shown that the ML and AP directions also correlate significantly with the corresponding load rate and no other study has measured and reported this.
The relationship between mean peak acceleration and corresponding mean load rate was found to be true regardless of whether the subjects reported pain or no limb pain. Thus, as ground reaction force patterns and mechanisms of loading for lower limb amputees are not greatly different from able-bodied subjects, it is deemed that the relationship found here will also apply to lower limb amputees.

The mean peak acceleration values (Tables 5.1 and 5.2) show the largest peak to occur in the AP direction, followed by the axial direction and the smallest occurring in the ML direction. This was found regardless of walking speed. Due to the large magnitudes of impact shock in directions other than axial, there is a clear need to measure accelerations in three dimensions.

Previous studies have shown magnitudes of 1.71 g in the axial, 2.09 g in the AP and 0.9 g in the ML direction for a bone mounted accelerometer (Lafortune, 1991), or for a uniaxial skin mounted accelerometer, 0.7 to 0.9 g (Voloshin, 1988) walking shod at a natural cadence. Whilst small differences in peak acceleration occur between studies using different accelerometer mountings, the results of the present study are comparable to the magnitudes previously reported in the literature.

Thus, the placement and mounting of the accelerometers used in the present study have been shown to be good, in that they measure acceleration in the correct direction (high correlations with corresponding load rate) and the peak magnitudes are comparable with the literature. Also, due to placing the accelerometers close to the tibial axis of rotation i.e. the ankle joint, effects of gravity and angular motion were deemed to be negligible for walking.
5.1.5 Conclusion.

The magnitudes of the results found in this study are comparable with those previously reported in the literature. There was a significant relationship between the peak acceleration at heel strike as measured by the accelerometer, and the heel strike transient as measured by the force platform. This relationship was true for the axial, ML and AP directions and also for all subjects regardless of whether they suffered joint pain or not. Thus, it is deemed that the accelerometer gives a suitable measurement of the heel strike transient as measured by the force platform. Another advantage is that the accelerometer is also able to be placed so that it measures impact shock and shock attenuation along the longitudinal axis of the shank, rather than in the vertical direction. The accelerometer will therefore be used in the next study as it also has the advantage of providing additional information from a number of consecutive steps, and permitting a measurement of shock attenuation.
5.2 The effect of speed on impact shock and shock attenuation and impact shock and shock attenuation asymmetry.

5.2.1 Introduction.

It has been previously reported that unilateral lower limb amputees load their intact limb more than their prosthetic limb whilst walking (Engsberg et al., 1993) and also suffer joint pain and/or joint degeneration in their intact limb (Hungerford and Cockin, 1975; Burke et al., 1978). It has not yet been proved whether these two aspects of amputee gait are linked, but studies have suggested that cyclic loading can lead to joint damage and osteoarthritis (Radin et al., 1982; Voloshin, 1988). It may not be just the load magnitude acting on the intact limb that causes this problem, but the shock acting on the system and whether the body is able to absorb this shock as assessed by shock attenuation capacity.

Measurement of shock and shock attenuation using accelerometry has previously been used in studying patients with lower back or knee pain (Wosk and Voloshin, 1981; Voloshin and Wosk, 1982). In able-bodied patients diagnosed as preosteoarthritic, some impact shock asymmetry has been reported in that they impact their affected limb more than their healthy limb. For patients suffering from osteoarthritis or joint degeneration, there is an inability to attenuate fully shock from the shank to the head. As a consequence, incoming impact shock needs to be reduced on the affected limb. As no study found has previously looked at impact shock and shock attenuation asymmetry in lower limb amputees during walking, findings from studies of able-bodied subjects have led to the rise of a plausible theory. Those amputees not experiencing intact limb pain or joint degeneration may produce greater impact shocks on their intact limb compared to prosthetic limb, exhibiting impact shock asymmetry. This, along with the previously
established high loading on the intact limb, could be the mechanism leading to joint pain and/or joint degeneration in later life.

For unilateral below-knee amputees during running, Therrien and Prince (1998) reported slightly higher mean impact shock magnitudes on the intact compared to the prosthetic limb. However, there was a high variability between subjects and individual data were not reported. No other study found has looked at impact shock and shock attenuation in lower limb amputees.

As only one study found has looked at the incidence of impact shock and shock attenuation in lower limb amputees, no study has determined whether asymmetry is present. If progressive demands are made on the limbs such as increasing walking speed (which increases limb loading), changes in the magnitude of shock and shock attenuation may be seen in one or either limbs. Consequently, there is a need to assess how the prosthetic limb helps or hinders shock attenuation in the intact limb, and if this changes when increased demands are placed on the amputee such as when walking speed is increased. The purposes of this study were (i) to establish impact shock and shock attenuation magnitudes in both limbs of unilateral lower limb amputees, (ii) to determine whether asymmetry in impact shock and shock attenuation is present and (iii) to investigate the effect of increasing walking speed on these variables.
5.2.2 Methodology.

5.2.2.1 Apparatus.

Two triaxial accelerometers (as used in study 5.1) and a biaxial accelerometer (Entran, model EGXT3-M-50) with a sensitivity of ± 50 g were used in this study. Each accelerometer signal was connected to its own adjustable amplifier and then to a data acquisition board by a long, insulated cable. The data acquisition board was then connected to a Lap-top computer. The accelerometers were sampled together at 1000 Hz using custom written Labview (National Instruments) software.

5.2.2.2 Subjects and procedure.

The amputee subjects were all unilateral, established and highly active males. The below-knee amputees (n=5) had a mean (± SD) age of 46.8 ±16.5 years and time as an amputee as 21.4 ±14.2 years. The above-knee amputees (n=4) had a mean (± SD) age of 40.3 ± 12.0 years and time as an amputee 6.25 ± 4.11 years. For individual details see table 5.4. The able-bodied subjects (n=10), the same as those taking part in study 5.1 but not the same data reported, were also all males and highly active, 5 with no history of injury or limb pain, mean (± SD) age of 25.0 ± 1.58 years and 5 reporting lower limb pain, mean (± SD) age of 27.2 ± 2.17 years. All able-bodied subjects wore the same footwear (Umbro astroturf elite footwear) whilst all the amputees wore their own rubber-soled sports shoes (subject details Table 5.4).

A triaxial accelerometer was mounted on each shank of able-bodied subjects and the intact limb of unilateral amputees as described in study 5.1. For the prosthetic limb, the cosmesis was removed and a triaxial accelerometer via a balsa wood mounting was attached on the prosthetic shank at the same height as the first accelerometer was
attached to the intact limb (Plate 3). A bite-bar containing a biaxial accelerometer was held firmly in the subjects’ teeth (Plate 3).

Table 5.4 - Amputee subject details. R indicates right limb, L left limb, AK above-knee amputee, BK below-knee amputee.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Level</th>
<th>Age</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
<th>Prosthesis used</th>
<th>Years as amputee</th>
<th>Activity level</th>
<th>Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.W.</td>
<td>RAK</td>
<td>23</td>
<td>1.76</td>
<td>63.1</td>
<td>intelligent knee, flex foot</td>
<td>1</td>
<td>comp. basketball, swimming</td>
<td>none</td>
</tr>
<tr>
<td>A.B.</td>
<td>RAK</td>
<td>50</td>
<td>1.75</td>
<td>82.6</td>
<td>intelligent knee, flex foot</td>
<td>10</td>
<td>rec. golf, swimming</td>
<td>low back</td>
</tr>
<tr>
<td>J.N.</td>
<td>RAK</td>
<td>42</td>
<td>1.80</td>
<td>83.0</td>
<td>intelligent knee, seattle foot</td>
<td>9</td>
<td>none</td>
<td>low back</td>
</tr>
<tr>
<td>B.M.</td>
<td>LAK</td>
<td>46</td>
<td>1.77</td>
<td>76.2</td>
<td>intelligent knee, seattle foot</td>
<td>5</td>
<td>walking</td>
<td>low back</td>
</tr>
<tr>
<td>D.C.</td>
<td>RBK</td>
<td>60</td>
<td>1.73</td>
<td>79.2</td>
<td>flex foot</td>
<td>39</td>
<td>rec. tennis, cricket</td>
<td>low back</td>
</tr>
<tr>
<td>N.L.</td>
<td>LBK</td>
<td>37</td>
<td>1.84</td>
<td>80.5</td>
<td>flex foot</td>
<td>32</td>
<td>rec. cycling, swimming</td>
<td>low back</td>
</tr>
<tr>
<td>R.B.</td>
<td>LBK</td>
<td>62</td>
<td>1.73</td>
<td>88.9</td>
<td>flex foot</td>
<td>11</td>
<td>comp. golf, football</td>
<td>intact knee, ankle</td>
</tr>
<tr>
<td>J.K.</td>
<td>RBK</td>
<td>23</td>
<td>1.73</td>
<td>72.5</td>
<td>multi axis foot</td>
<td>20</td>
<td>none</td>
<td>intact knee</td>
</tr>
<tr>
<td>M.K.</td>
<td>RBK</td>
<td>52</td>
<td>1.73</td>
<td>77.0</td>
<td>flex foot</td>
<td>5</td>
<td>none</td>
<td>intact knee</td>
</tr>
</tbody>
</table>

All subjects were allowed familiarisation with the walking speeds prior to data collection. Each subject walked along a walkway at four different speeds of 0.5, 0.9, 1.2 m.s\(^{-1}\) and maximum (which ranged from 1.5 to 1.9 m.s\(^{-1}\)) walking speed which were measured by timing lights. Any trial that was not within ±3% of the pre-determined speed was disregarded and the trial repeated. Three trials were completed at each speed.
Plate 3 - Accelerometer mountings on the amputees’ prosthetic and intact limb, and the bite-bar.

5.2.2.3 Data analysis.

The data collected were analysed using a specially written Labview (Scientific Instruments) program. From fast fourier transfer (FFT) analysis, the data were filtered in with a Hanning band pass filter of 8-100 Hz (see Chapter 5.1) for both the intact limb and prosthetic limb, and 8-60 Hz for the bite bar (Figure 5.8). The first three and last three strides were disregarded and data were collected for 5-8 strides in each trial. Peak tibial ML, AP and axial accelerations from both limbs of all subjects and peak axial bite bar acceleration were determined for all subjects using a specially written Labview (Scientific Instruments) program. Shock attenuation for each variable was calculated as the percentage difference between peak axial acceleration at heel strike and the corresponding peak bite bar acceleration, taking into consideration the short time lag due to the shock being attenuated through the body at the knee, hip, spine and other ligaments and soft tissues. Asymmetry was calculated for impact shock peaks and
shock attenuation between the right and left limbs of able-bodied subjects and between the intact and prosthetic limbs of the amputees using the absolute symmetry index (Herzog et al., 1989).

![FFT Analysis](image)

Figure 5.8 – The FFT analysis for the bite bar accelerometer signal during walking at 0.5 m.s\(^{-1}\).

The effect of speed on the chosen variables for able-bodied subjects was examined using a two-way analysis of variance in the same way as was reported in chapter 5.1. Level of significance was set at 5%. For the amputees, due to the uncontrollable variation in stump length and prosthesis components, each subject was treated as a case study. For each amputee, it was reported whether the variables measured fell inside or outside the able-bodied subject (no-pain group) range of ± 1 SD of the mean of the left and right limbs.
5.2.3 Results.

5.2.3.1 Able-bodied subjects.

Impact shock.

Figure 5.9 shows an example of raw data for the peak axial, ML, AP and bite-bar accelerations.

Figure 5.9 – Peak axial, ML, AP and bite bar data during walking at 1.2 m.s\(^{-1}\) for an able-bodied subject.

The results for the maximum walking speed which ranged from 1.5 m.s\(^{-1}\) in the amputees to 1.9 m.s\(^{-1}\) in the able-bodied subjects were compared directly, even though the speeds differed, because the effect of their maximum possible walking speed on the chosen variables for each individual needed to be seen. Whilst it is appreciated that these speeds differed from individual to individual, the aim was to investigate the effect of their absolute maximum walking speed as an indication of the maximum demands placed on that individual during walking. Thus, all maximum values will be compared across individuals, marked separately on the graphs by a discontinued line ///.
The results in Figure 5.10 show that for the axial peak accelerations, there was greater impact shock on the dominant limb (no-pain group) compared to the non-dominant limb, and on the pain-free limb (pain group) compared to the previously reported painful limb. However, no significant differences between limbs was found for ML or AP peak accelerations. For the axial peak acceleration, the difference between the limbs approached significance, $F_{3,16} = 3.07, p = 0.058$, but the power of the test was low (0.605) and this may account for the non-significant finding.

For the able-bodied subjects (both pain and no pain groups), peak ML ($F_{3,36} = 25.35, p=0.000$), AP ($F_{3,48} = 59.11, p=0.000$), and axial tibial accelerations ($F_{3,48} = 84.14, p=0.000$), increased with walking speed (Figure 5.10). The results were similar between the two groups, but subjects in the pain group were observed to produce greater pain-free limb impact shocks at the faster walking speeds. There was no significant interaction between group and speed.

Results for the bite bar (Table 5.7) show that in the axial direction, impact shocks were in the order of magnitude of 0.1 to 0.3 g, regardless of walking speed. There was no difference found between groups for the bite bar data. Also, there was no effect of walking speed on the bite bar data and no significant group by speed interaction.
Figure 5.10 - Impact shock in the ML, AP and axial directions at heel strike for able-bodied subjects walking with no pain and previously reported pain. The black line indicates dominant or pain free limb, red non-dominant or previously reported painful limb.
Shock attenuation.

For shock attenuation (Figure 5.11), both groups attenuated a similar amount of shock on both limbs. The percentage of shock attenuated was found to increase sequentially with walking speed $F_{3,48} = 34.54$, $p=0.000$, for both the pain and no pain groups. In all cases, the dominant limb attenuated slightly more shock than the non-dominant limb, but not significantly $F_{3,16} = 1.72$, $p=0.202$.

Figure 5.11 - Shock attenuation for able-bodied subjects walking with no pain and previously reported pain. Black indicates dominant or pain free limb, red non-dominant or previously reported painful limb.

Impact shock asymmetry.

Figure 5.12 shows impact shock asymmetry in the axial, ML and AP directions for the pain and no-pain groups. Subjects in the pain group were seen to be more asymmetrical than those in the non-pain group in each of the three acceleration directions measured (Figure 5.12). However, they were only slightly more asymmetrical than the non-pain group in terms of shock attenuation between the dominant and non-dominant limbs (less than 10% asymmetry for the non-pain group and less than 15%
asymmetry for the pain group). Axial (Z) asymmetry was found to increase with speed for both groups.

![Graphs showing asymmetry for pain and no pain groups](image)

Figure 5.12 - Impact shock asymmetry in a) the axial direction, b) the AP direction and c) the ML direction for able-bodied subjects. Black indicates pain free subjects, red subjects previously reported painful limb.

**Shock attenuation asymmetry.**

Shock attenuation asymmetry decreased slightly with speed for the no pain group, and generally decreased with speed for the pain group (Figure 5.13), but was not significant. No other effect of speed on asymmetry was noted.

![Graph showing shock attenuation asymmetry](image)

Figure 5.13 - Shock attenuation asymmetry for able-bodied subjects. Black indicates pain free subjects, red subjects previously reported painful limb.
Table 5.5 - Mean (± SD) of shock and shock attenuation asymmetry for able-bodied subjects without back or leg pain or injury.

<table>
<thead>
<tr>
<th>Speed (m.s⁻¹)</th>
<th>ML</th>
<th>% ASI</th>
<th>Axial</th>
<th>Shock attenuation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.5</td>
<td>44.7</td>
<td>21.0</td>
<td>11.0</td>
<td>9.1</td>
</tr>
<tr>
<td></td>
<td>[21.2]</td>
<td>[21.4]</td>
<td>[14.2]</td>
<td>[3.1]</td>
</tr>
<tr>
<td>0.9</td>
<td>50.2</td>
<td>36.3</td>
<td>17.9</td>
<td>6.7</td>
</tr>
<tr>
<td></td>
<td>[35.9]</td>
<td>[27.5]</td>
<td>[7.5]</td>
<td>[4.5]</td>
</tr>
<tr>
<td>1.2</td>
<td>31.4</td>
<td>37.7</td>
<td>20.0</td>
<td>4.9</td>
</tr>
<tr>
<td></td>
<td>[26.7]</td>
<td>[17.9]</td>
<td>[11.4]</td>
<td>[4.1]</td>
</tr>
<tr>
<td>max</td>
<td>32.6</td>
<td>29.0</td>
<td>20.5</td>
<td>3.6</td>
</tr>
<tr>
<td></td>
<td>[21.1]</td>
<td>[16.6]</td>
<td>[12.4]</td>
<td>[2.3]</td>
</tr>
</tbody>
</table>

Table 5.6 - Mean (± SD) of shock and shock attenuation asymmetry for able-bodied subjects with back or leg pain.

<table>
<thead>
<tr>
<th>Speed (m.s⁻¹)</th>
<th>ML</th>
<th>% ASI</th>
<th>Axial</th>
<th>Shock attenuation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.5</td>
<td>68.1</td>
<td>17.9</td>
<td>37.7</td>
<td>14.5</td>
</tr>
<tr>
<td></td>
<td>[28.4]</td>
<td>[11.4]</td>
<td>[29.3]</td>
<td>[13.3]</td>
</tr>
<tr>
<td>0.9</td>
<td>59.5</td>
<td>43.2</td>
<td>29.6</td>
<td>9.0</td>
</tr>
<tr>
<td></td>
<td>[52.6]</td>
<td>[27.6]</td>
<td>[23.7]</td>
<td>[8.3]</td>
</tr>
<tr>
<td>1.2</td>
<td>66.6</td>
<td>33.7</td>
<td>36.6</td>
<td>13.9</td>
</tr>
<tr>
<td></td>
<td>[45.4]</td>
<td>[38.7]</td>
<td>[24.6]</td>
<td>[10.4]</td>
</tr>
<tr>
<td>max</td>
<td>50.7</td>
<td>20.2</td>
<td>44.4</td>
<td>10.1</td>
</tr>
<tr>
<td></td>
<td>[45.2]</td>
<td>[17.7]</td>
<td>[18.0]</td>
<td>[6.7]</td>
</tr>
</tbody>
</table>

Table 5.7 - Mean (± SD) of peak axial impact shock, shock attenuated and peak bite bar impact shock for able-bodied subjects. Mean of left and right limbs for both 'with no back or leg pain' and 'back or leg pain'.

<table>
<thead>
<tr>
<th>Speed (m.s⁻¹)</th>
<th>Axial (g)</th>
<th>% Shock attenuated</th>
<th>Bite-bar (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.5</td>
<td>0.26</td>
<td>63.0</td>
<td>0.10</td>
</tr>
<tr>
<td></td>
<td>[0.12]</td>
<td>[22.3]</td>
<td>[0.01]</td>
</tr>
<tr>
<td>0.9</td>
<td>0.41</td>
<td>67.8</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>[0.19]</td>
<td>[19.7]</td>
<td>[0.02]</td>
</tr>
<tr>
<td>1.2</td>
<td>0.55</td>
<td>73.4</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>[0.20]</td>
<td>[24.6]</td>
<td>[0.02]</td>
</tr>
<tr>
<td>max</td>
<td>1.37</td>
<td>78.5</td>
<td>0.29</td>
</tr>
<tr>
<td></td>
<td>[0.4]</td>
<td>[29.3]</td>
<td>[0.03]</td>
</tr>
</tbody>
</table>
5.2.3.2 Amputees.

Impact shock.

Figure 5.15 shows an example of the axial, ML, AP and bite bar data for a below knee amputee walking.

For all amputees, the axial impact shock at heel-strike was observed to increase with increasing walking speed for both the prosthetic and intact limbs (Figure 5.15). For all below-knee amputees and three above-knee amputees, the axial shock at heel-strike was higher on the prosthetic limb than on the intact limb, although for one above-knee amputee the opposite was found. The results for impact shock in the ML and AP directions show they both increased with increasing walking speed from 0.2 g to 0.9 g for the ML direction and 0.6 g to 5.3 g for the AP direction for all amputee subjects. Generally, the ML and AP directional impact shocks were greater on the intact limb than the prosthetic limb, differing from the results found for the axial impact shock.
For the below-knee amputees (who all reported joint pain in the intact limb), impact shock for subjects MK, JK, NL and DC’s prosthetic limb all fell outside the able-bodied (no-pain) mean ± 1 SD (values reported in appendix). These amputees were all found to have higher prosthetic limb impact shocks than the able-bodied subjects (no-pain mean) at all but the maximum walking speed. Impact shock on the intact limb of subjects MK, RB and NL were all lower than those reported for the able-bodied (no-pain) group mean, but fell within the mean ± 1 SD range.

For the above-knee amputees, with the exception of subject CW (the only one not reporting pain in the intact limb), impact shocks on the prosthetic limb fell outside the able-bodied (no-pain) mean ± 1 SD. These prosthetic limb impact shocks were greater than those reported for able-bodied (no-pain) subjects at all but the maximum walking speed. Impact shock on the intact limb was less than for the prosthetic limb, but fell inside the reported able-bodied subject range at all except the maximum walking speed.
BELOW-KNEE AMPUTEES

ABOVE-KNEE AMPUTEES

Figure 5.15 - Axial impact shock for the prosthetic (dashed line) and intact (solid line) limbs of individual below-knee and above-knee amputees. Thick black line, able-bodied subjects for comparison.
At the maximum walking speed, impact shock on the intact limb was much lower than for the prosthetic limb and for able-bodied (no-pain) subjects. For subjects CW, greater impact shock was seen on his intact limb compared to the prosthetic limb. Impact shock on his intact limb was also greater than reported for the able-bodied (no-pain) mean, but fell inside the mean ± 1 SD range for all but the 1.2 m.s⁻¹ walking speed.

Shock attenuation.

Figure 5.16 shows shock attenuation with increasing walking speed for both the intact and prosthetic limbs of individual amputees, and the mean of the left and right limbs of able-bodied subjects (no-pain) for comparison. All raw and bite bar data for the amputees is reported in appendix 4. For all amputee subjects, regardless of whether axial impact shock was greater on their prosthetic or intact limbs (Figure 5.15), more shock was attenuated through their prosthetic limb than their intact limb (Figure 5.16). For some subjects (D.C., N.L., M.K. and A.B.), percentage shock attenuated in the prosthetic limb was almost constant with walking speed, illustrating that for those particular prostheses, the maximum amount of shock is attenuated regardless of walking speed. For those subjects, the greatest discrepancy was seen in the amount of shock attenuation between prosthetic and intact limbs at the lower walking speeds. At maximum walking speed (with the exception of M.K.), both limbs were found to attenuate similar amounts of shock. For the other subjects, similar amounts of shock were attenuated at maximum walking speed.

For the below-knee amputees, greater amounts of shock were attenuated through the prosthetic limb compared to the intact limb. The intact limb also attenuated less shock than able-bodied subjects (no-pain mean). However, with the exception of walking at 0.5 m.s⁻¹, the amount of shock attenuated by subjects RB, DC, JK and NL
Figure 5.16 – Axial shock attenuation for the prosthetic limb (dashed line) and intact limb (solid line) of individual below- and above-knee amputees. Thick black line, able-bodied subjects.
all fell within the able-bodied (no-pain) subject range of mean ± 1 SD for both the prosthetic and intact limb.

For the above-knee amputees, the same was found to be true. At all walking speeds, the amount of shock attenuated by the prosthetic or intact limb fell outside the able-bodied (no-pain) range of mean ± 1 SD.

Impact shock asymmetry.

Figure 5.17 shows impact shock asymmetry in each of the axial, ML and AP directions for individual amputees. For impact shock asymmetry in the ML direction of (Figure 5.17), there appeared to be no effect of speed for the amputees. For the above-knee amputees, impact shock asymmetry in the AP and axial directions generally increased with speed from 0.9 m.s⁻¹ to maximum walking speed. For the below-knee amputees, axial impact shock asymmetry increased with speed for three subjects, and reduced with speed for two subjects. A general trend could not be found for all amputees.

For the amputees, a general trend could not be found for all subjects as to whether they fell inside or outside the able-bodied (no-pain) range of mean ± 1 SD for impact shock asymmetry in the ML, AP or axial directions. For the below-knee amputees, subjects MK and NL were found to have a much higher axial impact shock asymmetry than able-bodied subjects, falling outside the mean ± 1 SD range at all walking speeds. With the exception of the slower walking speeds, subjects RB and DC fell within the mean ± 1 SD range of able-bodied subjects for axial impact shock asymmetry. For above-knee amputees, most subjects fell outside this range for axial impact shock asymmetry at all walking speeds.
X (medio-lateral acceleration).

Y (anterior-posterior acceleration).

Z (axial acceleration).

BELOW-KNEE AMPUTEES

ABOVE-KNEE AMPUTEES

Figure 5.17 - Impact shock asymmetry of individual above- and below-knee amputees with increasing walking speed.
Shock attenuation asymmetry.

For shock attenuation, asymmetry was greatest at the lowest walking speed (possibly due to lack of balance or not used to walking at such a slow speed). Generally, shock attenuation asymmetry decreased with increasing walking speed (with the exception of subject B.M.). These results were found to follow the same trends as the able-bodied group (although higher in asymmetry magnitude). Shock attenuation asymmetry was generally found to be greater for the below-knee amputees than for the above-knee amputees.

For the below-knee amputees, all subjects with the exception of subject RB (who reported low shock attenuation asymmetry at the faster walking speeds) exhibited shock attenuation asymmetry which fell outside the able-bodied subject (no-pain) range of mean ± 1 SD. The below-knee amputees reported higher shock attenuation asymmetry than the able-bodied (no-pain) subjects at all walking speeds. For the above-knee amputees, with the exception of subject JN who also reported low shock attenuation asymmetry at the faster walking speeds, the same was true (Figure 5.18).

Figure 5.18 - Shock attenuation asymmetry of individual above- and below-knee amputees.
5.2.4 Discussion.

5.2.4.1 Able-bodied subjects.

Impact shock and shock attenuation.

As few studies have reported accelerations in three dimensions during walking, it is difficult to compare the results of this study to the literature in the ML and AP directions. The results of this study show the axial accelerations at impact for walking at 1.2 m.s\(^{-1}\) to be in agreement with previous findings for walking at a natural cadence (Voloshin, 1988; Lafortune, 1991).

For all the able-bodied subjects, there was no difference between limbs for impact shock in the AP and ML directions. For the no-pain group, there was a small difference in impact shock between the dominant and non-dominant limbs in the axial direction with a greater impact peak on the dominant limb. For the pain group, there was a small difference between the limbs with a greater impact peak on the non-painful limb, but this was not significant. This may have been due to the small power of the test, or that the subjects reporting joint pain were not ‘extreme’ enough. This observation is in contrast with the results of studies by Voloshin and Wosk (1982) who found that patients with joint or back pain had a significantly reduced peak impact shock at the femoral condyle. They also stated that poor shock attenuation at the knee (from degenerative joint disease) probably leads to a change in walking pattern in order to prevent the over-loading of other shock absorbers above the knee by trying to reduce the absolute value of the incoming shocks (Wosk and Voloshin, 1981). These studies concluded that every patient who suffers from degenerative joint disease has an insufficient shock absorption capacity of the knee system. Even under normal physiological conditions, the continuous repetitive onslaught of the incoming waves...
generated at heel strike tends to cause a progressive weakening of the natural shock absorbers and may lead to joint degeneration and osteoarthritis (Radin et al., 1980).

With regard to shock attenuation, the results showed that for the no-pain group, there was little (non-significant) difference (less that 9%) between the two limbs. For the pain group, less shock was attenuated through the painful limb compared to the non-painful limb. This was in agreement with previous studies (Wosk and Voloshin, 1981; Voloshin and Wosk, 1982; Radin et al., 1991) reporting reduced shock attenuation in the affected limb of preosteoarthritic and osteoarthritic patients. Due to the insufficient shock absorption capacity of the knee or knee-forehead system seen in patients reporting lower limb joint pain, incoming impact shocks need to be reduced in order to prevent high impact shocks reaching the head and disturbing the vestibular system (Pozzo et al., 1991). Thus, the findings of this study that able-bodied subjects reporting knee joint pain attenuate less shock through that limb and as a result reduce incoming impact shocks to prevent over-loading the remaining shock absorbers, are in agreement with previous studies. However, no significant differences were found between the able-bodied groups previously reporting joint pain and those pain free. These subjects were not assessed for preosteoarthritis or osteoarthritis, and the numbers of subjects in these two groups were low.

Impact shock in all three directions was found to increase with increasing walking speed. This was as expected as increases in impact shock have been shown to result from increases in running speed (Hamill et al., 1983). It has been suggested that higher magnitude or more frequent shocks (such as walking at maximum speed) place greater stress on skeletal structures, increasing the risk of injury or degenerative joint disease (Hamill et al., 1995). Therefore, by increasing walking speed and thus impact
shock in all three directions, greater stress is being placed on the musculo-skeletal structure as the body now has to either attenuate more shock, or alter gait kinematics in order to try and reduce the incoming impact shock. This may lead to increasing asymmetry in other gait variables.

**Impact shock and shock attenuation asymmetry.**

The greatest impact shock asymmetry between the limbs of both able-bodied groups was in the ML direction (up to 50% in the no-pain group and up to 68% in the pain group). For the axial direction, the pain group exhibited greater asymmetry (up to 44%) compared to the non-pain group (up to 20%). This asymmetry was found to increase slightly with walking speed. The greater asymmetry in the pain group was expected due to problems with a reduced shock attenuation capacity and therefore the need to reduce incoming impact shocks. Thus if one limb is affected by joint pain/osteoarthritis, more asymmetry will be seen than is exhibited between two healthy limbs. The increase in axial impact shock asymmetry with increasing walking speed can be explained by the greater stress being placed on the musculo-skeletal system at the faster walking speeds. As impact shock increased in all three directions with walking speed, the body would have to attenuate more impact shock through the limb. As this has been shown to be difficult with a painful/osteoarthritic limb joint, incoming impact shocks need to be reduced, possibly by using altered gait kinematics, in order to prevent high impact shocks from reaching the head. Thus impact shock asymmetry would be expected to be increased at higher walking speeds. Further research is needed in order to establish whether individuals with joint degeneration reduce incoming impact shocks by altering their gait kinematics.
5.2.4.2 Amputees.

Impact shock and shock attenuation.

In this study, it was possible to include only one amputee not reporting any intact limb pain, such is the problem of intact limb or low-back pain in unilateral lower limb amputees. The results of this subject (C.W.) will be included in the discussion, but findings from this subject cannot be applied to other amputees reporting no intact limb pain.

With regard to impact shock, with the exception of one amputee (subject C.W.), there was a trend for impact shock to be greater on the prosthetic limb than intact limb at all walking speeds (Figure 5.8). This appears to be contrary to the previous findings of the vGRF, greater on the intact limb than prosthetic limb. However, it could be explained in terms of limb segment or whole body motion. It may be that the amputees have limited residual muscle control over how the prosthetic limb hits the ground, thus producing a higher axial impact shock on that limb compared to their intact limb where they have full muscular control. However, once the prosthetic limb has contacted the ground, because of the limited function of the artificial knee and ankle joint, the amputee is unable to accelerate the body over the prosthetic limb, resulting in a reduced vGRF. Subject C.W. was the only amputee not to suffer from intact limb pain, and consistently produced greater impact shock on his intact limb than prosthetic limb.

With regard to shock attenuation, the eight subjects (5 below-knee and 3 above-knee amputees) who all reported intact limb and/ or low-back pain exhibited a trend for reduced shock attenuation through their intact limb compared to their prosthetic limb, and to the healthy limbs of able-bodied subjects reporting no pain. These findings were
in agreement with the findings of the able-bodied subjects reporting joint pain, and the
findings of Wosk and Voloshin (1981) and Voloshin and Wosk (1982).

**Impact shock and shock attenuation asymmetry.**

For impact shock asymmetry, a general trend could not be found for all
amputees. In the axial direction, impact shock tended to increase with walking speed for
all above-knee amputees and three below-knee amputees, but decreased with walking
speed for two below-knee amputees. Differences seen at the slowest walking speed of
0.5 m.s\(^{-1}\) may have been due to the amputees being slightly unstable at such a slow
walking speed and altering their gait pattern. While it was expected that impact shock
asymmetry increased with increasing walking speed, this was not seen for two of the
nine amputees. These two amputees were more symmetrical than the other subjects and
became increasingly symmetrical at the higher walking speeds. It may be speculated that
this could have been due to the type of prosthesis worn.

Shock attenuation asymmetry decreased with increasing walking speed with the
exception of one subject. Some of the amputees indicated very little change in
percentage shock attenuation with walking speed for the prosthetic limb. Thus, for these
subjects, it is suggested that the amount of shock attenuated was mainly dependent on
the properties of the prosthesis worn. As there was no change in shock attenuation of the
prosthetic limb, and shock attenuation of the intact limb increased with walking speed,
overall shock attenuation asymmetry decreased with speed. This finding has
implications for the design and materials used in the prosthesis such as the fit of the
socket and ‘gel’ sock worn by the amputees, and the lack of natural shock absorbers like
natural knee flexion or ankle pronation. As incoming impact shock increases with
walking speed, more shock needs to be attenuated before it reaches the head. If the
increased impact shock is not attenuated sufficiently through the prosthetic limb, the prosthesis is not doing a good enough job at the faster walking speeds of attenuating the increased impact shocks before they reach the head. If this occurs, it may be that the amputees try and compensate for this by altering their gait mechanics, in order to reduce asymmetry at the higher walking speeds in terms of joint kinematics or joint kinetics.

For the above-knee amputee reporting no intact limb joint pain (C.W.), greater impact shock was seen for his intact limb compared to his prosthetic limb. The finding for this subject is in agreement with an earlier study (Radin et al., 1991) who reported that able-bodied preosteoarthritic patients produced a greater impact shock on their affected limb compared to their non-affected limb, possibly contributing to increased joint damage and osteoarthritis in that limb. It may be that new amputees who do not report intact limb pain/joint degeneration, impact their intact limb more in an attempt to protect their residual limb. This increase in impact shock may lead to cumulative damage to cartilage and joints. Once degenerative joint disease is present, less shock can be attenuated. As with the able-bodied subjects reporting joint pain, reduced capacity for shock attenuation leads to the need to reduce incoming impact shock in order to protect the head from excessive movement. As only one subject in the study did not report intact limb pain, there is not enough evidence to support this theory, and more research in this area needs to be done. However, for unilateral amputees experiencing intact limb pain, less shock was attenuated through their intact limb and incoming impact shocks were reduced compared to their prosthetic limb. Here, it may be the cumulative damage of not only producing greater impact shocks on the intact limb at heel strike, but also increasing the acceleration of the intact limb or whole body during the weight-acceptance phase of stance (Chapter 4) that increases the risk of developing degenerative joint disease for unilateral lower limb amputees. If this is the case, it may be...
be an advantage to include shock absorbing materials as an insole in the sole of the shoe on the intact limb. More research is needed in order to investigate how the amputee is further compensating for not only the functional loss of one or more lower limb joints, but also joint pain/osteoarthritis present in the remaining limb.

5.3 Summary.

It was found in study 5.1 that accelerometers were a suitable measurement of the heel strike transient and that they could be used to provide information about impact shocks over a number of consecutive steps. No differences were found between the limbs of the pain and no-pain groups, possibly because of the low power of the test or the fact that subjects were not 'extreme' enough in their pain and were assessed for joint pain only and not the presence of osteoarthritis.

In study 5.2, all except one amputee reported pain in the intact limb. The results agreed with previous studies of able-bodied subjects with degenerative joint disease in that less shock was attenuated through the affected (intact) limb, and as a consequence, incoming impact shocks on that limb were reduced.

Impact shock asymmetry in the axial direction increased with walking speed for most of the amputees, but not all. Shock attenuation asymmetry decreased with walking speed as a result of shock attenuation in the prosthetic limb staying relatively constant for some amputees, and increasing in the intact limb at the faster walking speeds. The increased stress placed on the intact limb at the faster walking speeds may have implications for increased joint degeneration. It may be that the amputees compensate for this and the lack of shock attenuation through the prosthetic limb by altering their gait kinematics. Further research is needed to see whether kinematic and kinetic
asymmetry is increased at the higher walking speeds in order to compensate for the problems of increased stress at the higher walking speeds.
6.0 Kinematic and kinetic asymmetry during walking for above- and below-knee amputees.
6.0 Kinematic and kinetic asymmetry during walking for above- and below-knee amputees.

This chapter is concerned with the effect of increased walking speed on kinematic and kinetic (joint moments and power output) variables and asymmetry in unilateral lower limb amputee gait. Chapter 4 has established that unilateral lower limb amputees load their intact limb more than their prosthetic limb, and chapter 5 that less impact shock is attenuated through the intact limb in those amputees reporting intact limb joint pain. Due to the increased intact limb loading and reduced shock attenuation, it may be that these lower limb amputees alter their gait kinematics and kinetics in order to try and protect the intact limb from further damage. By increasing walking speed, amputees may display changes in joint angles, joint moments and power output and their asymmetries as a way of coping with the demands at the faster walking speeds. The first study aims to establish the variation that could be due to errors in kinematic data measurement and kinetic (joint moment and power output) calculations. The second study aims to investigate the effect of increased walking speed on kinematic and kinetic gait variables and their asymmetry in above- and below-knee amputees using the previously established methodology.

6.1 Variability in kinematic measurements and joint moment and power output calculations in able-bodied gait.

6.1.1 Introduction.

The kinematic patterns of gait for able-bodied individuals have previously been shown to be less variable than kinetic patterns which are in turn less variable than EMG patterns (Winter, 1987). Whilst able-bodied gait has previously been reported to be variable, ‘norms’ have been established in which to compare ‘abnormal’ or pathological gait. It is thus necessary to keep variability in able-bodied gait to a minimum and due
almost entirely to a natural variation of their walking pattern.

For lower limb sagittal plane joint angles, studies have reported coefficients of variation of 15-52% for the hip, 14-23% for the knee and 64-72% for the ankle (Winter, 1987; Smith, 1993). However, Smith (1993) stated that for able-bodied gait, any variability above 40% was unacceptable. Apkarian et al. (1989) reported only a 1-2° variation between subjects for lower limb three-dimensional flexion/extension angles, but the % variation within subjects was not noted. Therefore, in kinematic analysis, the method and type of equipment used to measure lower limb angles, whether through digitising or the use of opto-electronic systems using reflective markers, may contribute to the amount of variability. Thus, establishing the amount variability in the subject and system is important.

The higher variability reported for joint moments (±24-36% for the ankle, 62-64% for the knee and 62-66% for the hip, Winter, 1987; Smith, 1993) could not only be dependent on the natural variability of the subject, but also arise from errors in calculations. These errors can be caused from marker placement (up to 50%, Davis et al., 1997), centre of pressure alignment, and errors in force and motion spatial synchronisation (up to 14%, McCaw and DeVita, 1995) or force and motion temporal synchronisation (up to 70%, O’Connor et al., 1995). The variability from error in calculation could increase intra-subject variability further for joint power output leading to large errors in results. The purpose of this study was to establish the variation, due to errors, in data collection and calculation of joint angles, joint moments and power output for able-bodied subjects. This was done by manipulating centre of pressure (COP) synchronisation of kinematic and force data, and calculating intra-subject variability over a number of trials.
6.1.2 Methodology.

6.1.2.1 Apparatus.

Kinematic data were obtained using a ProReflex (Qualysis) opto-electronic system with a maximum sample rate of 240 Hz, which tracked 19 mm retro-reflective markers, placed on the body, through space. The ProReflex used six genlocked cameras positioned so that each marker could be seen by at least 2 cameras at all times (Figure 6.1). The system allowed the tracked data to be manually checked to ensure no markers were mixed, then exported in order to calculate the kinematic and kinetic variables.

![Diagram of the six camera ProReflex system with a force platform and markers]

The marker system consisted of 14 retro-reflective markers attached with double-sided tape to several bony landmarks: 5th metatarsal, lateral malleolus, visual centre of the axis through the knee in the sagittal plane, greater trochanter and anterior superior iliac
spine (ASIS) and acromion process for the left and right limbs, L5/S1 and C7 (Plate 4). As little clothing as possible was worn to prevent marker movement.

Plate 4 - The 14 point marker system (front and side views).

Kinetic data were obtained by synchronising the ProReflex system with a Kistler force platform (Type 9281B11) sampled at 960 Hz. A trigger system allowed data collection to commence simultaneously from ProReflex and force platform, temporally synchronising the data to within one frame which was then accounted for in subsequent calculations. The calibration frame origin for the ProReflex was placed in the centre of the force platform to ensure spatial synchronisation.
6.1.2.2 Subjects and procedure.

Five able-bodied subjects (3 females and 2 males) with a mean (± SD) age of 27.4 (± 3.97) years, height 1.71 (± 0.08) m, mass 67.5 (± 12.5) kg and with no previous injury took part in the study. Retro-reflective markers were attached with double-sided tape to the bony landmarks as previously described. The subjects walked along the walkway containing a force platform. They were allowed familiarisation with the walking speed and contacting the force platform without making any stride adjustments. The subject approached the force platform in about 6-7 strides. Walking speed was measured by timing lights. Any trial where the force platform was targeted, not hit fully or the walking speed was not within ± 3% of 1.2 m.s⁻¹ was discarded and the trial repeated. Six trials were obtained from the right leg of each subject for each testing session. Prior to hitting the force platform, the trigger was pressed and both kinematic and force data were collected simultaneously.

6.1.2.3 Data analysis.

The kinematic data were automatically tracked using the ProReflex (Qualysis) software, then manually checked. Any missing data points were filled by interpolation when exporting the data to a C.S.V. file to be read by an Acorn 5000 computer with custom written software. The custom written software then calculated the hip, knee and ankle joint angles in the sagittal plane. The ankle and knee joint centres were taken from the lateral malleolus and knee axis centre joint markers. The hip joint centre (Figure 6.3) was estimated from the greater trochanter hip marker, taking the hip centre to be a point extrapolated back from the knee past the greater trochanter by 20 mm (Plagenhoff, 1979). The data were smoothed using a Butterworth 4th order zero lag filter with a cut-off frequency of 10 Hz. The cut-off frequency was based on residual analysis (Winter, 1990)
which tends to undersmooth the data so the filter choice needs to be modified slightly by visual inspection. The cut-off frequency used was chosen after using a number of cut-off frequencies of 5 Hz, 7 Hz, 10 Hz, 12 Hz, 15 Hz and 20 Hz to establish visually whether the data were under- or over-smoothed. Values were taken for the ankle angle at heel strike and toe-off, for the knee angle at heel strike, mid-stance and toe-off, and the maximum and minimum hip angles.

Figure 6.2 – Joint angle conventions for the kinematic data.

Figure 6.3 – Location of the hip joint centre and greater trochanter.
For the kinetic analysis, the force data were resampled at 240 Hz and also exported to the Acorn 5000 computer. The synchronised force and motion data-set was then used to calculate the kinetic variables using standard inverse dynamic equations (Winter, 1980) and anthropometric data (Dempster, 1955) (Figure 6.4). The data were smoothed using a Butterworth 4th order zero lag filter with a cut-off frequency of 10 Hz (as detailed previously) and normalised to 100% of the stance phase. Sagittal plane peak ankle dorsiflexor and plantar flexor moments, peak knee flexor/extensor moments and peak hip flexor/extensor moments were obtained as well as peak ankle, knee and hip power generation and absorption for each trial in order to perform statistical analysis.

Figure 6.4 – Free body diagrams used for joint moment calculations.
For all segments the equations of linear and rotational motion are used which in general form are $\Sigma F_y = m. a_y$; $\Sigma F_z = m. a_z$ and $\Sigma M = I. \alpha$, where $F_y$, $F_z$ = applied forces in the horizontal and vertical directions respectively, $M$ = applied moment, $m$ = mass, $I$ = moment of inertia, $a_y$, $a_z$ = linear accelerations in the horizontal and vertical directions respectively and $\alpha$ = angular acceleration of the segment respectively. The linear accelerations are calculated from the kinematic data. The data co-ordinates in their respective directions $y$ and $z$ at the ankle, knee and hip-extrapolated markers are differentiated twice from linear displacement to give linear acceleration in the $y$ and $z$ directions. The angular acceleration of the segment is also calculated from the kinematic data. The joint angle (see Figure 6.2 for joint angle conventions) displacements are differentiated twice to give angular acceleration for each joint respectively.

The following equations can be specified for the individual rigid bodies of Figure 6.4 :-

For the foot:

For the foot (f)

\[
F_z + A_z - m_f \cdot g = m_f \cdot a_{zf}.
\]
\[
F_y + A_y = m_f \cdot a_{yf}.
\]
Taking moments about the ankle gives:

\[ Ma - m_c g d + F_y (b+c) + F_z (d+e) = I_f \alpha_l \]
For the shank:

\[
\begin{align*}
Kz - Az - m_g = m_s \cdot az_s \\
Ky - Ay = m_s \cdot ay_s
\end{align*}
\]

Taking moments about the knee gives

\[
Mk - Ma + Az \cdot (j+k) - Ay \cdot (f+h) + m_s \cdot g \cdot k = I_s \cdot \alpha_s
\]
For the thigh:

\[ \text{Hz - Mh} \]

Greater trochanter marker

For the thigh (t):

\[ \text{Hz - Kz - m}_t \cdot g = m_t \cdot a_z_t \]

\[ \text{Hy - Ky} = m_t \cdot a_y_t \]

Taking moments about the hip centre gives

\[ \text{Mh - Mk - Kz} \cdot (p+q) - \text{Ky} \cdot (n+l) - m_t \cdot g = I_t \cdot \alpha_t \]

Whilst it is acknowledged that 3-D analysis of gait would give more accurate results than a 2-D analysis, this was not possible with the current equipment and so the net joint moments were analysed in the sagittal plane only. Winter et al. (1994) have shown that there is very little difference in net ankle and knee joint moments and power output estimated from 2-D analysis compared to 3-D. What does differ, however, is the hip joint moment and power output. While the hip joint moment and power output profiles were similar, estimates showed the peak hip flexor and extensor moments during stance were approximately twice the magnitude of the 2-D moments. The peak hip power inputs were also proportionally greater. This was due to the differences in defining the hip joint angle in
2-D and 3-D. Another problem with estimating the hip joint moment is the marker position. Whilst it is easier and more repeatable to place a marker over a bony landmark such as the greater trochanter, there are problems with estimating the hip joint centre from this marker position (Figure 6.3), particularly in 2-D analysis where there are additional problems with segment rotations. For example, if the pelvis is rotated 4° forward and 4° backward during normal walking (Inman et al., 1994), the marker will move forward or backward by a considerable distance relative to the hip centre when viewed from the side. If the radius of rotation from marker centre to hip joint centre is estimated to be 100 mm, then 100 x sin8 gives 14 mm error in each direction. For a GRF of 700N, this would give an error of 9.8 Nm in either direction, or for a peak hip joint moment of 130 Nm (Winter, 1980), a 7.5 % error. It has been shown that this error is negligible at the ankle and knee (Winter et al., 1994). This shows that there is mainly a problem with reporting hip joint moments and power output from a 2-D analysis. However, very few published studies have estimated joint moments from a 3-D analysis and many have reported only 2-D joint moments for lower limb amputees. It is thought that any error in location and the effect of the movement cycle is likely to be more systematic than random, and it is the relative changes which are important (even if the absolute values are underestimates). Thus, this will be taken into account during the discussion of the results for this and the proceeding study.

Spatial synchronisation of the ProReflex data to the force platform reference coordinate system was achieved by transforming the ProReflex data to a reference zero located at the centre of four markers placed at the four corners of the force platform. The origins of both were found to agree to within 1-2 mm in both the ML and AP directions. The data from one subject during one walking trial was then used to determine the magnitude of the error in joint moment estimations for an offset of the force platform origin.
of ± 1 mm, ± 2 mm and ± 5 mm in both the ML and AP directions by manipulating the force platform origin position.

Coefficient of variation was calculated for the kinematic and kinetic (joint moment and power output) variables to determine intra-subject variability.
6.1.3 Results.

Spatial synchronisation.

The peak hip, knee and ankle joint moments and power outputs were calculated for one subject and one walking trial using a synchronized ProReflex and force platform data set. The peak joint moments and power outputs were then re-calculated after manipulating the force platform origin position by 1, 2 and 5 mm in the AP and ML directions using custom written software. The results are presented in Tables 6.1 and 6.2.

Table 6.1 - The error in peak joint moment (in Nm) yielded with a change in force platform origin of 1, 2 and 5 mm in the AP and ML directions. PF = plantar flexor, DF= dorsi flexor, ext = extensor and flex = flexor moment.

<table>
<thead>
<tr>
<th>Force platform origin off-set (mm)</th>
<th>Peak moment ANKLE PF (Nm)</th>
<th>Force platform origin off-set (mm)</th>
<th>Force platform origin off-set (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+1</td>
<td>-1 +2 +5</td>
<td>+1 +2 +5</td>
</tr>
<tr>
<td>ankle PF</td>
<td>0.81 -0.76 1.67 1.89 3.77 -4.05</td>
<td>ankle DF -0.42 -0.41 -0.17 -0.29 -0.36 -0.41</td>
<td>knee ext -0.46 1.02 -0.87 1.65 -2.88 3.81</td>
</tr>
<tr>
<td>knee flex</td>
<td>0.65 -0.44 0.68 0.01 2.45 -1.01</td>
<td>knee flex 0.65 -0.44 0.68 0.01 2.45 -1.01</td>
<td>knee flex 0.65 -0.44 0.68 0.01 2.45 -1.01</td>
</tr>
<tr>
<td>hip ext</td>
<td>1.03 -0.21 1.87 -1.34 3.86 -3.15</td>
<td>hip flex -1.47 0.72 -1.27 0.81 -4.23 4.16</td>
<td>hip flex -1.47 0.72 -1.27 0.81 -4.23 4.16</td>
</tr>
</tbody>
</table>

Changing the force platform origin in the AP direction yielded the largest change in peak joint moment or power output compared to changing the origin in the ML direction. In the AP direction, moving the origin posterioraly reduced all moments except knee extensor and hip flexor. The largest change in magnitude was seen for the ankle plantar flexor moment, reducing by 0.8 Nm (0.8%) for an offset of 1 mm, by 1.9 Nm (2.0%) for an offset of 2 mm.
and by 4.1 Nm (4.3%) for an offset of 5 mm. Moving the origin anteriorly increased the magnitude of all joint moments except knee extensor, hip flexor and ankle dorsiflexor. The largest increase in magnitude was seen again for the ankle plantar flexor moment, increasing by 0.8 Nm (0.8%) for an offset of 1 mm, by 1.7 Nm (1.7%) for an offset of 2 mm and by 3.8 Nm (4.0%) for an offset of 5 mm.

Table 6.2 - The error in peak joint power output (W) yielded with a change in force platform origin of 1, 2 and 5 mm in the AP and ML directions. Gen indicates power generated, abs, power absorbed.

<table>
<thead>
<tr>
<th>Force platform origin off set (mm)</th>
<th>Peak power output (W)</th>
<th>AP</th>
<th>ML</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>+1</td>
<td>-1</td>
</tr>
<tr>
<td>ankle gen</td>
<td>1.12</td>
<td>-1.37</td>
<td>1.39</td>
</tr>
<tr>
<td>ankle abs</td>
<td>-0.81</td>
<td>-1.67</td>
<td>2.13</td>
</tr>
<tr>
<td>knee gen</td>
<td>0.43</td>
<td>-0.56</td>
<td>0.43</td>
</tr>
<tr>
<td>knee abs</td>
<td>-0.43</td>
<td>2.51</td>
<td>-1.88</td>
</tr>
<tr>
<td>hip gen</td>
<td>-0.32</td>
<td>0.56</td>
<td>-0.22</td>
</tr>
<tr>
<td>hip abs</td>
<td>-0.85</td>
<td>0.42</td>
<td>-1.02</td>
</tr>
</tbody>
</table>

The largest increase in peak power output was seen for knee power absorption.

Offsetting the force platform posteriorally in the AP direction yielded an increase of 2.5 W (2.3%) for 1 mm, 3.0 W (2.7%) for 2 mm and 7.2 W (6.6%) for 5 mm. Anteriorly, there was a decrease in magnitude of 0.4 W (0.3%) for 1 mm, 1.9 W (1.7%) for 2 mm and 5.8 W (5.3%) for 5 mm.
Intra-subject variability.

Table 6.3 shows intra-subject variation (coefficient of variation) for able-bodied kinematics as measured by the ProReflex system during walking at 1.2 m.s\(^{-1}\). The largest variation in joint angle appears for ankle plantar flexion (\(\pm 3.58\) %), with the hip maximum extension angle (\(\pm 0.31\) %) being the least variable. Coefficient of variation was low for all kinematic variables measured.

Table 6.3 - Intra-subject variability (coefficient of variation) for able-bodied kinematics during walking at 1.2 m.s\(^{-1}\) (6 trials used).

<table>
<thead>
<tr>
<th>Subject</th>
<th>Coefficient of variation (%)</th>
<th>Ankle angle</th>
<th>Knee angle</th>
<th>Hip angle</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Plantar flex dorsi flex</td>
<td>heel strike</td>
<td>midstance</td>
</tr>
<tr>
<td>1</td>
<td>3.58</td>
<td>1.08</td>
<td>0.63</td>
<td>1.14</td>
</tr>
<tr>
<td>2</td>
<td>3.54</td>
<td>0.96</td>
<td>0.55</td>
<td>1.02</td>
</tr>
<tr>
<td>3</td>
<td>2.28</td>
<td>1.28</td>
<td>0.77</td>
<td>0.90</td>
</tr>
<tr>
<td>4</td>
<td>3.21</td>
<td>1.12</td>
<td>0.58</td>
<td>1.08</td>
</tr>
<tr>
<td>5</td>
<td>3.25</td>
<td>0.98</td>
<td>0.61</td>
<td>1.12</td>
</tr>
</tbody>
</table>

Table 6.4 shows intra-subject variation (coefficient of variation) for able-bodied kinetics as measured by the ProReflex and force platform during walking at 1.2 m.s\(^{-1}\). The largest variation in joint moment for all subjects appears for the hip extensor moment (\(\pm 10.6\) %), with the ankle plantar flexor moment (\(\pm 1.40\) %) being the least variable of all subjects. The coefficient of variation appears to be larger for joint power output for all subjects, \(\pm 20.6\) % for hip power generation, with ankle power generation exhibiting the smallest intra-subject variation of all subjects (\(\pm 7.78\) %).
Table 6.4 - Intra-subject variability (coefficient of variation %) for able-bodied kinetics during walking at 1.2 m.s\(^{-1}\). PF = planar flexor, DF= dorsi flexor, ext = extensor and flex = flexor moment. Gen indicates power generated, abs, power absorbed (6 trials used).

<table>
<thead>
<tr>
<th>Subject</th>
<th>Coefficient of variation (%)</th>
<th>Net Joint Moment</th>
<th>Joint Power Output</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ankle PF 7.11 7.15 7.15</td>
<td></td>
<td>Ankle Hip Gen Abs</td>
</tr>
<tr>
<td>1</td>
<td>1.91 7.52 9.68 8.94</td>
<td></td>
<td>13.8 15.3 13.6 7.90 9.46</td>
</tr>
<tr>
<td>2</td>
<td>4.25 4.07 3.81 9.19</td>
<td></td>
<td>7.78 17.7 12.1 15.8 12.1</td>
</tr>
<tr>
<td>3</td>
<td>1.40 8.33 10.6 10.1</td>
<td></td>
<td>8.78 11.2 19.1 20.2 19.1</td>
</tr>
<tr>
<td>4</td>
<td>2.30 5.15 5.73 8.98</td>
<td></td>
<td>9.21 11.3 16.2 15.8 7.96 14.8</td>
</tr>
<tr>
<td>5</td>
<td>3.21 9.42 9.89</td>
<td></td>
<td>7.64 10.4 16.5 16.1 9.17 11.3</td>
</tr>
</tbody>
</table>

213
6.1.4 Discussion.

The purpose of this study was to establish the amount of variation that could arise from errors in data collection and calculation of joint angles, joint moments and power output for able-bodied subjects by manipulating centre of pressure (COP) synchronisation of kinematic and force data, and calculating intra-subject variability over a number of trials. Whilst it is acknowledged that errors arise from hip joint centre location from a hip marker placed on the greater trochanter in 2-D analysis, many studies have reported hip joint angles and moments using this technique (Winter et al., 1994). It is expected that the error is more systematic than random and that this could account for up to a 7.5% error in joint moments.

The spatial synchronisation of ProReflex system and force platform was found to yield an error of up to 1-2 mm in the AP and ML directions. The first part of this study determined what effect this error would have on net joint moment and joint power output magnitude. The largest effect on joint moment and power output magnitude was found when offsetting the centre of pressure (COP) in the AP direction. For the net joint moments, the largest change in magnitude was seen for the ankle plantar flexor moment, a change of 0.8 to 2.0% for an offset of 1 to 2 mm which rose to 4.3% for an offset of 5 mm.

For an adult male, when normalised to body weight, this error would equate from 0.01 Nm.kg\(^{-1}\) to 0.02 Nm.kg\(^{-1}\), or 0.05 Nm.kg\(^{-1}\) for a 5 mm offset. McCaw and DeVita (1995) investigated errors in alignment of COP in force and motion synchronisation and found that moving the COP posteriorly by 5 mm led to a 7% error in peak joint moment magnitude. These results are slightly higher but comparable to the present study, which reported a maximum increase in joint moment magnitude of 4.3% and joint power output of 6.6% for a 5 mm offset. Thus, the results in this study for an error in COP alignment of 1 to 2 mm
yields a maximum of 2.0% error in peak joint moment magnitude and a maximum error of
2.7% in peak power output. This error is expected to fall within the natural variation of
joint moments and power output in human walking.

The coefficient of variation was found to be lower for all kinematic variables
(maximum ± 3.58%) than for all kinetic variables (maximum joint moment ± 10.6%,
maximum power output ± 20.6%). This difference has also been reported in previous
studies. Winter (1987) suggested that kinematic aspects of gait tended to be less variable
than kinetic patterns. Smith (1993) reported coefficient of variation of one subject over a
number of trial for sagittal plane lower limb angles as ± 13% for the hip, ± 11% for the
knee and ± 56% for the ankle. Whilst the ankle showed the greatest variability in the
present study, the results of kinematic variability were all lower than those reported by
Smith (1993). As all intra-subject kinematic variability in the present study was less than ±
4% for all subjects walking, this was deemed to be good repeatability.

For net joint moments and power output, the coefficient of variation is expected to be
higher than for kinematic variables (Winter, 1987). In the present study, the highest
variation in net joint moments for all subjects was ± 10.6%. Joint moment intra-subject
variability has previously been reported between ± 24-36% for the ankle, ± 62-64% for the
knee and ± 62-66% for the hip (Winter, 1987; Smith, 1993), all greater than for the present
study. In the present study, spatial synchronisation errors of kinematic and force data were
kept to a minimum, a high sampling rate was used to reduce possible temporal
synchronisation errors, and user-digitising errors were reduced by using an opto-electronic
motion analysis system. In manual digitizing, it can be difficult for even a highly trained
human eye and perception system to pinpoint joint centres, increasing the likelihood of errors from inconsistencies or inaccuracies. For opto-electronic systems, the size of the reflective marker is known, and the system is able to pinpoint the marker centre with high accuracy. What does become a problem with these systems is when a marker is completely obscured, or partially obscured such as when the hand passes the hip marker. When a part of the marker is obscured, the opto-electronic system will calculate the centre of the marker from the remaining part it can detect. Thus, errors can arise in located hip marker position at certain parts of the gait cycle. The above discussed factors may be responsible for the low intra-subject variability in the present study compared to previous research.

Joint power outputs have previously been reported to be more variable than for net joint moments (Winter, 1987; Smith, 1993). In the present study, the greatest intra-subject variability was seen for hip power generation at ± 20.6%. Whilst few studies have investigated intra-subject variability for joint power output, two studies reported coefficients of variation of ± 51-129% for the ankle, ± 68-110% for the knee and ± 74-80% for the hip (Winter, 1987; Smith, 1993). Again, the results of the present study are much lower than those previously reported. While this is true, an intra-subject variation of ± 20% is not particularly low, joint power outputs have previously been reported to be highly variable, both within- and between-subjects due to natural movement variability. Earlier results in this study have shown low intra-subject variability and therefore the methodology used was deemed to produce good repeatability.

Previous studies have reported that the most influencing errors in kinetic calculations have been shown to stem from spatial and temporal synchronisation problems of force and
motion data (McCaw and DeVita, 1995; O'Connor et al., 1995). The results of the present study have shown that the possible errors in spatial synchronisation that could occur with this methodology are very small (less than 3%). There is low intra-subject variability compared to previous studies. Thus, using this methodology, errors are kept to a minimum and intra-subject variability for kinematics and joint moments is low.

6.1.5 Conclusion.

The results of this study have shown that the spatial synchronisation errors in calculating kinetic variables were small (less than 3%) when manipulating COP synchronisation by 1-2 mm. Intra-subject variation was very low for all kinematic variables (less than ±4%) and low for net joint moments (less than ±10.6%). Joint power outputs yielded the highest intra-subject variation (up to ±20.6%) but humans naturally exhibit greater variability in joint power output whilst walking. Intra-subject variation for all variables in this study was lower than previously reported for able-bodied walking. Thus the methodology used was deemed to produce good repeatability.
6.2 The effect of walking speed on kinematic and kinetic asymmetry during walking.

6.2.1 Introduction.

Research into gait patterns of amputees with different prostheses and different prosthetic alignment has been well documented. For kinematic analysis, studies have looked at 2-D and 3-D joint angles of above- and below-knee amputees, but not the level of asymmetry between the limbs. These studies are difficult to compare as usually the subject's natural cadence is reported, thus walking speed is not consistent between studies. For the analysis of joint moments and joint power output, there are additional problems. Few studies have taken into account that the anthropometry of the residual plus prosthetic limb differs to that of an able-bodied person. It has been shown that measuring the dimensions of the residual plus prosthetic limb, and modifying the joint moment calculations accordingly yield only a ± 4 Nm difference in magnitude compared to using standard anthropometric data (Miller, 1987) for amputees running. Thus, for walking, it is expected that the difference between modifying the joint moment calculations and using able-bodied anthropometric data would give error values that would be expected to fall within natural movement variability.

Past literature suggests that unilateral lower limb amputees compensate for the functional loss of one or more joints by increasing ankle and knee range of motion and joint moment and power output amplitude on their intact limb, and thus illustrate kinematic and kinetic asymmetry. They have also been reported to increase hip moment and power output on their prosthetic limb compared to both their intact limb and to able-bodied subjects. However, no studies found have looked at the effect of placing increased demands on the amputee, such as increased walking speed, on kinematic and kinetic asymmetry. As lower limb amputees have been found to increase loading asymmetry at the higher speeds of
walking, and need to attenuate more impact shock, they may compensate by altering their
gait kinematics or kinetics. In this case, asymmetry observed in their joint kinematic and
kinetics pattern may increase with walking speed as they cope with the greater demands, or
decrease as observed in chapter 4 for temporal kinematics, allowing them to co-ordinate
their gait pattern to reach the faster walking speeds. Further investigation in this area will
lead to a better understanding of compensatory gait patterns seen in lower limb unilateral
amputees. The purpose of this study was to investigate the effect of increased walking
speed on kinematic and kinetic gait variables and their asymmetry in unilateral lower limb
amputees.

For the kinematic analysis:

It is hypothesised that:

Hypothesis 1: Above-knee amputees have reduced ankle plantar flexion angles on
their prosthetic limb compared to their intact limb and to able-bodied subjects due to problems with actively flexing the prosthetic ankle joint.

Hypothesis 2: Below-knee amputees have reduced ankle plantar flexion angles on
their prosthetic limb compared to their intact limb and to able-bodied subjects due to problems with actively flexing the prosthetic ankle joint.

Hypothesis 3: Above-knee amputees have reduced knee flexion angles on their
prosthetic limb compared to their intact limb and to able-bodied
subjects due to problems with actively flexing the prosthetic knee joint.

**Hypothesis 4:** Joint kinematic asymmetry does not increase with walking speed for amputees or able-bodied subjects.

For the kinetic analysis:

It is hypothesised that:

**Hypothesis 5:** Above-knee amputees have reduced ankle joint moments and power output on their prosthetic limb compared to their intact limb and to able-bodied subjects.

**Hypothesis 6:** Below-knee amputees have reduced ankle joint moments and power output on their prosthetic limb compared to their intact limb and to able-bodied subjects.

**Hypothesis 7:** Above-knee amputees have reduced knee joint moments and power output on their prosthetic limb compared to their intact limb and to able-bodied subjects.

**Hypothesis 8:** Below-knee amputees have reduced knee joint moments and power output on their prosthetic limb compared to their intact limb and to able-bodied subjects.
Hypothesis 9: Above-knee amputees produce greater hip flexor and extensor moments on their prosthetic limb compared to their intact limb and to able-bodied subjects.

Hypothesis 10: Below-knee amputees produce greater hip flexor and extensor moments on their prosthetic limb compared to their intact limb and to able-bodied subjects.

Hypothesis 11: Above-knee amputees generate and absorb more hip power on their prosthetic limb compared to their intact limb and to able-bodied subjects.

Hypothesis 12: Below-knee amputees generate and absorb more hip power on their prosthetic limb compared to their intact limb and to able-bodied subjects.

Hypothesis 13: Joint moment asymmetry does not increase with walking speed for amputees and able-bodied subjects.

Hypothesis 14: Joint power output asymmetry does not increase with walking speed for amputees and able-bodied subjects.
6.2.2 Methodology.

6.2.2.1 Apparatus.

The apparatus - ProReflex (Qualysis) system, Kistler force platform and 14 point marker system - used in this study was the same as was established in study 6.1.

6.2.2.2 Subjects and procedure.

The subjects were all established unilateral below-knee (n=4, 1 female, 3 males) or above-knee (n=4, all male) amputees. They were all volunteers and regularly took part in sport either recreationally or competitively. The below-knee amputees had a mean age (± SD) of 44.3 ± 17.5 years, mass 82.7 ± 12.5 kg and had been amputees for a mean of 14.0 ± 10.1 years. The above-knee amputees had a mean age (± SD) of 59.3 ± 16.8 years, mass 85.6 ± 12.6 kg and had been amputees for a mean of 21.3 ± 16.0 years. The able-bodied subjects (n=8, 3 females, 5 males) had a mean age (± SD) of 26.1 ± 3.98 years and mass 74.4 ± 14.0 kg. They also participated regularly in sport and had no history of injury prior to testing. Limb dominance was ascertained as in study 5.1. Retro-reflective markers were attached with double-sided tape to several bony landmarks as in study 6.1: 5th metatarsal, lateral malleolus, visual centre of the axis of the knee in the sagittal plane, greater trochanter and anterior superior iliac spine (ASIS) and acromion process for the left and right limbs, L5/S1 and C7. For the amputees’ prosthetic limb, the 5th metatarsal marker was placed in the same position (distance measured from the front of the shoe and up from the floor) as on the intact limb. For the lateral malleolus, the distance of the marker up from the floor was measured on the intact limb and placed in the same position on the prosthetic limb. For the above-knee amputees, no subjects wore a cosmesis and the reflective marker
was placed on the exact centre of the knee joint. As little clothing as possible was worn to prevent marker movement.

The subjects walked along a walkway containing a force platform. They were allowed familiarisation with the walking speed and hitting the force platform without making any stride adjustments. The subject approached the force platform in about 6-7 strides and carried on walking at the same speed for about 6-7 strides. Walking speed was measured by means of timing lights. Any trial where the force platform was targeted, not hit fully or the walking speed was not within ± 3% of the predetermined speeds of 0.5, 0.9 and 1.2 m.s⁻¹ was discarded and the trial repeated. The subjects also walked at maximum walking speed, and this speed was also recorded (ranging from 1.5 to 1.9 m.s⁻¹). Three trials were obtained for each speed for both legs of each subject. Prior to hitting the force platform, a trigger was pressed and both kinematic (sample rate 240 Hz) and force data (sample rate 960 Hz) were collected simultaneously to within one frame error which was then accounted for in subsequent calculations.

6.2.2.3 Data analysis.

The kinematic data were automatically tracked using the ProReflex (Qualysis) software, then manually checked. Any missing data points were filled by interpolation when exporting the data to a C.S.V. file to be read by an Acorn 5000 computer with custom written software. The custom written software then calculated the hip, knee and ankle joint angles in the sagittal plane, and smoothed the data using a Butterworth 4th order zero lag filter with a cut-off frequency of 10 Hz (as established in study 6.1). Mean values were taken for the ankle at heel strike and toe-off, for the knee at heel strike, mid-stance and toe-off, and for the maximum and minimum hip values of each limb.
For the kinetic analysis, the force data were resampled at 240 Hz and also exported to the Acorn 5000 computer. The synchronised force and motion data-set was then used to calculate the kinetic variables using standard inverse dynamics procedure (Winter, 1980) and anthropometric data (Dempster, 1955) for the able-bodied subjects, which were then normalized to body weight and 100% of the stance phase. Kinetic variables for the amputees were then calculated in the same way as for able-bodied subjects. Sagittal plane peak ankle dorsiflexor and plantarflexor moments, peak knee flexor/ extensor moments and peak hip flexor/ extensor moments were obtained as well as peak ankle, knee and hip power generation and absorption for each trial in order to perform statistical analysis. Means were calculated for each subject for the three trials. These mean results for the amputees were then grouped for the below-knee amputee prosthetic limb, below-knee amputee intact limb, above-knee amputee prosthetic limb and above-knee amputee intact limb. The amputees were grouped instead of being treated as case studies because they all wore the same prosthesis, were all 'established amputees' and therefore considered homogenous.

The data were initially tested for normality and homogeneity. Correlations were performed for each variable between the intact and prosthetic limbs of the above- and below-knee amputees to ensure they were independent, and thus did not violate the assumptions of an analysis of variance test (appendix 5). There was no significant correlation at the 5% level between the prosthetic and intact limbs either for above- or below-knee amputees for any variable. The intact and prosthetic limbs of the amputees were thus deemed to be independent and could therefore be included as separate groups in a two-way analysis of variance.
A two-way analysis of variance was performed on all kinematic and kinetic variables separately (i.e. mean peak ankle joint angle, mean peak knee extensor moment, mean peak hip power generation etc.) to determine any differences between group, walking speed and any group by speed interaction. Four walking speeds (within-factor) of 0.5, 0.9, 1.2 m.s\(^{-1}\) and maximum, and five groups (between-factor) were used: (i) the average of both limbs for able-bodied subjects, (ii) the below-knee amputee intact limb, (iii) the below-knee amputee prosthetic limb, (iv) the above-knee amputee intact limb, (v) the above-knee amputee prosthetic limb. Thus, the results of group, speed and group by speed interaction effects will be discussed separately for each variable. All statistical analyses were performed using SPSS (version 9.0) which gave the option of including calculations of the statistical power of the test and allowed for different numbers of subjects in each group. Level of significance was set at 5%. A post-hoc Tukey HSD test was performed for all two-way analysis of variance tests.

The absolute symmetry index (Herzog et al., 1989) was used to calculate asymmetry for the peak values between the limbs of each subject. For asymmetry, a two-way analysis of variance was also performed to investigate (i) differences between groups (between-factor), (ii) the effect of walking speed on each variable (within-factor) and (iii) any group by speed interaction. Four walking speeds (0.5, 0.9, 1.2 m.s\(^{-1}\) and maximum) and three groups were used: (i) asymmetry calculated between the left and right limbs of able-bodied subjects, (ii) asymmetry calculated between the prosthetic and intact limbs of below-knee amputees and (iii) asymmetry calculated between the prosthetic and intact limbs of above-knee amputees. The two-way analysis of variance was performed for each variable (i.e. ankle plantar flexor moment asymmetry, knee joint power generation asymmetry and so on) using the averaged asymmetry values calculated from consecutive steps for each
subject. The level of significance was set at 5%. Thus the results of group, speed and group by speed effects will be discussed separately for each asymmetry variable calculated. Again, a post-hoc test (Tukey HSD) was performed on all the analysis of variance tests.
6.2.3 Results.

The results for the maximum walking speed which ranged from 1.5 m.s\(^{-1}\) in the amputees to 1.9 m.s\(^{-1}\) in the able-bodied subjects were compared directly, even though the speeds differed, because the effect of their maximum possible walking speed on the chosen variables for each individual needed to be seen. Whilst it is appreciated that these speeds differed from individual to individual, the aim was to investigate the effect of their absolute maximum walking speed as an indication of the maximum demands placed on that individual during walking. Thus, all maximum values will be compared across individuals, marked separately on the graphs by a discontinued line //.

6.2.3.1 Kinematic analysis.

Able-bodied subjects.

Joint angles.

Data on mean peak joint angles of able-bodied subjects at different walking speeds are given in Figure 6.5. It appears that ankle plantar flexion angle increased with increasing walking speed. Whilst the knee appears to be only slightly more flexed at heel strike at the maximum walking speed compared to the other speeds, knee angle at mid-stance appears to decrease with walking speed. Thus, the knee flexes more at mid-stance, the faster the walking speed. Hip flexion angle also appears to reduce slightly with increasing walking speed. There appears to be no other effect of walking speed on lower limb joint angles. The data for the mean of the left and right limb are reported in Table 6.7.
Figure 6.5 – Mean peak joint angles of able-bodied subjects walking at different speeds.

Black line indicates dominant limb, red non-dominant limb.
Table 6.5 - Maximum and minimum mean joint angles (degrees) from the mean of both limbs of able-bodied subjects walking at different speeds. HS indicates heel strike, MS mid-stance and TO toe off.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Max Ankle (°)</th>
<th>Min Ankle (°)</th>
<th>HS Knee (°)</th>
<th>Min Knee (°)</th>
<th>Max Hip (°)</th>
<th>Min Hip (°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>115.3</td>
<td>89.0</td>
<td>170.0</td>
<td>163.8</td>
<td>158.1</td>
<td>175.7</td>
</tr>
<tr>
<td>0.9</td>
<td>118.8</td>
<td>89.7</td>
<td>170.3</td>
<td>160.2</td>
<td>157.4</td>
<td>174.8</td>
</tr>
<tr>
<td>1.2</td>
<td>120.6</td>
<td>89.4</td>
<td>171.4</td>
<td>153.7</td>
<td>155.6</td>
<td>174.4</td>
</tr>
<tr>
<td>max</td>
<td>126.2</td>
<td>90.7</td>
<td>168.0</td>
<td>146.6</td>
<td>151.5</td>
<td>171.1</td>
</tr>
</tbody>
</table>

Joint angle asymmetry.

For all lower limb joint angles, asymmetry was less than 4% for able-bodied subjects (Table 6.6). There appeared to be no effect of speed on joint angle asymmetry for able-bodied subjects (Figure 6.6).

Figure 6.6 - Lower limb joint angle asymmetry for able-bodied subjects walking at different speeds.
Table 6.6 - Joint angle asymmetry (%) for able-bodied subjects walking at different speeds.

HS indicates heel strike, MS midstance and TO toe off.

<table>
<thead>
<tr>
<th>Walking speed (m.s(^{-1}))</th>
<th>Ankle ASI (%)</th>
<th>Knee ASI (%)</th>
<th>Hip ASI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max</td>
<td>Min</td>
<td>HS</td>
<td>MS</td>
</tr>
<tr>
<td>0.5</td>
<td>0.23</td>
<td>1.03</td>
<td>1.57</td>
</tr>
<tr>
<td>0.9</td>
<td>0.29</td>
<td>1.05</td>
<td>2.13</td>
</tr>
<tr>
<td>1.2</td>
<td>0.81</td>
<td>0.78</td>
<td>1.33</td>
</tr>
<tr>
<td>max</td>
<td>0.62</td>
<td>2.87</td>
<td>0.43</td>
</tr>
</tbody>
</table>

The level of asymmetry between the limbs of the able-bodied subjects was found to be less than 10.5% for all kinematic and kinetic variables. Thus, the mean value for the two limbs was calculated for all variables and used for comparison to the amputees.

**Amputees.**

**Joint angles.**

Figure 6.7 shows mean peak joint angles of amputees and able-bodied subjects walking at different speeds. It appeared that for both the intact and prosthetic limb, the maximum ankle plantar flexion angle increased with walking speed. Intact knee flexion angle at mid-stance was seen to decrease with walking speed, whilst the prosthetic limb appeared to be affected little by walking speed.
Figure 6.7 – Mean peak joint angles of amputees and able-bodied subjects walking at different speeds. Thick black line indicates able-bodied subjects, red line above-knee and green line below-knee amputees. Solid line indicates intact limb, dotted line prosthetic limb.
The results of the two-way analysis of variance will now be presented. The exact p values for each significant test will also be presented. A p value of p = 0.000 does not represent p = 0, rather that p is less that 0.0005. As all other p values are given exactly, this convention of reporting p to three decimal places was undertaken for consistency. From the results of the two-way analysis of variance, there was a difference between groups for plantarflexion angle \( (F_{4,19} = 6.24, p=0.000) \). From the results of the post-hoc test, differences were found between the intact and prosthetic limbs of above-knee \( (p=0.018) \) and below-knee amputees \( (p=0.032) \) and also between the prosthetic limb and able-bodied subjects for above-knee amputees \( (p=0.05) \) and below-knee amputees \( (p=0.047) \). The plantarflexion angle increased with walking speed \( (F_{1,19} = 11.34, p=0.003) \). There was no significant interaction.

No differences were found for the dorsiflexion angle.

For all amputees, intact limb knee angle at heel strike was slightly greater than for the prosthetic limb and for able-bodied subjects, but was not significant \( (F_{4,19} = 2.55, p=0.073) \). However, the power of the test was low \( (0.604) \) and this may account for the non-significant result. There was no effect of walking speed found. For knee angle at mid-stance (the ‘cushioning’ phase) there was a significant difference between groups \( (F_{4,19} = 23.34, p=0.000) \). From the results of the post-hoc test, these differences were evident between the prosthetic and intact limbs of the above-knee amputees \( (p=0.002) \) and below-knee amputees \( (p=0.043) \). Differences were also observed between the above-knee amputee prosthetic limb and able-bodied subjects \( (p=0.000) \) and the below-knee amputee intact limb and able-bodied subjects \( (p=0.002) \). There was also a significant interaction of group by speed \( (F_{4,19} = 15.97, p=0.000) \) showing that one limb (the intact limb) was more affected by speed than the other (the prosthetic limb), the intact limb knee flexion angle reducing with walking speed whilst the knee flexion angle in the prosthetic limb reduced very little. There was a difference.
between groups for knee extension at toe-off ($F_{4,19} = 6.31, p=0.002$), the post-hoc test revealing differences between the above-knee amputee prosthetic and intact limbs ($p=0.016$) and above-knee amputee prosthetic limb and able-bodied subjects ($p=0.001$). There was no significant effect of speed and no group by speed interaction.

For the hip, there was a difference between subjects for hip flexion ($F_{4,19} = 5.14, p=0.006$). The post-hoc test showed differences between the below-knee amputee intact and prosthetic limbs ($p=0.013$). Hip flexion was found to increase with increasing walking speed ($F_{1,19} = 10.97, p=0.004$). There was no significant group by speed interaction. For hip extension angle, there was a difference between groups ($F_{4,19} = 5.01, p=0.006$). The post-hoc test showed differences between the below-knee amputee intact and prosthetic limbs ($p=0.029$), and between the below-knee amputee prosthetic limb and able-bodied subjects ($p=0.009$). There was no effect of speed on hip extension angle and no significant interaction.

Joint angle asymmetry.

From the results of the two-way analysis of variance and post-hoc test on gait asymmetry, differences were found between groups for ankle plantar flexion angle ($F_{2,13} = 7.99, p=0.005$) and ankle dorsiflexion angle ($F_{2,13} = 6.03, p=0.014$) asymmetry. The results of the post-hoc test showed differences between above-knee amputees and able-bodied subjects for ankle plantar flexion asymmetry ($p=0.013$) and ankle dorsiflexion asymmetry ($p=0.014$), the above-knee amputees being more asymmetrical. Differences were also established between above- and below-knee amputees ($p=0.012$) for ankle dorsiflexion asymmetry. No effect of speed was found on ankle joint angle asymmetry.
For knee angle asymmetry, no differences between the groups were reported for knee angle at heel strike, nor was there an effect of walking speed. For knee angle asymmetry at mid-stance, a difference was found between groups ($F_{2,13} = 18.25, p=0.000$), specifically between above-knee amputees and able-bodied subjects ($p=0.000$), below-knee amputees and able-bodied subjects ($p=0.031$) and between above- and below-knee amputees ($p=0.048$). The above-knee amputees were more asymmetrical than the below-knee amputees, who were more asymmetrical than the able-bodied subjects. Knee angle asymmetry at mid-stance was found to increase with walking speed ($F_{3,39} = 21.52, p=0.000$) and there was a significant group by speed interaction ($F_{6,39} = 11.11, p=0.000$), amputee knee joint angle asymmetry being more affected by walking speed than able-bodied subjects. For knee angle asymmetry at toe-off, there was no effect of walking speed, but there was a difference found between groups ($F_{2,13} = 17.76, p=0.000$) with above-knee amputees being more asymmetrical than below-knee amputees ($p=0.006$) and able-bodied subjects ($p=0.000$).

For hip flexion angle asymmetry, there was no effect of walking speed, but a difference was found between groups ($F_{2,13} = 7.33, p=0.007$) with both the above-knee ($p=0.036$) and below-knee amputees ($p=0.013$) being more asymmetrical than able-bodied subjects. The same was found to be true for hip extension asymmetry. A difference between groups ($F_{2,13} = 9.12, p=0.003$) was evident as both the above-knee ($p=0.006$) and below-knee amputees ($p=0.020$) were more asymmetrical than the able-bodied subjects.
Figure 6.8 – Asymmetry of kinematic variables for a) above-knee and b) below-knee amputees during walking at different speeds.

6.2.3.2 Kinetic analysis.

Able-bodied subjects.

Figures 6.9 to 6.11 show examples of the average net joint moment curves for one able-bodied subject.

Figure 6.9 - an example of the average ankle joint moment curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).
Figure 6.10 - an example of the average knee joint moment curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).

Figure 6.11 - an example of the average hip joint moment curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).

Net joint moments.

For net joint moments of able-bodied subjects (Figure 6.12) it appears that both the mean peak knee and mean peak hip flexor and extensor moments increased with increasing speed. Black line indicates dominant femoral condylar and anterolateral femoral condylar moments (Table 6.7).
walking speed. There was only a slight increase in mean peak ankle plantar flexor and dorsi
flexor moments (Table 6.7).

Figure 6.12 – Mean peak net joint moments of able-bodied subjects walking at different
speeds. Black line indicates dominant limb, red non-dominant limb.
Table 6.7 - Mean peak net joint moments (Nm.kg\(^{-1}\)) from both limbs of able-bodied subjects walking at different speeds. PF indicates plantar flexor moment, DF dorsiflexor moment, Flex flexor moment and Ext extensor moment.

<table>
<thead>
<tr>
<th>Walking speed (m.s(^{-1}))</th>
<th>Ankle PF</th>
<th>Ankle DF</th>
<th>Knee Flex</th>
<th>Knee Ext</th>
<th>Hip Flex</th>
<th>Hip Ext</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>1.26</td>
<td>0.03</td>
<td>0.17</td>
<td>0.32</td>
<td>0.38</td>
<td>0.42</td>
</tr>
<tr>
<td>0.9</td>
<td>1.31</td>
<td>0.04</td>
<td>0.23</td>
<td>0.45</td>
<td>0.56</td>
<td>0.57</td>
</tr>
<tr>
<td>1.2</td>
<td>1.37</td>
<td>0.04</td>
<td>0.33</td>
<td>0.57</td>
<td>0.75</td>
<td>0.86</td>
</tr>
<tr>
<td>max</td>
<td>1.43</td>
<td>0.06</td>
<td>0.63</td>
<td>1.01</td>
<td>1.20</td>
<td>1.67</td>
</tr>
</tbody>
</table>

Joint moment asymmetry.

Asymmetry for all able-bodied joint moments was found to be under 10.5% (Table 6.8). There also appeared to be no effect of walking speed on able-bodied joint moment asymmetry (Figure 6.13).

Figure 6.13 – Mean net joint moment asymmetry for able-bodied subjects walking at different speeds.
Table 6.8 – Mean net joint moment asymmetry (%) for able-bodied subjects walking at different speeds. PF indicates plantar flexor moment, DF dorsi flexor moment, Flex flexor moment and Ext extensor moment.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Ankle ASI (%)</th>
<th>Knee ASI (%)</th>
<th>Hip ASI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PF</td>
<td>DF</td>
<td>Flex</td>
</tr>
<tr>
<td>0.5</td>
<td>0.67</td>
<td>2.56</td>
<td>3.34</td>
</tr>
<tr>
<td>0.9</td>
<td>3.44</td>
<td>3.68</td>
<td>6.63</td>
</tr>
<tr>
<td>1.2</td>
<td>5.18</td>
<td>1.20</td>
<td>3.70</td>
</tr>
<tr>
<td>max</td>
<td>2.83</td>
<td>0.98</td>
<td>1.01</td>
</tr>
</tbody>
</table>

Amputees.

Net joint moments.

Figures 6.14 to 6.16 show an example of the net joint moment traces for an above-knee and a below-knee amputee.

Figure 6.14 – an example of the net ankle joint moment curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s⁻¹.
Figure 6.15 – an example of the net knee joint moment curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s\(^{-1}\).

Figure 6.16 – an example of the net hip joint moment curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s\(^{-1}\).

Figure 6.17 shows mean peak net joint moments of above-knee amputees and able-bodied subjects walking at different speeds. It appears that for the intact limb, net ankle, knee and hip joint moments all increased with walking speed. For the prosthetic limb ankle moments and knee peak extensor moment, there seems to be little effect of walking speed.
Figure 6.17 – Mean peak net joint moments of amputees and able-bodied subjects walking at different speeds. Thick black line indicates able-bodied subjects, red line above-knee amputees, green line below-knee amputees. Solid line indicates intact limb, dotted line prosthetic limb.
From the results of the two-way analysis of variance and post-hoc test, a difference was found between groups for plantar flexor moment. \( (F_{4,19} = 11.31, p=0.000) \). The results of the post-hoc-test revealed differences between the intact and prosthetic limbs for both the above-knee amputees \( (p=0.022) \) and below-knee amputees \( (p=0.000) \). Differences for plantar flexor moment were also apparent between the above-knee amputee prosthetic limb and able-bodied subjects \( (p=0.010) \), and the below-knee amputee intact limb and able-bodied subjects \( (p=0.002) \). The plantar flexor moment was found to increase with walking speed \( (F_{1,19} = 29.74, p=0.000) \) and there was no significant interaction. No differences were found for the dorsi flexor moment.

For peak knee flexor moment, no difference was found between groups \( (F_{4,19} = 2.31, p=0.095) \). However, the power of the test was low \( (0.557) \) and this may account for the non-significant finding. The peak knee flexor moment increased with walking speed \( (F_{1,19} = 74.37, p=0.000) \) for all subjects. For the peak knee extensor moment, a difference was found between groups \( (F_{4,19} = 24.70, p=0.000) \). The results of the post-hoc test showed there were differences between the above-knee amputee intact and prosthetic limbs \( (p=0.034) \) and between able-bodied subjects and the above-knee amputee intact limb \( (p=0.001) \), above-knee amputee prosthetic limb \( (p=0.000) \), below-knee amputee intact limb \( (p=0.000) \) and below-knee amputee prosthetic limb \( (p=0.000) \). The peak knee extensor moment increased with walking speed \( (F_{1,19} = 32.82, p=0.000) \) and there was a significant group by speed interaction \( F_{4,19} = 5.94, p=0.003 \), because the increase with walking speed was greater for able-bodied subjects and the intact limb of all amputees compared to their prosthetic limb.
A difference was found between groups for the peak hip flexor moment ($F_{4,19} = 4.81, p=0.008$). From the results of the post-hoc test, the difference was found between able-bodied subjects and the above-knee amputee prosthetic limb ($p=0.019$), the prosthetic limb exhibiting a lower peak hip flexor moment. The peak hip flexor moment increased with walking speed ($F_{1,19} = 519.74, p=0.000$). There was no significant group by speed interaction. For the peak hip extensor moment, there was no significant difference between groups, but the peak moment was found to increase with walking speed ($F_{1,19} = 142.87, p=0.000$). No significant interactions were found.

**Joint moment asymmetry.**

From the results of the two-way analysis of variance and post-hoc test on the joint moment asymmetry data, no effect of walking speed was found for ankle plantar flexor or dorsiflexor moment asymmetry. For ankle plantar flexor moment asymmetry, differences were found between groups ($F_{2,13} = 19.14, p=0.000$) with both the above-knee amputees ($p=0.007$) and below-knee amputees ($p=0.000$) being more asymmetrical than able-bodied subjects. The same was found for ankle dorsiflexor moment asymmetry. Differences between the groups ($F_{2,13} = 6.77, p=0.010$) were found with both the above-knee ($p=0.037$) and below-knee amputees ($p=0.019$) being more asymmetrical than able-bodied subjects.

For knee joint moment asymmetry, no differences between groups or effect of walking speed on knee flexor moment asymmetry were found. For knee extensor moment asymmetry, no effect of walking speed was evident but there was a difference between groups ($F_{2,13} = 23.11, p=0.000$). Above-knee amputees were found to be more asymmetrical than below-knee amputees ($p=0.007$) and able-bodied subjects ($p=0.000$).
For hip moment asymmetry, no differences were found between groups for the hip flexor moment, nor was hip flexor moment asymmetry found to increase with walking speed \((F_{1,13} = 3.87, p=0.071)\). However, the power of the test was low (0.45) and this could possibly account for a non-significant finding. No effect of group or speed was found for hip extensor moment asymmetry.

![Graphs showing asymmetry of kinetic variables](image)

**Figure 6.18**—Mean asymmetry of kinetic variables for a) above-knee and b) below-knee amputees during walking at different speeds.

**Joint power output.**

**Able-bodied subjects.**

Figures 6.19 to 6.21 show examples of the average joint power output curves for one able-bodied subject.

For able-bodied joint power output (Figure 6.22), peak power generation and absorption for the hip, knee and ankle were found to increase with increasing walking speed.
Figure 6.19 – an example of the average ankle power output curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).

Figure 6.20 – an example of the average knee power output curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).

Figure 6.21 – an example of the average hip power output curves for an able-bodied subject walking at 1.2 m.s\(^{-1}\).
Figure 6.22 – Mean peak power output of able-bodied subjects walking at different speeds.

Black line indicates dominant limb, red non-dominant limb.
Table 6.9 - Mean peak joint power output (W.kg⁻¹) from both limbs of able-bodied subjects walking at different speeds. Gen indicates power generated, Abs indicates power absorbed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Joint Power Output (W/kg)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ankle Gen</td>
<td>Ankle Abs</td>
</tr>
<tr>
<td>0.5</td>
<td>0.99</td>
<td>0.63</td>
</tr>
<tr>
<td>0.9</td>
<td>1.55</td>
<td>0.87</td>
</tr>
<tr>
<td>1.2</td>
<td>2.03</td>
<td>1.20</td>
</tr>
<tr>
<td>max</td>
<td>3.46</td>
<td>1.22</td>
</tr>
</tbody>
</table>

Joint power asymmetry.

For asymmetry, there appeared to be no effect of walking speed on able-bodied joint power output (Figure 6.23). Peak power output asymmetry was below 10.5% for all able-bodied subjects (Table 6.10).

![Figure 6.23](image)

Figure 6.23 – Mean peak power output asymmetry for able-bodied subjects walking at different speeds.
Table 6.10 – Mean joint power output asymmetry (%) for able-bodied subjects walking at different speeds. Gen indicates power generated, Abs indicates power absorbed.

<table>
<thead>
<tr>
<th>Walking speed (m.s⁻¹)</th>
<th>Ankle</th>
<th>ASI (%)</th>
<th>Knee</th>
<th>ASI (%)</th>
<th>Hip</th>
<th>ASI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gen</td>
<td>Abs</td>
<td>Gen</td>
<td>Abs</td>
<td>Gen</td>
<td>Abs</td>
</tr>
<tr>
<td>0.5</td>
<td>6.49</td>
<td>6.80</td>
<td>8.57</td>
<td>8.33</td>
<td>4.17</td>
<td>15.5</td>
</tr>
<tr>
<td>0.9</td>
<td>1.41</td>
<td>8.67</td>
<td>0.98</td>
<td>7.02</td>
<td>1.19</td>
<td>15.2</td>
</tr>
<tr>
<td>1.2</td>
<td>9.49</td>
<td>3.98</td>
<td>2.47</td>
<td>4.15</td>
<td>9.78</td>
<td>3.21</td>
</tr>
<tr>
<td>max</td>
<td>4.15</td>
<td>6.24</td>
<td>3.40</td>
<td>6.01</td>
<td>5.75</td>
<td>0.93</td>
</tr>
</tbody>
</table>

Amputees.

Joint power output.

Figures 6.24 to 6.26 show an example of the net joint moment traces for an above-knee and a below-knee amputee.

Figure 6.24 – an example of the ankle power output curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s⁻¹.
Figure 6.25 – an example of the knee power output curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s⁻¹.

Figure 6.26 – an example of the hip power output curves for a) an above-knee amputee and b) a below-knee amputee walking at 1.2 m.s⁻¹.

Figure 6.27 shows mean peak power output for amputee and able-bodied subjects walking at different speeds. For the amputees’ intact limb, it appears that joint power output increases with increasing walking speed. For prosthetic limb ankle and knee power generation, there appeared to be no effect of walking speed.
Figure 6.27 – Mean peak power output for amputee and able-bodied subjects walking at different speeds. Thick black line indicates able-bodied subjects, red line above-knee amputees and green below-knee amputees. Solid line indicates intact limb, dotted line prosthetic limb.
From the results of the two-way analysis of variance and post-hoc test, ankle power generation was found to differ between the groups ($F_{4,19} = 29.34, p=0.000$). From the results of the post-hoc test, differences were found between the intact and prosthetic limbs of the above-knee amputees ($p=0.000$) and below-knee amputees ($p=0.000$), and between able-bodied subjects and the prosthetic limb of above-knee amputees ($p=0.000$) and below-knee amputees ($p=0.000$). Power generation increased with walking speed for able-bodied subjects and the intact limb of all amputees ($F_{1,19} = 112.86, p=0.000$), but seemed not to increase for the prosthetic limb. Thus, there was a significant group by speed interaction ($F_{4,19} = 15.67, p=0.000$) because ankle power generation was more affected by speed on the intact limb than on the prosthetic limb. There was no significant difference between groups for ankle power absorption, but more ankle power was absorbed with increasing walking speed ($F_{1,19} = 40.22, p=0.000$).

Differences in knee power generation were found between groups ($F_{4,19} = 11.61, p=0.000$). Differences were found between the above-knee amputee's intact and prosthetic limbs ($p=0.012$) and between the above-knee ($p=0.000$) and below-knee amputee prosthetic limb ($p=0.000$) and able-bodied subjects. Knee power generation increased with speed ($F_{1,19} = 85.96, p=0.000$), more so for able-bodied subjects and the intact limb of amputees than for the prosthetic limb leading to a significant group by speed interaction ($F_{4,19} = 11.51, p=0.000$). For knee power absorption, a difference was found between groups ($F_{4,19} = 9.51, p=0.000$). The post-hoc analysis showed differences between the above-knee amputee prosthetic and intact limbs ($p=0.028$) and between the above-knee amputee prosthetic limb and able-bodied subjects ($p=0.000$). Knee power absorption increased with walking speed ($F_{1,19} = 73.93, p=0.000$), more so on the intact limb than on the prosthetic limb, leading to a significant group by speed interaction ($F_{4,19} = 3.23, p=0.035$).
For the hip, no difference in peak power generation was found between the groups. Peak hip power generation increased with walking speed ($F_{1,19} = 157.24$, $p=0.000$) and there was a significant interaction of group by speed ($F_{4,19} = 4.70$, $p=0.008$) because speed was seen to affect above-knee amputees more than below-knee amputees. For hip power absorption, there was again no significant difference between the groups, but power absorption increased with increasing walking speed ($F_{1,19} = 62.08$, $p=0.000$). There was no significant group by speed interaction.

**Joint power output asymmetry.**

From the results of the two-way analysis of variance and post-hoc test, a difference was found between groups for ankle power generation asymmetry ($F_{2,13} = 42.84$, $p=0.000$), both above-knee amputees ($p=0.000$) and below-knee amputees ($p=0.000$) being more asymmetrical than able-bodied subjects. There was also an increase in ankle power generation asymmetry with walking speed ($F_{3,39} = 5.78$, $p=0.002$) and a significant group by speed interaction ($F_{6,39} = 3.53$, $p=0.007$). Asymmetry in ankle power generation was more affected by walking speed for the amputees than for able-bodied subjects. There were no significant findings for ankle power absorption asymmetry.

For asymmetry in knee power generation, a difference was found between the groups ($F_{2,13} = 25.02$, $p=0.000$), with both above-knee amputees ($p=0.000$) and below-knee amputees ($p=0.005$) being more asymmetrical than able-bodied subjects. For knee power absorption asymmetry, there was also a difference between groups ($F_{2,13} = 22.22$, $p=0.000$). Above-knee amputees were more asymmetrical than below-knee amputees ($p=0.010$) and able-bodied subjects ($p=0.000$).
For the hip, no differences were seen between groups for hip power generation asymmetry. However, the hip power generation asymmetry was found to be affected by walking speed (F_{1,13} = 5.12, p=0.041). There was no significant group by speed interaction. For hip power absorption, there was a significant difference between groups (F_{2,13} = 6.59, p=0.011). From the results of the post-hoc test, the difference was seen between the above-knee amputees and able-bodied subjects (p=0.008), the above-knee amputees being more asymmetrical.

Figure 6.28 - Asymmetry of joint power output variables for a) above-knee and b) below-knee amputees during walking at different speeds.
6.2.4 Discussion.

Kinematic analysis.

Able-bodied subjects.

The purpose of this study was to investigate the effect of increasing walking speed on kinematic and kinetic gait variables and their asymmetry in unilateral lower limb amputees. For the able-bodied subjects, hip, knee and ankle angles were all comparable with the distinct and recognisable patterns previously reported in the literature (Sutherland et al., 1994). Maximum ankle joint angle was found to increase by 11° with increasing walking speed, agreeing with the results of Nilsson et al. (1985).

Knee joint function both at heel strike and mid-stance was found to be affected by walking speed. This observation is in agreement with Nilsson et al. (1985) and Holden et al. (1997). Knee flexion angle at heel strike increased slightly with walking speed, as did knee flexion at mid-stance, during the 'cushioning' phase. A 10° increase in knee flexion at mid-stance was reported with an increase in walking speed from 0.5 to 1.2 m.s⁻¹. A further increase of 7° was seen at the fastest walking speed. This is comparable with the 10° increase in knee flexion at mid-stance with increasing walking speed up to 1.5 m.s⁻¹ reported by Nilsson et al. (1985). Thus, able-bodied subjects adapt to increasing walking speeds by increasing knee joint flexion.

Hip joint flexion angle increased with walking speed by 7° from the slowest to the fastest speed. This value is slightly lower but comparable to the 11° increase in hip joint angle with walking speed reported by Nilsson et al. (1985). Kinematic asymmetry for able-
bodied subjects was found to be less than 4%, and no effect of speed was seen on able-bodied joint angle asymmetry.

Amputees.

For all the amputees, a greater plantar flexion angle was found for the intact limb compared to both their prosthetic limb and for able-bodied subjects. As an artificial foot deprives the user of the ability to plantarflex and dorsiflex the ankle (Whittle, 1991), the limited ankle ROM on the prosthetic limb is determined by the type of prosthesis worn. All amputee subjects in the present study used the same foot mechanism and showed similar results. As a possible compensation mechanism, the amputees in this study increased ankle angle on their intact limb. This finding has also been reported by Smith (1990). Thus, hypotheses 1 and 2 were accepted.

For the knee joint, at heel strike, flexion was slightly greater on the intact limb compared to prosthetic limb for all amputees but was not significant. This observation is comparable to previous results where Culham et al. (1984) reported 12° of intact limb knee flexion at heel strike for below-knee amputees compared to 4° knee flexion at heel strike on their prosthetic limb. Knee flexion at mid-stance was greater on the intact limb than on the prosthetic limb for the amputees. Knee flexion on the prosthetic limb was limited, particularly for above-knee amputees where the artificial knee joint mechanism is mainly responsible for the amount of knee flexion. Thus, hypothesis 3 was accepted. The limited knee flexion on the residual limb has also been previously reported for below-knee amputees (Smith, 1990). If knee flexion was not reduced during the ‘cushioning’ phase, there would be an active raising of the centre of gravity, dissipating more energy (Breakey,
At toe-off, the prosthetic knee joint in the above-knee amputees was more extended than was their intact limb. This could have been due to compensating for the lack of plantar or dorsiflexion and no 'push off' on the prosthetic foot and also the lack of knee flexion in the prosthetic knee mechanism. This increased residual knee extension at toe-off was also seen for below-knee amputees at the slower walking speeds, but was a non-significant finding. Breakey (1976) also reported this, suggesting that knee flexion at toe-off is reduced in the below-knee amputee residual limb in order to compensate for poor ankle plantar flexion, and thus prevent the centre of mass from lowering excessively.

In the present study, below-knee amputees had a reduced knee flexion angle on their residual limb compared to their intact limb. The same was observed for the hip extension angle. This reduced hip angle on the residual limb may be a result of limited prosthetic ankle movement. The intact limb showed similar hip angles to those of able-bodied subjects. No significant differences were found between the limbs of the above-knee amputees and this could possibly be due to the additional problem of the prosthetic knee joint and thus compensatory mechanisms may occur at both the intact and residual hip in above-knee amputees. Few studies have previously reported hip joint angles in lower limb amputees. Hip extension has been reported to start early in the stance phase and to increase to a greater extension angle before toe-off, whilst hip flexion has been shown to occur late in swing and is very limited for above-knee amputees (Radcliffe, 1994). A loss of muscle action of the hip due to above-knee amputation results in a loss of initiation of hip extension during the stance phase (Radcliffe, 1994). This loss, or the partial loss of the hamstrings also results in an inability to stabilise the pelvis resulting in a tendency to bend the lumbar spine laterally in order to carry the trunk more directly over the prosthesis (Radcliffe, 1994). Whilst trunk movement was not measured in this study, it was noted that
all above-knee amputees bent the trunk laterally over towards the prosthetic limb during prosthetic limb stance. Thus, further analysis is needed to investigate muscle activity in amputee gait, particularly the action of the hip extensors/flexors and abductor muscles.

Only one study has been found reporting the effect of walking speed on spatial variables of amputee gait (Boonstra et al., 1993) although values of joint angles or level of asymmetry were not noted. For the present study, amputee plantar flexion angle increased with walking speed, as was seen for able-bodied subjects. For knee angle at mid-stance, knee flexion angle increased with walking speed and there was a significant interaction of group by speed. The intact knee angle at mid-stance was observed to increase with speed more than the prosthetic knee angle for all amputees. Thus, whilst the intact knee behaves as an able-bodied knee, increasing flexion with walking speed, the prosthetic/residual knee behaves differently. The only other effect of speed was noted for hip flexion which increased with increasing walking speed as with able-bodied subjects. Thus, the intact limb hip of below-knee amputees behaves as with able-bodied subjects.

For the amputees, asymmetry for knee angle at mid-stance increased with walking speed. Whilst intact knee flexion was affected by walking speed, prosthetic knee flexion did not appear to be greatly changed with walking speed. All other kinematic asymmetry variables were not found to be significantly affected by walking speed. Hypothesis 4 was rejected. Knee angle asymmetry at mid-stance increased with walking speed for the amputees.

For amputee joints, problems with the prosthetic ankle and/or knee mechanism lead to problems with the residual knee for below-knee amputees, and residual hip for both
above- and below-knee amputees. Pain in the intact knee has previously been reported in unilateral above- and below-knee amputees (Chapter 5) and the increased knee flexion asymmetry with walking speed may have some relation to this. Further analysis is needed in terms of joint moments and power output to investigate fully the compensatory mechanisms used by lower limb amputees for the partial loss of a limb as indicated in these results.

Kinetic analysis.

Net joint moments.

Able-bodied subjects.

Joint moments in able-bodied subjects have been widely reported in the literature for natural walking cadence. The magnitude of net ankle plantar flexor moments has previously been reported to be between 1 and 1.5 Nm.kg\(^{-1}\) (Winter, 1983; Ounpuu et al., 1988; Kuster et al., 1995; Novacheck, 1995). In the present study, the range of net plantar flexor moments increases from 1.26 Nm.kg\(^{-1}\) at the slowest walking speed to 1.43 Nm.kg\(^{-1}\) at the fastest walking speed, thus falling into the previously reported magnitude range.

For net knee moments, the previously reported extensor moments of 0.3 to 1.2 Nm.kg\(^{-1}\) (Winter, 1983; Ounpuu et al., 1988; Kuster et al., 1995; Novacheck, 1995) for able-bodied walking again is comparable with the present study of 0.32 to 1.01 Nm.kg\(^{-1}\). The net flexor moment of 0.2 Nm.kg\(^{-1}\) (Kuster et al., 1995) is also comparable with the present study for natural cadence walking. For the hip, able-bodied peak flexor moments 0.4 to 1 Nm.kg\(^{-1}\) and extensor moments of 0.5 to 0.6 Nm.kg\(^{-1}\) (Winter, 1983; Ounpuu et al., 1988; Novacheck, 1995) are also comparable with the present study for flexor moments, but slightly lower for extensor moments. In the present study, all joint moment variables
increased with increasing walking speed. Asymmetry was found to be below 10.5% for all able-bodied joint moments. There appeared to be no effect of speed on able-bodied joint moment asymmetry.

Amputees.

Both above- and below-knee amputees exhibited a greater plantar flexor moment on their intact limb compared to their prosthetic limb and to able-bodied subjects. This finding has previously been reported for below-knee amputees (Schneider et al., 1993) and above-knee amputees (Lewallen et al., 1985). Whilst alignment (Yang et al., 1991) and prosthesis type (Schneider et al., 1993) affect the magnitude of joint moments, all amputee subjects in this study wore the same prosthetic foot and exhibited similar results. Whilst limited movement of the prosthetic ankle will limit ankle joint moment magnitude on that limb, the amputees appear to be compensating by increasing plantar flexion angle and net plantar flexion moment in the intact limb.

Peak knee flexor moments were slightly greater for all amputees’ intact than their prosthetic limb and for able-bodied subjects but not significant, whilst the peak knee extensor moment was observed to be reduced for the above-knee amputees’ prosthetic compared to their intact limb and to able-bodied subjects. For the below-knee amputees, a reduced peak knee extensor moment was observed in the prosthetic limb compared to able-bodied subjects. A previous study has reported knee flexor and extensor moments of below-knee amputees of similar magnitude to the present study (Lemaire et al., 1993). Hoy et al. (1982) and Schneider et al. (1993) both reported lower knee flexor and extensor moments on the prosthetic limb of below-knee amputees compared to their intact limb in agreement with the present study. For above-knee amputees, Lewallen et al. (1985) reported a lower
knee flexor moment for the prosthetic limb compared to the intact limb, again in agreement with the present study, but with a greater knee extensor moment on the prosthetic limb. This contradicts the results of the present study, and those of previous studies. The results of the present study show that both above- and below-knee amputees exhibited reduced knee flexor and extensor moments on their prosthetic limb compared to their intact limb. For all amputees, the peak knee flexor moment was greater than that exhibited for able-bodied subjects, but not significantly.

For the hip, no differences were noted between the intact and prosthetic limbs for the peak extensor moment. For the flexor moment, the only difference was found between able-bodied subjects and the above-knee amputee prosthetic limb, the prosthetic limb having a reduced peak hip flexor moment compared to the able-bodied subjects. Past literature has reported high variability in hip moments for below-knee amputees (Winter and Sienko, 1988). Lewallen et al. (1985) reported similar magnitudes of hip flexor moment for both the prosthetic and intact limbs of above-knee amputees, whilst Winter and Sienko (1988) reported large hip flexor and extensor moments on the residual limb of below-knee amputees. Few studies have reported differences in hip moments between the intact and prosthetic limbs of above- or below-knee amputees, or the difference in magnitude to that of able-bodied subjects. Thus, it is difficult to compare the results of this study to previous literature. Also, the low number of subjects used in the present study may have given a non-significant result, or problems with locating the hip joint centre from the greater trochanter marker and errors in segment rotations from 2-D analysis may have lead to errors and thus possible non-significant findings. Thus, hypotheses 9 and 10 were rejected. For the amputees, a greater joint moment was produced on their intact limb compared to prosthetic limb. This would be in agreement with the results of chapter 4 in
that, due to problems associated with the function of the prosthetic limb, the amputee has a lower whole body acceleration during the weight-acceptance and push-off phases of the vGRF, lowering the Fz1 and Fz2 peaks. Thus, if the vGRF is reduced due to the whole body motion whilst stepping onto the prosthetic limb, it would be expected that the net joint moments would be reduced on the prosthetic limb compared to the intact limb. This was seen in the current study. For all amputees, as with able-bodied subjects, the magnitude of all kinetic variables, except ankle dorsiflexor moment, increased with increasing walking speed. For the peak knee extensor moment, there was a significant effect of group by speed as the moments on the intact limb increased by a greater amount than the moments on the prosthetic limb with walking speed.

When looking at asymmetry, no joint moment asymmetries were found to be significantly affected by walking speed. No significant effect of walking speed was seen for knee joint moment asymmetry, but the knee extensor moment was observed to increase asymmetry with walking speed for above-knee amputees. Thus, further investigation may be needed with greater subject numbers. Hypothesis 13 is accepted. Joint moment asymmetry does not increase with increasing walking speed.

This study only investigated sagittal plane joint moments and omitted frontal plane joint moments. Whilst the majority of studies have not reported frontal plane joint moments, particularly for lower limb amputees, it may be advantageous to do so. Whilst it was not possible to report frontal plane joint moments here, it is recommended that this aspect of amputee gait be investigated in the future.
Joint power output.

Able-bodied subjects.

In able-bodied walking, the ankle plantar flexors are the main source of power generation, and knee extensors the main source of power absorption (Winter, 1983). The results of the present study illustrate these statements. The ankle generated the most power at toe-off from 0.99 W.kg\(^{-1}\) at the slowest walking speed to 2.03 W.kg\(^{-1}\) at 1.2 m.s\(^{-1}\); 3.46 W.kg\(^{-1}\) was reported for the fastest walking speed. Previous studies have reported ankle power generation of 1.58 to 2 W.kg\(^{-1}\) at natural walking cadence (Harris et al., 1994; Kuster et al., 1995) for able-bodied subjects. Ankle power absorption of 1 W.kg\(^{-1}\) has been reported for natural cadence walking (Harris et al., 1994) which is also comparable to the present study.

For the knee, power absorption in the present study was reported from 0.53 W.kg\(^{-1}\) at the slowest walking speed to 1.59 W.kg\(^{-1}\) at 1.2 m.s\(^{-1}\). A value of 3.06 W.kg\(^{-1}\) was reported for the maximum walking speed. This figure is comparable with the previous literature of 1.5 W/kg to 7.5 W.kg\(^{-1}\) for able-bodied walking (Harris et al., 1994; McFadyen, 1994; Kuster et al., 1995). Peak knee power generation of 0.5 to 1 W.kg\(^{-1}\) (Harris et al., 1994; McFadyen, 1994) was also found to be comparable with the present study.

Hip power generation (0.34 to 2.07 W.kg\(^{-1}\)) was found to be less than ankle power generation as would be expected. This again was found to be comparable to previous studies reporting 1.2 to 1.8 W.kg\(^{-1}\) (Harris et al., 1994; Kuster et al., 1995) for natural cadence walking. All hip, knee and ankle power generation and absorption values were found to increase with walking speed for able-bodied subjects. Joint power output
asymmetry was found to be below 15.5% for all power output variables. There appeared to be no effect of walking speed on able-bodied power output asymmetry.

Amputees.

Ankle power generation was reduced on all the amputees' prosthetic limb compared to their intact limb and to able-bodied subjects as would be expected due to problems with actively flexing the prosthetic ankle. Thus, hypotheses 5 and 6 were accepted. It has previously been reported that the type of prosthetic foot worn will affect ankle power output (Lemaire et al., 1993). However, all amputees in the present study wore the same type of prosthetic foot mechanism and exhibited similar results. As the major power generating source of the stance phase is lost on the prosthetic limb, it is necessary for the amputee to compensate by another mechanism.

Less knee power was observed to be both generated and absorbed on the above-knee amputees' prosthetic limb compared to their intact limb and to able-bodied subjects. This was also seen for the limbs of below-knee amputees, but was not significant, possibly due to the low numbers in the group. Again, this is as would be expected due to problems with the above-knee amputee prosthetic knee mechanism, and the compensatory mechanism at the residual knee used by the below-knee amputees seen in the kinematic and joint moment results. Thus, hypothesis 7 was accepted and hypothesis 8 was rejected. Whilst these observations were also seen for below-knee amputees, the findings were not significant.

As ankle power generation and knee power generation and absorption were found to be reduced on the prosthetic limb compared to the intact limb, it has previously been suggested that the hip extensors on the residual limb compensate for this by generating a
considerable amount of hip power (Smith, 1990). This greater residual limb hip power was observed in the present study only for hip power absorption, but this was not a significant finding. This could have been due to the small number of subjects used in this study, the error in hip joint centre location and problems with segment rotation as seen in hip joint moment calculations, or that the subjects are highly active, and generating a similar amount of hip power on both limbs. Thus hypotheses 11 and 12 were rejected.

Whilst few studies have investigated the effect of walking speed on amputee joint power output, Schneider et al. (1993) reported that both ankle power absorption and generation increased with walking speed. In the present study, all amputee power output variables increased with walking speed as with able-bodied subjects. For ankle power generation, knee power generation, knee power absorption and hip power generation, there was a significant group by speed interaction. For the ankle and knee power output variables, joint power increased by a greater amount on the amputees’ intact limb compared to their prosthetic limb. For the hip, the opposite was true. The increase in knee and ankle power output may be limited by the prosthetic ankle and knee joint mechanisms for the amputees. Thus, it is the problems associated with amputee gait i.e. the limitations of the prosthesis and remaining residual limb musculature that contribute to amputee gait asymmetry. For the hip joint, as compensatory mechanisms were found at the hip for both the below- and above-knee amputees (but not significantly), the increase in power output on the residual hip may need to be greater than for the intact limb hip at the faster walking speeds as the prosthetic ankle and knee power becomes limited. This could be one explanation for the greater increase in hip power generation on the prosthetic limb compared to the intact limb at the faster walking speeds.
From the significant group by speed interaction for joint power output, it has been observed that there is an increase in asymmetry for some of the joint power output variables. On looking at power output asymmetry for the amputees, ankle power generation and hip power generation asymmetry increased with walking speed. Asymmetry was not significantly affected for the other power output variables. Thus again, asymmetry at the hip was found to increase with increasing walking speed. Whilst it would be expected that ankle power generation asymmetry would increase with walking speed due to the prosthetic limb not being able to increase ankle power output with walking speed, compensation was seen with hip power asymmetry increasing. Hypothesis 14 was rejected. Asymmetry in ankle power generation and in hip power absorption increased with walking speed. Thus it appears that for both above- and below-knee amputees, there is some compensation for the functional loss of one or more joints at the hip.

6.3 Summary.

This study aimed to investigate the effect of walking speed on kinematic and kinetic gait variables and their asymmetry in amputee gait. The results showed that for able-bodied subjects, the joint angles, joint moments and power output values contained were comparable with the literature. Whilst all variables increased in magnitude with walking speed, there was little asymmetry between the limbs of the able-bodied subjects, and there was no effect of walking speed on able-bodied kinematic or kinetic asymmetry.

The amputees exhibited a greater plantar flexion angle on their intact limb and a greater plantar flexor moment and power generation compared to their prosthetic limb. This was deemed a possible compensatory mechanism for overcoming problems of flexing the prosthetic ankle. Knee flexion angle at mid-stance was also observed to be greater on the
intact limb compared to the prosthetic limb. This was thought to be due to problems with the prosthetic knee mechanism for the above-knee amputees and compensation for the residual knee in below-knee amputees due to problems with the prosthetic ankle joint. For amputees, active raising of the centre of gravity needs to be limited in order to avoid increased energy cost. Reduced knee flexor and extensor moments and reduced knee power absorption and generation were also found for the prosthetic limb of all amputees compared to their intact limb. Hip flexor moments were found to be greater for able-bodied subjects compared to the above-knee amputee prosthetic limb. No significant difference was found for hip extensor moments. Greater hip power was absorbed on the prosthetic limb compared to the intact limb, possibly to compensate for the limitations of the prosthetic knee and/or ankle, but this was not a significant finding. However, a small number of subjects took part in the study and if further analysis is performed in this area, it is recommended that a greater number of subjects are included, a 3-D analysis be performed and greater accuracy in hip joint centre location is undertaken.

The magnitude of all kinematic and kinetic variables was found to increase with walking speed for the amputees, but walking speed had a greater effect on the intact limb than prosthetic limb for some variables. For the kinematic variables, knee angle at midstance increased its asymmetry with walking speed. For kinetic variables, hip power generation asymmetry was found to increase with walking speed. Other asymmetry variables were not found to be significantly affected by walking speed. Hypotheses 1,2,3,5,6,7 and 13 were accepted and hypotheses 4,8,9,10,11,12 and 14 were rejected. It appears that increases in asymmetry with walking speed occur mostly at the hip. Further analysis is needed to look at the response of amputee muscle activation patterns in the
intact limb of above- and below-knee amputees, and the residual limb of below-knee amputees to investigate the effect this reported asymmetry further.
7.0 Muscle activation asymmetry during walking for above- and below-knee amputees.
7.0 Muscle activation asymmetry during walking for above- and below-knee amputees.

This chapter is concerned with investigating the effect of increasing walking speed on muscle activation patterns for above- and below-knee amputees. The first study aims to establish both the best normalisation technique to use for EMG data during able-bodied walking, and the amount of variation in EMG activity. The second study aims to determine whether there is asymmetrical muscle activity in the remaining muscles of below-knee amputees, and whether muscle activation patterns in the intact limb of below- and above-knee amputees differ from those of able-bodied subjects. From previous studies it has been found that as the intact limb of unilateral amputees is loaded more than their prosthetic limb and it experiences reduced impact shock, increased joint moments and power output, it would be expected that muscle activity is also increased in that limb.

7.1 Normalisation techniques of the EMG signal.

7.1.1 Introduction.

Muscle activity, as measured by EMG, can vary both within- and between-subjects due to incorrect placement of electrodes, skin preparation, muscle size, level of activity and strength. Thus, there is a need to normalise signals in order to compare results between subjects. Normalisation of EMG signal has varied across studies. Most have normalised to a maximum voluntary contraction (MVC) or to mean or peak signal amplitude. The problems with normalising EMG signals to 100% maximum voluntary isometric contraction are that it is normalising a dynamic movement to a static maximum,
that two subjects with the same dynamic signal could have a different magnitude of EMG once normalised to MVC and that intra-subject variability has been reported to be high when using MVCs (Yang and Winter, 1984).

Intra-subject variability has been found to reduce if the EMG signal obtained is normalised to peak or mean amplitude measured during that activity (Yang and Winter, 1984). The coefficient of variation was reported to be lowest using this normalisation technique compared to others. Therefore, it is necessary to determine which is the best normalisation technique to use to reduce variability in EMG. The purpose of this study was to determine a normalising technique that yields a low intra-subject variation, and to establish whether the methodology and electrode placement used is repeatable.
7.1.2. Methodology.

7.1.2.1 Apparatus.

Surface EMG was recorded for 6 muscles on the right limb of able-bodied subjects using an MP100 (Biopac) EMG system. The MP100 (Biopac) system consisted of an interface unit which attached to a small amplifier, containing four channels, by cable. As 6 muscles were used in this study, two amplifier units containing 8 channels were placed in a pack which was worn around the subject’s waist. Each amplifier channel was attached by leads to surface electrodes, two placed over the visual part of the muscle belly, the other over a bony prominence. Channels from a Kistler force platform (type9281B11) also led into the interface unit where both the EMG and force platform data were sampled together at 960 Hz using Acknowledge (Biopac) software. The Fz data from the force platform were used to establish the presence of heel strike and toe off in the EMG data. The cables connecting the amplifiers to the interface unit were long enough so as not to interfere with the subject’s walking.

7.1.2.2 Subjects and procedure.

Five able-bodied subjects (3 females and 2 males) with mean (± SD) age 27.4 ± 3.97 years, height 1.71 ± 0.08 m and mass 67.5 ± 12.5 kg took part in the study. The skin was prepared by shaving, lightly abrasing the surface and cleaning with soap and water. Silver-silver chloride electrodes were placed either side of the muscle belly 2 cm apart, and one ground electrode over a bony area for the tibialis anterior, medial gastrocnemius, rectus femoris, vastus lateralis, biceps femoris and tensor fascia latae following the procedure set out by Delagi et al. (1975). Skin resistance was then measured by means of
a voltmeter to ensure resistance was below 5000 Ω. All EMG signals were taken from the right leg. The electrodes were attached to the amplifier unit by leads which were bound to the leg by crepe bandage to reduce noise from cable movement. Each subject was allowed familiarisation with walking speed prior to testing. Walking speed was measured by means of timing lights. Any trial where the walking speed was not within ± 3% of 1.2 m.s\(^{-1}\) was discarded and the trial repeated. Six trials were obtained from the right leg of each subject for each test session. Electrode position was marked to ensure accurate replacement. After each session of testing, MVCs were performed. These were performed for all muscles with the subject seated with the back of the chair placed against a wall and the hip, knee and ankle at 90°. For the rectus femoris (RF) and vastus lateralis (VL), the foot was placed against an immovable object and the subject instructed to kick out maximally, without lifting the foot from the ground, and hold for 3 seconds. For the biceps femoris (BF), the heel was placed against the leg of the chair and the subject was instructed to kick maximally against the leg of the chair without lifting the foot off the ground and hold for 3 seconds. For the tibialis anterior (TA), the subject’s toes were held down and the subject was instructed to pull up their toes maximally without moving their heel and hold for 3 seconds. For the gastrocnemius (GA), the knee was placed under a table top and the subject was instructed to lift up their heel maximally without moving their toes and hold for 3 seconds. For the tensor fascia latae (TFL), the left side of the chair was placed against a wall and the right thigh against an immovable object. The subject was then instructed to push maximally against the immovable object and hold for 3 seconds. For each muscle, the subject performed three maximum isometric contractions each held for 3 seconds. Verbal encouragement was given throughout the contraction.
7.1.2.3 Data analysis.

A fast fourier transfer (FFT) was used to determine the frequency content of the EMG signal. All EMG data (heel strike to toe off) were filtered using a Hanning high pass filter of 10 Hz. The signal was then rectified and smoothed over a window of 30 data points (Figure 7.1). The data (normalised from heel strike to toe off) were then normalised to a) MVC, b) mean peak amplitude and c) ensemble average.

![Fast Fourier Transfer analysis of the EMG signal during walking at 1.2 m.s\(^{-1}\).](Figure 7.1)

For MVC normalisation, the middle 1 s of each analysed MVC (filtered, rectified and smoothed) (most stable part of the signal) was averaged over the three maximum contractions and expressed as 100% (Arsenault et al., 1986). For mean peak amplitude, the peak amplitude of each trial (filtered, rectified and smoothed) was averaged over the 6 trials and taken as 100%. For ensemble average, the mean amplitude of the signal for each trial (filtered, rectified and smoothed) was averaged over the six trials and taken as
100%. The coefficient of variation was calculated within-trial for each normalising technique.
7.1.3 Results.

Figure 7.2 shows an example of the raw EMG data for an able-bodied subject walking.

![Graphs of EMG data for various muscle groups](image)

Figure 7.2 – An example of raw EMG data from one limb of an able-bodied subject walking at 1.2 m.s\(^{-1}\).
Intra-subject variation.

Normalising EMG to either peak or average signal amplitude yielded a lower intra-subject variability compared to normalising to MVC (Table 7.1). The two amplitude normalisation techniques gave similar results for intra-subject variability. However, normalising to ensemble average gave slightly lower coefficient of variation values for most muscles. Thus, all muscle activity in further sections of this study will be normalised to ensemble average.

Table 7.1 – Coefficient of variation (c.v.) of the different normalisation techniques for each muscle.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Coefficient of variation (%)</th>
<th>MVC</th>
<th>Peak amplitude</th>
<th>Ensemble average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tibialis anterior</td>
<td>29.3</td>
<td>30.1</td>
<td>27.5</td>
<td></td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>38.4</td>
<td>32.8</td>
<td>31.1</td>
<td></td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>62.6</td>
<td>29.2</td>
<td>29.8</td>
<td></td>
</tr>
<tr>
<td>Vastus lateralis</td>
<td>45.4</td>
<td>37.8</td>
<td>34.1</td>
<td></td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>41.3</td>
<td>13.1</td>
<td>34.3</td>
<td></td>
</tr>
<tr>
<td>Tensor fascia latae</td>
<td>40.2</td>
<td>31.6</td>
<td>26.7</td>
<td></td>
</tr>
</tbody>
</table>

Figure 7.3 shows the mean ± SD for each of the muscles for one able-bodied subject walking at 1.2m.s⁻¹. The muscle patterns are from heel strike to toe-off and show similar traces to those previously reported in the literature.
Figure 7.3— An example of mean (± SD) muscle activation patterns for an able-bodied subject walking at 1.2 m s⁻¹.
7.1.4 Discussion.

The results showed that for normalising EMG to either peak or average signal amplitude gave a lower intra-subject variability compared to normalising to MVC. Whilst many EMG studies have normalised EMG during walking to maximum isometric voluntary contraction, errors can arise not least that a dynamic activity is being normalised to a static event. Normalising the EMG signal to peak or average signal amplitude yielded similar results for intra-subject variability. However, normalising to ensemble average gives a slightly lower coefficient of variation (cv) for most muscles.

The c.v. values found for the present study are comparable to those reported in the literature, and often slightly lower. Winter and Yack (1987) reported cv for normalising to ensemble average of ± 33% for the tibialis anterior (TA), 33% for gastrocnemius (GA), 51% for the rectus femoris (RF), 46% for vastus lateralis (VL) and 63% for the biceps femoris (BF). Yang and Winter (1984), also normalising to ensemble average reported cv of 32% for TA, 56% for RF, 40% for VL and 56% for BF. These values are comparable to, and slightly higher than those reported for the present study. Thus the methodology used in this study showed that the lowest possible intra-subject variability was reported using the technique of normalising the EMG signal to the ensemble average of six strides.

The intra-subject variability for EMG associated with the selected muscles acting at each of the hip, knee and ankle joints was higher than the variability reported for power output and moments reported for those joints in study 6.1. These, in turn, were more variable than the hip, knee and ankle joint angle variability also reported in that
study. It has previously been suggested that this may be evidence that the CNS may not be controlling each individual muscle as tightly as it is controlling the net position of torques at one or more joints (Winter and Yack, 1987). This has implications for the muscle activation pattern of amputees following postural reorganisation and adaptation to the partial loss of a limb. The muscle activation pattern of amputees will be investigated in study 7.2.

The pattern of each individual muscle measured was similar to those previously reported (Yang and Winter, 1984; Winter and Yack, 1987; Kabada et al., 1989). From this study, it is deemed that electrode placement was satisfactory, and the EMG signal analysed and normalised to ensemble average is a method used to give the lowest intra-subject variability.

7.1.5 Conclusion.

The results from this study showed that the EMG normalisation technique that yielded the lowest intra-subject variability was normalising to ensemble average. The EMG signal patterns for each muscle were comparable with those reported in the literature for able-bodied subjects walking. The cv in this study was comparable to, and often slightly lower than the cv for muscle activation patterns reported in the literature. Thus the present study showed that the method used was repeatable.
7.2 The effect of walking speed on muscle activation patterns in above- and below-knee amputees.

7.2.1 Introduction.

Few studies of amputee gait have used electromyography (EMG), particularly with above-knee amputees. Those that have, have generally inadequately reported electrode placement, walking speed, normalisation of signal and analysis of signal. The problem with electrode placement on the residual limb concerns muscle belly location (Rose and Gamble, 1995) and electrode placement underneath the prosthetic socket (Peeraer et al., 1990). Therefore, due to these problems, it is possible to only study EMG of the intact limb and the intact muscles on the residual limb of unilateral lower limb amputees.

Below-knee amputee EMG studies have looked at the effects of lost ankle function on muscle activity (Breakey, 1976), the effects of different prosthetic feet on EMG (Culhon et al., 1986), and variability of walking speed on EMG (Boonstra et al., 1993) and differences in muscle activity between the residual and intact limb (Batyee and Joseph, 1966; Culham et al., 1986). The latter two studies reported prolonged quadriceps and hamstrings activity in the residual limb of below-knee amputees compared to their intact limb. Few researchers have compared muscle activity in the intact or residual limb to that of able-bodied subjects. Those that have, have reported similar muscle activity in the intact limb to able-bodied subjects, and prolonged quadriceps and hamstrings activity in the residual limb of below-knee amputees compared to able-bodied subjects. In all these studies, walking speeds were not quantified, expressed as natural cadence at 'comfortable' and 'as fast as possible' speeds. No studies have reported the effect of

279
walking speed on muscle activity in amputees. When greater demands such as increased walking speed are placed on the amputee, greater stress is put on their limbs and they may alter their gait mechanics and muscle activity to try and reduce this stress. The mechanism by which they do this is not yet known, but as increased walking speed increases limb loading asymmetry, muscle activation patterns may also change. The purpose of this study was to (i) determine whether the muscle activation patterns in the intact limb of above- and below-knee amputees differs to those of able-bodied subjects, and (ii) whether the muscle activation patterns in the remaining muscles of below-knee amputees differs from the muscle activation patterns in their intact limb.

It is hypothesised that:

**Hypothesis 1:** The muscle activation patterns on the intact limb of above-knee amputees cross-correlate highly with the muscle activation patterns of able-bodied subjects.

**Hypothesis 2:** The muscle activation patterns on the intact limb of below-knee amputees cross-correlate highly with the muscle activation patterns of able-bodied subjects.

**Hypothesis 3:** Below-knee amputee muscle activity in their residual limb do not cross-correlate highly with muscle activation patterns in their intact limb.
7.2.2. Methodology.

7.2.2.1 Apparatus.

The same apparatus was used as described in study 7.1.

7.2.2.2 Subjects and procedure.

Four below-knee amputees (1 female, 3 males) with mean (± SD) age 44.3 ± 17.5 years, mass 82.7 ± 12.5 kg and years as an amputee 14.0 ± 10.1 years and four above-knee amputees (all male) with mean (± SD) age 59.3 ± 16.8 years, mass 85.6 ± 12.6 kg and years as an amputee 21.3 ± 16.0 years took part in the study. All were unilateral, highly active amputees who were involved in either recreational or competitive sport. The able-bodied subjects (n=8, 3 females, 5 males) had a mean (± SD) age of 26.1 ± 3.98 years, and mass 74.4 ± 14.0 kg were also highly active. The skin was prepared by shaving, lightly abrasing the surface and cleaning with soap and water. Silver-silver chloride electrodes were placed either side of the muscle belly 2 cm apart, and one ground electrode over a bony area for the tibialis anterior, medial gastrocnemius, rectus femoris, vastus lateralis, biceps femoris and tensor fascia latae following the procedure set out by Delagi et al. (1975). Skin resistance was then measured by a voltmeter to ensure resistance was below 5000 Ω. The EMG signals were taken from both legs of able-bodied subjects, the intact limb of above-knee amputees and both the intact limb and intact muscles on the residual limb (RF, VL, BF, TFL) for below-knee amputees. The electrodes were attached to the amplifier unit by leads which were bound to the leg by crepe bandage to reduce noise from cable movement. Each subject was allowed familiarisation with the walking speeds prior to testing. Walking speed was measured by
timing lights. Any trial where the walking speed was not within ± 3% of the predetermined speeds of 0.5, 0.9 and 1.2 m.s⁻¹, and maximum, was discarded and the trial repeated. Six trials were obtained from each leg walking at 0.5 m.s⁻¹. Three trials were obtained from each leg of each subject for the other speeds.

7.2.2.3 Data analysis.

A fast fourrier transformation (FFT) was used to determine the frequency content of the EMG signal (see study 7.1). All EMG data (heel strike to toe off) were filtered using a Hanning high pass filter of 10 Hz. The signal was then rectified and smoothed over a window of 30 data points. This enabled the data to be averaged over the first 30 data points, then the second 30 data points etc., smoothing out any unwanted peaks. The data for each muscle (from heel strike to toe off) were then normalised to the ensemble average of the signal (six trials) for walking at 0.5 m.s⁻¹ for each subject. The mean activity of the three trials for each subject and each muscle was calculated in Excel. The amputees were grouped for the above-knee amputee intact limb, below-knee amputee intact limb and below-knee amputee residual limb for each muscle. The group mean was then calculated for further statistical analysis. The amputees were grouped in this study because they all wore the same prosthesis, were all 'established' and deemed 'good to excellent' walkers and only their intact muscles were measured. Thus the group was deemed homogenous. Cross–correlations were performed in order to look at the conformity of the entire mean EMG curve from heel strike to toe-off and not just the peak muscle activity. Cross-correlations were performed for each muscle between a) the mean of able-bodied subjects and the mean of the intact limb for above- and below-knee
amputees and b) the mean of the residual limb muscle and corresponding mean of intact limb muscle for below-knee amputees. Whilst the shapes of the EMG signal are assessed statistically, the peaks and duration of the EMG signal will be assessed qualitatively.
7.2.3 Results.

It was assumed that there was no muscle asymmetry between the limbs of able-bodied subjects. Therefore, the mean was taken for the activity in each muscle between the two limbs. Figure 7.5 shows the effect of speed on muscle activity for able-bodied subjects. For all muscles, walking speed increased the magnitude of muscle activity. At the maximum walking speed, three of the muscles, TA, RF and VL exhibited an extra muscle burst not seen at the slower walking speeds. Figure 7.4 shows an example of raw EMG data for the intact limb of an above-knee amputee.

Figure 7.6 shows muscle activity of the TA and GA for both the intact limb of above- and below-knee amputees, and the residual limb of below-knee amputees. For the amputees, greater TA activity was noted at all walking speeds compared to able-bodied subjects. Muscle activity increased in magnitude with walking speed for all subjects. The extra burst of activity seen for the able-bodied subjects at the maximum walking speed was also observed for the amputees. For the medial gastrocnemius (GA), amputee subjects exhibited lower activity than able-bodied subjects. Muscle activity in the GA was found to increase with walking speed for all subjects.

Figure 7.7 shows muscle activity of the RF and VL for both the intact limb of above- and below-knee amputees, and the residual limb of below-knee amputees. Muscle activity for both muscles was found to be reduced for the amputees compared to able-bodied subjects, and increased in magnitude with increasing walking speed. For the RF, muscle activity for the below-knee amputee residual limb was very limited compared to
the other groups. This difference appeared greater at the maximum walking speed. For the VL, muscle activity for the residual limb in below-knee amputees was also limited compared to the other groups, but increased at maximum walking speed.

Figure 7.4 – an example of raw EMG data from the intact limb of an above-knee amputee walking at 1.2 m.s\(^{-1}\).
Figure 7.5 – Mean EMG muscle activity for able-bodied subjects walking at increasing walking speed.
Figure 7.6 – Mean tibialis anterior and medial gastrocnemius muscle activity for able-bodied subjects, the intact limb of above-knee and below-knee amputees walking at a) 0.5 m.s\(^{-1}\), b) 0.9 m.s\(^{-1}\), c) 1.2 m.s\(^{-1}\) and d) maximum walking speed. Black line indicates able-bodied subjects, red below-knee and green above-knee amputees.
Figure 7.7 – Mean rectus femoris and vastus lateralis muscle activity for able-bodied subjects, the intact limb of above-knee and below-knee amputees walking at a) 0.5 m.s\(^{-1}\), b) 0.9 m.s\(^{-1}\), c) 1.2 m.s\(^{-1}\) and d) maximum walking speed. Black line indicates able-bodied subjects, red below-knee and green above-knee amputees. Solid line indicates intact limb, dotted residual limb.
Figure 7.8 shows muscle activity for able-bodied subjects, the intact limb of above-knee and below-knee amputees walking at a) 0.5 m.s\(^{-1}\), b) 0.9 m.s\(^{-1}\), c) 1.2 m.s\(^{-1}\) and d) maximum walking speed. Black line indicates able-bodied subjects, red below-knee and green above-knee amputees. Solid line indicates intact limb, dotted residual limb.
Figure 7.8 shows muscle activity for the biceps femoris (BF) and tensor fascia latae (TFL). For the BF, there appeared to be little effect of walking speed on muscle activity magnitude. However, at the slower walking speeds, muscle activity was greater for able-bodied subjects. At the faster speeds, amputee muscle activity became greater. At maximum walking speed, an extra burst of muscle activity towards the end of the stance phase was seen for the amputees’ intact limb but not for able-bodied subjects or the residual limb of below-knee amputees. For the TFL, muscle activity was limited for the amputees compared to the able-bodied subjects, particularly for the below-knee amputee residual limb. The difference between the below-knee amputee intact and residual limb was greatest at the maximum walking speed.

On looking at the results of the cross-correlation of muscle activity between able-bodied subjects and the intact limb of above-knee amputees (Table 7.2), the cross-correlations for all muscles except BF were high. The cross-correlations (with the exception of BF) also appeared to increase with walking speed up to 1.2 m.s⁻¹.
Table 7.2 – Cross-correlation for the mean of EMG activity for each muscle between able-bodied subjects and the intact limb of above-knee amputees.

<table>
<thead>
<tr>
<th>Speed (m.s(^{-1}))</th>
<th>TA</th>
<th>GA</th>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>BF</td>
</tr>
<tr>
<td>0.5</td>
<td>0.794</td>
<td>0.712</td>
<td>0.521</td>
</tr>
<tr>
<td>0.9</td>
<td>0.856</td>
<td>0.838</td>
<td>0.650</td>
</tr>
<tr>
<td>1.2</td>
<td>0.910</td>
<td>0.828</td>
<td>0.456</td>
</tr>
<tr>
<td>MAX</td>
<td>0.849</td>
<td>0.836</td>
<td>0.525</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
</tr>
<tr>
<td>0.694</td>
</tr>
<tr>
<td>0.759</td>
</tr>
<tr>
<td>0.896</td>
</tr>
<tr>
<td>0.875</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>TFL</td>
</tr>
<tr>
<td>0.756</td>
</tr>
<tr>
<td>0.740</td>
</tr>
<tr>
<td>0.821</td>
</tr>
<tr>
<td>0.772</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL</td>
</tr>
<tr>
<td>0.832</td>
</tr>
<tr>
<td>0.875</td>
</tr>
<tr>
<td>0.920</td>
</tr>
<tr>
<td>0.870</td>
</tr>
</tbody>
</table>

The results of the cross-correlation for muscles between able-bodied subjects and below-knee amputees’ intact limb (Table 7.3) show high cross-correlations for most muscles (a slightly lower correlation was seen for the BF). Generally, with the exception of the BF, the muscles correlated better at the faster walking speeds. The cross-correlations between the muscles of the residual limb and intact limb for the below-knee amputees (Table 7.4) were generally lower than for between able-bodied subjects and below-knee amputees’ intact limb. At the faster walking speeds, the BF and VL intact limb muscles cross-correlated more highly with the residual limb muscles.
Table 7.3 – Cross-correlation for the mean of EMG activity for each muscle between able-bodied subjects and the intact limb of below-knee amputees.

<table>
<thead>
<tr>
<th>Speed (m/s)</th>
<th>TA</th>
<th>GA</th>
<th>Muscle</th>
<th>BF</th>
<th>RF</th>
<th>TFL</th>
<th>VL</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>0.836</td>
<td>0.687</td>
<td>0.695</td>
<td>0.767</td>
<td>0.543</td>
<td>0.687</td>
<td></td>
</tr>
<tr>
<td>0.9</td>
<td>0.903</td>
<td>0.855</td>
<td>0.781</td>
<td>0.711</td>
<td>0.749</td>
<td>0.693</td>
<td></td>
</tr>
<tr>
<td>1.2</td>
<td>0.960</td>
<td>0.660</td>
<td>0.687</td>
<td>0.872</td>
<td>0.837</td>
<td>0.888</td>
<td></td>
</tr>
<tr>
<td>MAX</td>
<td>0.794</td>
<td>0.946</td>
<td>0.350</td>
<td>0.873</td>
<td>0.897</td>
<td>0.847</td>
<td></td>
</tr>
</tbody>
</table>

Table 7.4 – Cross-correlation for the mean of EMG activity for each muscle between the residual and intact limb of below-knee amputees.

<table>
<thead>
<tr>
<th>Speed (m/s)</th>
<th>BF</th>
<th>RF</th>
<th>TFL</th>
<th>VL</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>0.011</td>
<td>0.203</td>
<td>0.409</td>
<td>0.264</td>
</tr>
<tr>
<td>0.9</td>
<td>0.508</td>
<td>0.491</td>
<td>0.024</td>
<td>0.477</td>
</tr>
<tr>
<td>1.2</td>
<td>0.792</td>
<td>0.843</td>
<td>0.520</td>
<td>0.919</td>
</tr>
<tr>
<td>MAX</td>
<td>0.741</td>
<td>0.845</td>
<td>0.917</td>
<td>0.883</td>
</tr>
</tbody>
</table>
7.2.4 Discussion.

Able-bodied subjects.

This study aimed to determine (i) whether the muscle activation patterns in the intact limb of above- and below-knee amputees differ from those of able-bodied subjects, and (ii) whether the muscle activation patterns in the remaining muscles of below-knee amputees differ from the muscle activation patterns in their intact limb. For the able-bodied subjects, the muscle activity reported in this study is comparable with that from previous literature. As the majority of studies have normalised muscle activity during walking to 100% MVC and this study normalised EMG to ensemble average, it is difficult to compare the magnitude of the results of this study to the previous literature. However, the timing of muscle activity can be compared. Ericson et al. (1986) reported that the tibialis anterior (TA) exhibited a peak burst of activity at heel strike. The peak activity of the gastrocnemius (GA) was reached at 40-45% of the gait cycle, the beginning of the push-off phase, thus comparable with the present study. The biceps femoris (BF) was reported to reach peak activity at 5% of the gait cycle, whilst the rectus femoris (RF) and vastus lateralis (VL) reached peaks at 10% of the gait cycle (Ericson et al., 1986). This is also comparable to the present study. The tensor fascia latae (TFL) has previously been reported to be consistently active in the first half of stance, as was seen in the present study, counteracting the external adducting moment and absorbing energy (Cappozzo et al., 1976).

Few studies have reported the effect of walking speed on muscle activity. Those that have, have reported increases in both magnitude and duration. In the present study,
the TA was monophasic at the slower walking speeds and became biphasic at the maximum walking speed. The initial peak was also seen to increase in duration. For the GA, the muscle was seen to switch on sooner in the stance phase and was on for longer. Both muscles increased in magnitude of activity with walking speed. Shiavi et al. (1981) reported that the TA was monophasic at slow walking speed and multiphasic at fast speed. The GA became active earlier in the stance phase as walking speed increased, thus agreeing with the present study.

For the hamstrings and quadriceps in the present study, muscle activity was found to increase with walking speed and the muscles were activated for longer. The RF, VL and TFL became biphasic at the maximum walking speed. Lyons et al. (1983), using fine wire electrodes, reported an increase in BF magnitude. The TFL was biphasic at fast walking and monophasic at natural cadence walking. All other muscles increased their duration of activity and were activated sooner in the gait cycle during faster walking, thus agreeing with the present study.

Amputees.

Different prostheses have been found to affect muscle activity in the residual limb of unilateral amputees (Culhon et al., 1986). However, all amputee subjects in this study wore the same foot and foot and knee mechanism and thus exhibited similar results. On comparing the intact limb of above- and below-knee amputees to able-bodied subjects, the greatest burst of activity for the TA was seen for below-knee amputees. This was observed at all walking speeds. Above-knee amputees also exhibited greater activity than
that of able-bodied subjects. This would correspond to the increased kinematic and kinetic output observed on the intact limb of above- and below-knee amputees (chapter 6). If the intact limb ankle increases ROM/ power output in order to compensate for the loss of a fully functional ankle joint on the corresponding limb, it would be expected that the activity of muscles around the ankle is also increased. The TA muscle activation pattern was observed to be monophasic at the slower walking speeds but became biphasic at the maximum speed for all subjects.

The TA was found to be activated for longer, and the GA was seen to switch on sooner in stance for both the above- and below-knee amputees compared to able-bodied subjects. The duration of muscle activity was observed to increase slightly with walking speed. No study has been found reporting muscle activity for the TA or GA in the intact limb of above- or below-knee amputees. For the GA, muscle activity was observed to increase with walking speed. Activity was seen to be slightly greater for the able-bodied subjects than the amputees. Whilst there were some slight differences noted between the groups for TA and GA muscle activity, both muscles correlated highly between the above- and below-knee amputees’ intact limb and the limbs of able-bodied subjects.

For the quadriceps, RF and VL, muscle activity was lower on the intact limb of both above- and below-knee amputees, and very small for the below-knee amputee residual limb. Muscle activity was observed to increase with walking speed, but the difference between the below-knee amputee residual and intact limbs was increased in magnitude at the faster walking speed. The extra burst of activity seen in able-bodied
subjects for the RF at the maximum walking speed was absent in the amputees. This could be due to either the amputees not achieving a high enough walking speed, or it is not a characteristic of muscle activity in amputees.

Muscle activity in the amputees' residual limb has previously been found to be of longer duration than that of able-bodied subjects (Erberhart et al., 1947; 1954; Batyee and Joseph, 1966; Sutherland and Hagy, 1972). It has been suggested that this is probably a result of the damping effect of the quadriceps on the heel rise of the prosthesis to counteract the increased activity of the hip flexors, and also accelerate the leg in swing after maximum knee flexion.

For the intact limb, muscle activity of below-knee amputees has been reported to be similar to that of able-bodied subjects (Batyee and Joseph, 1966). Thus, from past studies, quadriceps activity in the intact limb of below-knee amputees is close in similarity to able-bodied subjects than to the residual limb. The present study also showed this resemblance as, particularly at the slower walking speeds, the RF and VL on the intact limb gave a higher cross-correlation with able-bodied subjects compared to the residual limb. However, Bach (1999) reported the opposite findings. The average cross-correlation between the intact and residual limb was higher than the average cross-correlation between the intact limb and able-bodied subjects for below-knee amputees. These results not only disagree with the present study, but also with previous studies reporting muscle activity in below-knee amputee gait.
For the hamstrings, the BF activity appeared to be greater for able-bodied subjects at the slower walking speeds, but the amputees' intact limb muscle activity became greatest at the faster speeds. At the maximum walking speed, a second peak of BF activity was evident at the start of the push-off phase for the intact limb of above- and below-knee amputees, but not for able-bodied subjects or the below-knee amputee residual limb.

At the slower walking speeds, the intact limb for below-knee amputees correlated more highly with able-bodied subjects than with the residual limb, but at faster speeds, the opposite was true. Previous studies have reported differences in hamstring activity between the residual and intact limbs of below-knee amputees (Batyee and Joseph, 1966; Culhon et al., 1986). Hamstring muscle activity in the intact limb was found to be comparable with that of able-bodied subjects (Milner et al., 1971) thus agreeing with the results of the present study.

The TFL muscle activity was greater for able-bodied subjects compared to the intact limb of above- and below-knee amputees. Muscle activity on the below-knee amputee residual limb was observed to be small. The difference between the below-knee intact and residual limbs was found to be greatest at the maximum walking speed. Cross-correlation between the below-knee intact limb and able-bodied subjects was greater than between the intact and residual limbs for all except the maximum walking speed. Thus, the TFL in the intact limb behaves as with able-bodied subjects, whilst the TFL on the residual limb exhibits a slightly different pattern possibly due to compensation for
problems associated with the prosthesis. No studies have been found investigating TFL muscle activity in below- or above-knee amputees.

From analysis of the muscle activity in the residual and intact limbs of below-knee amputees, it has been found that muscle activity in the residual limb differs from muscle activity in the intact limb in terms of the quadriceps, hamstrings and hip abductor activity (low cross-correlations). Thus, hypothesis 3 is accepted. From analysis of muscle activity in the above- and below-knee amputee intact limb, activation patterns have been found to cross-correlate highly with those of able-bodied subjects. Thus, hypotheses 1 and 2 are accepted. Whilst it may have been expected that muscle activity in the intact limb of above- and below-knee amputees would differ from that of able-bodied subjects due to compensatory mechanisms for the partial loss of a lower limb, it was not found in this study, or a few previous studies.

For below-knee amputees, it has been shown that there is some muscle asymmetry between muscles in the intact and residual limbs. If the vGRF is lower on the prosthetic limb than intact limb due to a reduced acceleration of the whole body when stepping onto the prosthetic limb and at push-off (Chapter 4), and that the net joint moment and power output is also reduced for the prosthetic limb (Chapter 6) due to this reduced vGRF, then it would be expected that there is reduced muscle activity in the residual limb compared to the intact limb. This was seen in the current study for below-knee amputees. The reduced vGRF and joint moments on the prosthetic limb due to the amputees’ gait requires less muscle activity than for the intact limb. This has implications for the design
of the prosthetic limb as, due to the function, or lack of function of the limb, the prosthetic limb is seen to cause asymmetries in amputee gait and could possibly be responsible for the increased loading on the intact limb.

Whilst it has been shown that there is some asymmetry between muscles in the intact and residual limbs, i.e. low cross-correlations, the cross-correlations between the two limbs became stronger at the fastest walking speed for all the muscles. This could be due to the able-bodied subjects walking faster than the amputees at the maximum walking speed, and thus may not be comparable. Amputees may adapt to walking at a faster speed by altering the muscle activation pattern on their residual limb, exhibiting a more symmetrical pattern, possibly so that they are able to reduce temporal asymmetry (Chapter 4). Whilst this is only speculated in this study, further research is needed to investigate more fully and asymmetry in muscle activation patterns during walking for below-knee amputees.

7.3 Summary.

This study aimed to investigate muscle activity in below-knee amputees, and whether the muscle activity patterns of the above- and below-knee amputees’ intact limb differ from those of able-bodied subjects. For the able-bodied subjects, muscle activity in the present study was comparable with that of previous studies. Able-bodied muscle activity was found to increase with increasing walking speed, and TA, RF and VL muscles became biphasic at the maximum walking speed.
For the amputees, no previous study has been found reporting TA and GA activity in the intact limb of above- and below-knee amputees. Muscle activity in the TA of the intact limb was greater for the amputees during the gait cycle compared to able-bodied subjects. This finding is comparable to the previous results obtained for kinematic and kinetic variables (chapter 6) and vGRFs (chapter 4) as the amputees were compensating for the functional loss of the ankle on the contralateral limb. Muscle activity in both the TA and GA increased with increasing walking speed. Both muscles correlated highly with the TA and GA muscle activity for able-bodied subjects.

For the quadriceps, muscle activity in the intact limb of the amputees was observed to be slightly less than for able-bodied subjects. Residual limb muscle activity in below-knee amputees was seen to be limited. At the maximum walking speed, whilst able-bodied RF muscle activity became biphasic, this was not seen for the amputees. For both the RF and VL, muscle activity in the intact limb of below-knee amputees gave a higher correlation with able-bodied subjects than with the below-knee amputee residual limb. The above-knee amputees' muscle activity in the intact limb cross-correlated highly with able-bodied subjects. This thus showed that muscle activity differed between the limbs of below-knee amputees, whilst muscle activity in the intact limb was similar to that of able-bodied subjects.

For the hamstrings, muscle activity for the amputees was reduced compared to that of able-bodied subjects. For the BF at the maximum walking speed, a second peak burst of activity in the intact limb of above- and below-knee amputees was seen, but not in the
residual limb of below-knee amputees. This finding again illustrates a difference between
the limbs of below-knee amputees which was reiterated in the results of the cross-
correlations. Again, muscle activity in the intact limb of below-knee amputees cross-
correlated more highly with able-bodied subjects than with their residual limb.

For the TFL, muscle activity was observed to be greater for able-bodied subjects
than in the intact limb of above- and below-knee amputees whilst muscle activity in the
below-knee residual limb was limited. As with the quadriceps and hamstrings, the muscle
activation pattern on the below-knee amputee intact limb cross-correlated more highly
with the able-bodied limb than with the pattern in the residual limb of the below-knee
amputees. The above-knee amputee intact limb also cross-correlated highly with the able-
bodied limb.

Thus, this study has shown that the muscle activation patterns on the residual limb
of below-knee amputees differ from their intact limb. Also, the muscle activation patterns
on the above- and below-knee intact limb correlated highly with those of able-bodied
subjects. Hypotheses 1, 2 and 3 were accepted.
8.0 Concluding remarks.
8.0 Concluding remarks.

This thesis investigated amputee gait variables and their asymmetry, but manipulated walking speed in order to assess how the amputee adapts and copes with the faster walking speeds. Increased asymmetry may cause further problems in amputee gait, possibly leading to increased pain or discomfort on the intact limb. No study found has previously addressed the effect that greater demands, such as increasing walking speed, have on amputee gait asymmetry. By increasing walking speeds, it was suggested that amputees will demonstrate how they cope with these demands either by reducing gait asymmetry or exhibiting increased gait asymmetry.

All amputees were found to exhibit aspects of gait similar to that previously reported in the literature. They had a reduced vGRF, net joint moments, power outputs and reduced EMG activity in their residual/prosthetic limb compared to their intact limb. This is partly explained by the limitations of the prosthetic limb. After heel strike, the body moves over the stance limb during ‘weight-acceptance’. In normal walking, the body is initially accelerating, producing a peak vGRF of slightly more than one body weight. Due to limitations of the prosthetic limb, in that there is a lack of a fully functional ankle or ankle and knee joint, there is a reduced movement speed of the body over the prosthetic limb. From the results of Chapter 4, the amputees are spending longer stepping onto their prosthetic limb than intact limb, thus they are not able to move the body over the prosthetic limb as quickly as when they step onto their intact limb. They also have a longer swing phase and spend less time in stance on their prosthetic limb as though they do not ‘trust’ the limb, or as if there is discomfort or pain. Any problems with the fit of the moulded plastic socket such as being too tight over a bony prominence, or being too slack causing friction and abrasions, will influence the
gait cycle, causing more asymmetry because of discomfort or pain. None of the amputees in the studies in this thesis reported such problems at the time of testing.

The problems associated with a non-fully functioning prosthetic limb may be responsible for the body movements of the amputee, resulting in a reduced vGRF Fz1 peak. Other reasons may include the prosthesis being lighter than the intact limb and the amputees' centre of mass being closer to their intact limb than would otherwise be the case. As the vGRF is reduced on the prosthetic limb compared to the intact limb, and increased problems with a lack of full ROM from the prosthetic ankle and/or knee joints, the net joint moments and power outputs are also reduced on the prosthetic limb compared to the intact limb.

For the below-knee amputees, it was seen that muscle activity on their residual limb was reduced compared to their intact limb. From the cross-correlation results (Chapter 7), the below-knee amputees' intact limb was seen to function more like an able-bodied subject's limb than like their residual limb. Thus, residual limb muscle activity in below-knee amputees may differ to that of their intact limb due to compensation by the amputee for the limitations of their prosthesis. Due to the reduced vGRF, spending longer stepping onto the prosthetic limb, and the reduced net joint moments and power output on the prosthetic limb, the below-knee amputee exhibits a reduced magnitude but prolonged duration of muscle activity. The prolonged residual limb muscle activity may be due to the longer step time during stance.

Many of these amputee gait asymmetries have previously been reported. What has not been previously been investigated, however, is the effect of increasing walking speed on amputee gait asymmetry. With increasing walking speed, vGRF increased on
both the intact and prosthetic limbs as, with increasing speed, the body will increase acceleration as it moves over the stance limb, increasing the Fz1 peak. However, vGRF increased more on the intact limb than prosthetic limb, increasing asymmetry. This was also seen for the joint angles and net joint moments and power outputs. Knee angle at midstance, peak knee extensor moment, ankle power generation, knee power absorption and generation, and hip power generation were all found to increase on the intact limb more than on the prosthetic limb with increasing walking speed, thus increasing asymmetry. However, possibly due to the low subject numbers in the studies, not all these variables showed a significant effect of walking speed on asymmetry.

The amputees did reduce temporal asymmetry with increasing walking speed. This may have been due to the amputees attempting to reduce some gait asymmetry in order to achieve the maximum walking speed. If stance and swing time asymmetry was not reduced, the amputees may not have been able to increase their walking speed. This reduction in temporal asymmetry with increasing walking speed may reflect the need for the amputee to balance and achieve dynamic co-ordination to allow the faster walking speeds to be achieved. This could be being achieved at the expense of increasing the load on the intact limb and therefore increasing loading asymmetry with walking speed. Therefore, it may be that as the intact limb joints degenerate, amputees begin to naturally walk less fast in order to keep the load off the intact limb. Thus, the amputee gets less exercise and this could reduce their mobility and possibly quality of life.

Compensation for the limitations of the prosthesis have been seen on the intact limb. Greater time spent on the intact limb than prosthetic limb and greater intact limb vGRF loading compared to able-bodied subjects have been reported in this thesis. Pain and/or joint degeneration in the intact limb is a common problem for unilateral lower
limb amputees. It has previously been suggested that joint pain and/or joint
degeneration could be due to repeated cyclic loading. If the increased intact limb loading
with walking speed reported in this thesis is representative of all unilateral amputees,
then this may explain the high incidence of intact limb joint pain and joint degeneration
reported in unilateral amputees. The results of the study investigating impact shock and
impact shock attenuation showed that for all amputees reporting intact limb joint pain,
shock attenuation was reduced through that limb compared to their prosthetic limb.
Thus, the damaged joint, possibly as a result of increased cyclic loading, has a reduced
capacity to attenuate impact shocks. In order to try and protect the intact limb from
further damage, and to prevent higher than desirable impact shocks from reaching the
head, incoming impact shocks were reduced on the affected limb. The only amputee not
reporting intact limb pain exhibited the opposite results. Greater incoming impact
shocks were seen on his intact limb compared to his prosthetic limb. It may be that new
amputees not only load their intact limb more than their prosthetic limb during walking
(possibly due to mass and COP distribution of the prosthetic limb), but also impact the
ground harder too. Thus, it may be a combination of these two mechanisms that are
responsible for the intact limb pain/joint degeneration in unilateral amputees. Once
joint degeneration is present, the amputees may alter their gait patterns in order to
compensate for this. As with limb loading, incoming impact shocks were seen to
increase with increasing walking speed for both limbs of all amputees and able-bodied
subjects. Shock attenuation was seen to increase with walking speed for all able-bodied
subjects and for the intact limb of all amputees. For some of the amputees, shock
attenuation through the prosthetic limb stayed consistent with walking speed indicating
that the amount of shock attenuated was mainly dependent on the material used in the
prosthesis. Generally, shock attenuation asymmetry decreased with walking speed, and
impact shock asymmetry in the axial direction increased with increasing walking speed.
Thus again, for those amputees suffering from intact limb joint pain/joint degeneration, walking at the faster walking speeds could possibly increase the damage already present.

It was previously stated that it was not known whether these asymmetries contribute to problems experienced in amputee gait or vice versa (Dingwell et al., 1994). It may be suggested that from the results of this thesis, problems with the prosthesis are contributing to amputee gait asymmetry. Due to the limited ankle or ankle and knee movement, the amputees are spending longer stepping onto their prosthetic limb and are unable to accelerate the body during the weight-acceptance phase as much as they are when stepping onto their intact limb. Consequently, net joint moments and joint power output and thus muscle activity are reduced on their residual/prosthetic limb compared to their intact limb. In compensation, greater demands are placed on their intact limb. As there is a limit to the function of the prosthesis and no limit on their intact limb, the gait variables studied were found to increase asymmetry with increasing walking speed. The amputees were seen to reduce temporal gait asymmetry, possibly in an attempt to reduce some part of gait asymmetry, achieve balance and co-ordination and attain the faster walking speeds. This has implications for prosthesis design. Previously, the inertial properties of the prosthesis have been manipulated in order to try and reduce amputee gait asymmetry. However, as the amputees are more able to reduce swing phase asymmetry (which would be most affected by manipulating the inertial properties of the prosthesis) with walking speed than any other variable, other areas of prosthesis design should be investigated. If it was possible to design a prosthesis where the knee and ankle joint angles could increase with walking speed as with their intact limb, and the prosthesis was weighted so that the step time was more symmetrical, amputee gait asymmetry may reduce. However, there are also other issues to consider. A change in design could increase energy cost of walking for the amputees, or discomfort. The
results of this thesis have shown the adaptations to the partial loss of a lower limb in above-knee and below-knee amputees during walking with increasing speed. However, there are limitations to this thesis and more research is needed in this area to provide greater information for prosthesis design and possible improvements in prosthetic limbs and thus amputee gait.
9.0 Recommendations for future study.

Amputee gait asymmetry has previously been investigated, but few studies have looked at either the role of the intact limb and how it differs to able-bodied subjects, or the effect of walking speed on gait asymmetry. This study has indicated possible compensatory mechanisms used by above- and below-knee amputees for the partial loss of a lower limb, and described the effect of increased walking speed on amputee gait asymmetry. Further work is required to establish the ideas suggested here.

It has been well documented that many unilateral amputees, whether above- or below-knee, suffer from intact knee joint pain or joint degeneration. However, two previous studies have given conflicting reasons as to why this occurs. From the results of this study, it is suggested that new amputees who do not suffer joint pain, load and impact the intact limb at heel strike excessively, thus increasing the risk of joint degeneration in later life. Established amputees who suffer from intact knee pain/joint degeneration exhibit a reduced shock attenuation capacity and thus reduce incoming impact shocks in order to try to protect that limb. Whilst this theory is suggested here, further work needs to be done to establish whether this is the mechanism that occurs. It may be that a longitudinal study could be performed to monitor changes in impact shock and shock attenuation in newly established amputees in order to assess any degenerative joint changes over time.

Sagittal plane joint moments and power output have been reported in this study. It has been found that lower limb amputees exhibit kinematic and kinetic asymmetry at the hip, knee and ankle. However, due to the nature of amputee gait it is suggested that frontal plane kinetics also be investigated in future studies.
Muscle activation patterns in the intact limb of above- and below-knee amputees have been found to exhibit patterns similar to those of able-bodied subjects. Whilst muscle activity in the residual limb of below-knee amputees has been investigated in this study, and thus muscle activation pattern asymmetry established, little is known about above-knee amputee muscle activation asymmetry. Studies investigating muscle activation pattern asymmetry in above-knee amputees may give further information as to the effect of the additional loss of a knee joint.

The effect of increasing walking speed on amputee gait asymmetry has been investigated in this study, but the results are variable. Whilst the asymmetry of some of the gait variables have been found to increase with walking speed, some have reduced and others have appeared not to be affected by walking speed. The low numbers of amputee subjects in these studies may have been responsible for some of the non-significant findings. Thus, further investigation with a greater number of subjects may lead to more conclusive findings.
References.
References.


Batyee C.K. and Joseph J. (1966) Investigation by telemetering of activity of some muscles in walking. *Medical and Biological Engineering*, 4, 125-1132


Burke M.J., Roman V. and Wright V. (1978) Bone and joint changes in lower limb amputees. *Annals of Rheumatic Disease*, 37, 252-254


Stefanyshyn D., Engsberg J., Tedford K. and Harder J. (1994) A pilot study to test the influence of specific prosthetic features in preventing trans-tibial amputees from walking like able-bodied subjects. *Prosthetics and Orthotics International*. 18, 180-190


Appendix.
Appendix 1

Table 1 - Able-bodied subject details for study 4.1.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (y)</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
<th>Male/female</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>30</td>
<td>1.84</td>
<td>68</td>
<td>M</td>
</tr>
<tr>
<td>B</td>
<td>34</td>
<td>1.69</td>
<td>78</td>
<td>M</td>
</tr>
<tr>
<td>C</td>
<td>26</td>
<td>1.78</td>
<td>78</td>
<td>M</td>
</tr>
<tr>
<td>D</td>
<td>48</td>
<td>1.82</td>
<td>84</td>
<td>M</td>
</tr>
</tbody>
</table>

Table 2 - Able-bodied subject details for study 4.2.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (y)</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
<th>Male/female</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>31</td>
<td>1.73</td>
<td>64</td>
<td>M</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>1.86</td>
<td>88</td>
<td>M</td>
</tr>
<tr>
<td>3</td>
<td>35</td>
<td>1.71</td>
<td>65</td>
<td>M</td>
</tr>
<tr>
<td>4</td>
<td>39</td>
<td>1.76</td>
<td>79</td>
<td>M</td>
</tr>
<tr>
<td>5</td>
<td>23</td>
<td>1.92</td>
<td>84</td>
<td>M</td>
</tr>
<tr>
<td>6</td>
<td>21</td>
<td>1.86</td>
<td>78</td>
<td>M</td>
</tr>
</tbody>
</table>

Table 3 - Amputee subject details for study 4.2.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Level of amputation</th>
<th>Age (y)</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
<th>Male/female</th>
<th>Years as amputee</th>
<th>Prosthesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BK</td>
<td>15</td>
<td>1.77</td>
<td>65</td>
<td>M</td>
<td>15</td>
<td>SACH</td>
</tr>
<tr>
<td>2</td>
<td>BK</td>
<td>23</td>
<td>1.82</td>
<td>65</td>
<td>M</td>
<td>20</td>
<td>SACH</td>
</tr>
<tr>
<td>3</td>
<td>BK</td>
<td>21</td>
<td>1.75</td>
<td>72</td>
<td>M</td>
<td>21</td>
<td>SACH</td>
</tr>
<tr>
<td>4</td>
<td>BK</td>
<td>57</td>
<td>1.72</td>
<td>65</td>
<td>F</td>
<td>33</td>
<td>SACH</td>
</tr>
<tr>
<td>5</td>
<td>AK</td>
<td>23</td>
<td>1.72</td>
<td>80</td>
<td>M</td>
<td>23</td>
<td>hinge knee/ SACH</td>
</tr>
<tr>
<td>6</td>
<td>AK</td>
<td>25</td>
<td>1.75</td>
<td>70</td>
<td>F</td>
<td>15</td>
<td>hinge knee/ SACH</td>
</tr>
<tr>
<td>7</td>
<td>AK</td>
<td>47</td>
<td>1.81</td>
<td>82</td>
<td>M</td>
<td>24</td>
<td>hinge knee/ SACH</td>
</tr>
<tr>
<td>8</td>
<td>AK</td>
<td>31</td>
<td>1.79</td>
<td>77</td>
<td>M</td>
<td>14</td>
<td>hinge knee/ SACH</td>
</tr>
</tbody>
</table>
Table 4 - Able-bodied subject details for studies 5.1 and 5.2.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (y)</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
<th>Male/female</th>
<th>Joint pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>29</td>
<td>1.75</td>
<td>74</td>
<td>M</td>
<td>back/ knee</td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td>1.72</td>
<td>73</td>
<td>M</td>
<td>back</td>
</tr>
<tr>
<td>3</td>
<td>29</td>
<td>1.84</td>
<td>64</td>
<td>M</td>
<td>knee</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>1.79</td>
<td>93</td>
<td>M</td>
<td>ankle/ knee</td>
</tr>
<tr>
<td>5</td>
<td>28</td>
<td>1.73</td>
<td>71</td>
<td>M</td>
<td>knee</td>
</tr>
<tr>
<td>6</td>
<td>25</td>
<td>1.76</td>
<td>75</td>
<td>M</td>
<td>no pain</td>
</tr>
<tr>
<td>7</td>
<td>23</td>
<td>1.85</td>
<td>72.5</td>
<td>M</td>
<td>no pain</td>
</tr>
<tr>
<td>8</td>
<td>26</td>
<td>1.79</td>
<td>86.2</td>
<td>M</td>
<td>no pain</td>
</tr>
<tr>
<td>9</td>
<td>24</td>
<td>1.79</td>
<td>61</td>
<td>M</td>
<td>no pain</td>
</tr>
<tr>
<td>10</td>
<td>27</td>
<td>1.73</td>
<td>81.2</td>
<td>M</td>
<td>no pain</td>
</tr>
</tbody>
</table>

Table 5 - Able-bodied subject details for studies 6.1 and 7.1.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (y)</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
<th>Male/female</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24</td>
<td>1.83</td>
<td>76</td>
<td>M</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>1.69</td>
<td>70.5</td>
<td>F</td>
</tr>
<tr>
<td>3</td>
<td>34</td>
<td>1.67</td>
<td>54</td>
<td>F</td>
</tr>
<tr>
<td>4</td>
<td>28</td>
<td>1.64</td>
<td>55</td>
<td>F</td>
</tr>
<tr>
<td>5</td>
<td>26</td>
<td>1.89</td>
<td>82</td>
<td>M</td>
</tr>
</tbody>
</table>

Table 6 - Able-bodied subject details for studies 6.2 and 7.2.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (y)</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
<th>Male/female</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24</td>
<td>1.83</td>
<td>76</td>
<td>M</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>1.69</td>
<td>70.5</td>
<td>F</td>
</tr>
<tr>
<td>3</td>
<td>34</td>
<td>1.67</td>
<td>54</td>
<td>F</td>
</tr>
<tr>
<td>4</td>
<td>28</td>
<td>1.64</td>
<td>55</td>
<td>F</td>
</tr>
<tr>
<td>5</td>
<td>26</td>
<td>1.89</td>
<td>82</td>
<td>M</td>
</tr>
<tr>
<td>6</td>
<td>27</td>
<td>1.73</td>
<td>87.5</td>
<td>M</td>
</tr>
<tr>
<td>7</td>
<td>20</td>
<td>1.76</td>
<td>78.2</td>
<td>M</td>
</tr>
<tr>
<td>8</td>
<td>25</td>
<td>1.79</td>
<td>92</td>
<td>M</td>
</tr>
</tbody>
</table>

Table 7 - Amputee subject details for studies 6.2 and 7.2.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Level of amputation</th>
<th>Age (y)</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
<th>Male/female</th>
<th>Years as amputee</th>
<th>Prosthesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BK</td>
<td>32</td>
<td>1.8</td>
<td>94.4</td>
<td>M</td>
<td>3</td>
<td>Mutli axis foot</td>
</tr>
<tr>
<td>2</td>
<td>BK</td>
<td>29</td>
<td>1.81</td>
<td>86.9</td>
<td>M</td>
<td>26</td>
<td>Mutli axis foot</td>
</tr>
<tr>
<td>3</td>
<td>BK</td>
<td>49</td>
<td>1.64</td>
<td>84.4</td>
<td>F</td>
<td>9</td>
<td>Mutli axis foot</td>
</tr>
<tr>
<td>4</td>
<td>BK</td>
<td>67</td>
<td>1.67</td>
<td>65</td>
<td>M</td>
<td>18</td>
<td>Mutli axis foot</td>
</tr>
<tr>
<td>5</td>
<td>AK</td>
<td>72</td>
<td>1.78</td>
<td>69.8</td>
<td>M</td>
<td>43</td>
<td>Mutli axis/ hydraulic</td>
</tr>
<tr>
<td>6</td>
<td>AK</td>
<td>61</td>
<td>1.68</td>
<td>84.4</td>
<td>M</td>
<td>23</td>
<td>Mutli axis/ hydraulic</td>
</tr>
<tr>
<td>7</td>
<td>AK</td>
<td>35</td>
<td>1.81</td>
<td>100.5</td>
<td>M</td>
<td>12</td>
<td>Mutli axis/ hydraulic</td>
</tr>
<tr>
<td>8</td>
<td>AK</td>
<td>69</td>
<td>1.79</td>
<td>87.8</td>
<td>M</td>
<td>7</td>
<td>Mutli axis/ hydraulic</td>
</tr>
</tbody>
</table>
Appendix 2

Correlation coefficient (r) for the correlation between the prosthetic and intact limb of above- and below-knee amputees for Fz1, impulse, stance, swing and step times (study 4.2). * denotes significant at p<0.05.

Table 8 - Correlation coefficient for below-knee amputees.

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fz1</td>
<td>-0.463</td>
<td>-0.634</td>
<td>-0.801</td>
<td>0.303</td>
</tr>
<tr>
<td>Impulse</td>
<td>0.547</td>
<td>0.710</td>
<td>0.655</td>
<td>0.181</td>
</tr>
<tr>
<td>Stance time</td>
<td>0.641</td>
<td>0.122</td>
<td>0.064</td>
<td>0.474</td>
</tr>
<tr>
<td>Swing time</td>
<td>0.783</td>
<td>0.567</td>
<td>0.642</td>
<td>0.242</td>
</tr>
<tr>
<td>Step time</td>
<td>0.738</td>
<td>0.732</td>
<td>0.537</td>
<td>0.242</td>
</tr>
</tbody>
</table>

Table 9 - Correlation coefficient for above-knee amputees.

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fz1</td>
<td>0.060</td>
<td>0.528</td>
<td>0.602</td>
<td>0.182</td>
</tr>
<tr>
<td>Impulse</td>
<td>-0.182</td>
<td>-0.158</td>
<td>-0.115</td>
<td>0.568</td>
</tr>
<tr>
<td>Stance time</td>
<td>0.743</td>
<td>-0.019</td>
<td>0.254</td>
<td>0.659</td>
</tr>
<tr>
<td>Swing time</td>
<td>0.803</td>
<td>0.795</td>
<td>0.349</td>
<td>0.571</td>
</tr>
<tr>
<td>Step time</td>
<td>0.137</td>
<td>0.743</td>
<td>0.510</td>
<td>0.324</td>
</tr>
</tbody>
</table>
Appendix 3

Mean able-bodied impact shock and shock attenuation values (+/- 1 SD) and individual amputee data (study 5.2)

Impact shock (g)
Able-bodied subjects:

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.264</td>
<td>0.40595</td>
<td>0.545</td>
<td>1.37</td>
</tr>
<tr>
<td>SD</td>
<td>0.12</td>
<td>0.19</td>
<td>0.2</td>
<td>0.4</td>
</tr>
<tr>
<td>Range</td>
<td>0.14-0.38</td>
<td>0.22-0.60</td>
<td>0.33-0.77</td>
<td>0.97-1.77</td>
</tr>
</tbody>
</table>

Below-knee amputees:

<table>
<thead>
<tr>
<th>Subject</th>
<th>Limb</th>
<th>Walking speed</th>
</tr>
</thead>
<tbody>
<tr>
<td>MK</td>
<td>intact limb</td>
<td>0.25 0.36 0.42 0.42</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.56 0.74 1.18 1.65</td>
</tr>
<tr>
<td>R.B.</td>
<td>intact limb</td>
<td>0.32 0.47 0.6 0.99</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.36 0.58 0.6 0.99</td>
</tr>
<tr>
<td>J.K.</td>
<td>intact limb</td>
<td>0.51 0.43 0.54 0.75</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.54 0.95 1.25 1.72</td>
</tr>
<tr>
<td>N.L.</td>
<td>intact limb</td>
<td>0.19 0.71 0.97 1.36</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.53 0.71 0.97 1.36</td>
</tr>
<tr>
<td>D.C.</td>
<td>intact limb</td>
<td>0.42 0.67 1.01 1.19</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.76 0.88 1.33 1.61</td>
</tr>
</tbody>
</table>

Above-knee amputees:

<table>
<thead>
<tr>
<th>Subject</th>
<th>Limb</th>
<th>Walking speed</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.W.</td>
<td>intact limb</td>
<td>0.35 0.59 1.32 1.58</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.35 0.44 0.93 1.1</td>
</tr>
<tr>
<td>J.N.</td>
<td>intact limb</td>
<td>0.32 0.77 0.92 1.34</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.2 0.6 0.71 0.93</td>
</tr>
<tr>
<td>B.M.</td>
<td>intact limb</td>
<td>0.27 0.56 0.57 0.63</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.41 0.74 0.75 1.06</td>
</tr>
<tr>
<td>A.B.</td>
<td>intact limb</td>
<td>0.38 0.53 0.72 0.91</td>
</tr>
<tr>
<td></td>
<td>prosthetic</td>
<td>0.49 0.76 1.07 1.36</td>
</tr>
</tbody>
</table>

Shock attenuation (%).

Able-bodied subjects:

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.264</td>
<td>0.40595</td>
<td>0.545</td>
<td>1.37</td>
</tr>
<tr>
<td>SD</td>
<td>22.3</td>
<td>19.7</td>
<td>24.6</td>
<td>29.3</td>
</tr>
<tr>
<td>Range</td>
<td>40.7-85.3</td>
<td>48.1-87.5</td>
<td>48.8-98.0</td>
<td>49.2-107.8</td>
</tr>
</tbody>
</table>
### Below-knee amputees:

<table>
<thead>
<tr>
<th>Subject</th>
<th>Limb</th>
<th>Walking speed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0.5</td>
</tr>
<tr>
<td>RB</td>
<td>prosthetic</td>
<td>50.1</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>37.14</td>
</tr>
<tr>
<td>MK</td>
<td>prosthetic</td>
<td>76</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>40.1</td>
</tr>
<tr>
<td>D.C.</td>
<td>prosthetic</td>
<td>74</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>49.41</td>
</tr>
<tr>
<td>J.K.</td>
<td>prosthetic</td>
<td>68.8</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>32.14</td>
</tr>
<tr>
<td>N.L.</td>
<td>prosthetic</td>
<td>73.4</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>44</td>
</tr>
</tbody>
</table>

### Above-knee amputees:

<table>
<thead>
<tr>
<th>Subject</th>
<th>Limb</th>
<th>Walking speed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0.5</td>
</tr>
<tr>
<td>C.W.</td>
<td>prosthetic</td>
<td>51.7</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>43.62</td>
</tr>
<tr>
<td>J.N.</td>
<td>prosthetic</td>
<td>61.97</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>59.6</td>
</tr>
<tr>
<td>B.M.</td>
<td>prosthetic</td>
<td>67.74</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>64.59</td>
</tr>
<tr>
<td>A.B.</td>
<td>prosthetic</td>
<td>77.8</td>
</tr>
<tr>
<td></td>
<td>intact lim</td>
<td>26.59</td>
</tr>
</tbody>
</table>
Appendix 4

Mean able-bodied impact shock asymmetry and shock attenuation asymmetry values (+/- 1 SD) and individual amputee data (study 5.2)

Impact shock asymmetry (%).

Able-bodied subjects:

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>X</td>
<td>Mean</td>
<td>44.7</td>
<td>50.2</td>
<td>31.4</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>21.2</td>
<td>35.9</td>
<td>26.7</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>23.5-65.9</td>
<td>14.3-86.1</td>
<td>4.7-58.1</td>
</tr>
<tr>
<td>Y</td>
<td>Mean</td>
<td>21</td>
<td>36.3</td>
<td>37.7</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>21.4</td>
<td>27.5</td>
<td>17.9</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>0-42.4</td>
<td>8.8-63.8</td>
<td>19.8</td>
</tr>
<tr>
<td>Z</td>
<td>Mean</td>
<td>11</td>
<td>17.9</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>14.2</td>
<td>7.5</td>
<td>11.4</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>0-25.2</td>
<td>10.4-25.4</td>
<td>8.6-31.4</td>
</tr>
</tbody>
</table>

Below-knee amputees:

<table>
<thead>
<tr>
<th></th>
<th>Subject</th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>X</td>
<td>M.K.</td>
<td>16.1</td>
<td>26.5</td>
<td>18.2</td>
<td>27.3</td>
</tr>
<tr>
<td></td>
<td>R.B.</td>
<td>97.8</td>
<td>94.6</td>
<td>87.9</td>
<td>27.6</td>
</tr>
<tr>
<td></td>
<td>J.K.</td>
<td>37.5</td>
<td>113</td>
<td>18.2</td>
<td>55.3</td>
</tr>
<tr>
<td></td>
<td>N.L.</td>
<td>49.1</td>
<td>53.2</td>
<td>101</td>
<td>37.7</td>
</tr>
<tr>
<td></td>
<td>D.C.</td>
<td>75</td>
<td>12.7</td>
<td>20.3</td>
<td>8.5</td>
</tr>
<tr>
<td>Y</td>
<td>M.K.</td>
<td>9.4</td>
<td>17.8</td>
<td>3.4</td>
<td>17.3</td>
</tr>
<tr>
<td></td>
<td>R.B.</td>
<td>44.9</td>
<td>18.6</td>
<td>12.7</td>
<td>28.3</td>
</tr>
<tr>
<td></td>
<td>J.K.</td>
<td>45.7</td>
<td>30.4</td>
<td>12.3</td>
<td>25.3</td>
</tr>
<tr>
<td></td>
<td>N.L.</td>
<td>33.2</td>
<td>17.7</td>
<td>38.7</td>
<td>33.8</td>
</tr>
<tr>
<td></td>
<td>D.C.</td>
<td>56.6</td>
<td>0</td>
<td>5.4</td>
<td>19.7</td>
</tr>
<tr>
<td>Z</td>
<td>M.K.</td>
<td>76.5</td>
<td>111.1</td>
<td>118.64</td>
<td>120</td>
</tr>
<tr>
<td></td>
<td>R.B.</td>
<td>11.8</td>
<td>39.2</td>
<td>24.3</td>
<td>15.2</td>
</tr>
<tr>
<td></td>
<td>J.K.</td>
<td>5.7</td>
<td>24.8</td>
<td>5.76</td>
<td>4.2</td>
</tr>
<tr>
<td></td>
<td>N.L.</td>
<td>94.4</td>
<td>49.1</td>
<td>57</td>
<td>57.8</td>
</tr>
<tr>
<td></td>
<td>D.C.</td>
<td>59.8</td>
<td>14.6</td>
<td>27.4</td>
<td>30</td>
</tr>
</tbody>
</table>
### Above-knee amputees:

<table>
<thead>
<tr>
<th></th>
<th>Subject</th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>X</td>
<td>A.B.</td>
<td>72.1</td>
<td>48.6</td>
<td>4</td>
<td>9.3</td>
</tr>
<tr>
<td></td>
<td>B.M.</td>
<td>51.3</td>
<td>1.8</td>
<td>26.2</td>
<td>3.2</td>
</tr>
<tr>
<td></td>
<td>J.N.</td>
<td>91.4</td>
<td>55</td>
<td>20.5</td>
<td>35.7</td>
</tr>
<tr>
<td></td>
<td>C.W.</td>
<td>86.8</td>
<td>69.1</td>
<td>75.2</td>
<td>139.1</td>
</tr>
</tbody>
</table>

### Below-knee amputees:

<table>
<thead>
<tr>
<th></th>
<th>Subject</th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.K.</td>
<td>61.8</td>
<td>56.8</td>
<td>44</td>
<td>43.1</td>
<td></td>
</tr>
<tr>
<td>R.B.</td>
<td>29.7</td>
<td>21.9</td>
<td>5.7</td>
<td>5.58</td>
<td></td>
</tr>
<tr>
<td>J.K.</td>
<td>72.6</td>
<td>19.9</td>
<td>14.1</td>
<td>10.26</td>
<td></td>
</tr>
<tr>
<td>N.L.</td>
<td>50.1</td>
<td>43.3</td>
<td>15.3</td>
<td>16.13</td>
<td></td>
</tr>
<tr>
<td>D.C.</td>
<td>39.9</td>
<td>39.3</td>
<td>24</td>
<td>13.2</td>
<td></td>
</tr>
</tbody>
</table>

### Able-bodied subjects:

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>9.1</td>
<td>6.7</td>
<td>4.9</td>
<td>3.6</td>
</tr>
<tr>
<td>SD</td>
<td>3.1</td>
<td>4.5</td>
<td>4.1</td>
<td>2.3</td>
</tr>
<tr>
<td>Range</td>
<td>6-12.2</td>
<td>2.2-11.2</td>
<td>0.8-9.0</td>
<td>1.3-5.9</td>
</tr>
</tbody>
</table>

### Shock attenuation asymmetry (%).

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.B.</td>
<td>17.5</td>
<td>2.2</td>
<td>49.9</td>
<td>54</td>
</tr>
<tr>
<td>B.M.</td>
<td>47.8</td>
<td>8.6</td>
<td>8.6</td>
<td>46.8</td>
</tr>
<tr>
<td>J.N.</td>
<td>40</td>
<td>42</td>
<td>43</td>
<td>51.4</td>
</tr>
<tr>
<td>C.W.</td>
<td>18.6</td>
<td>5.2</td>
<td>14.7</td>
<td>63.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.B.</td>
<td>25.3</td>
<td>35.7</td>
<td>39.1</td>
<td>39.6</td>
</tr>
<tr>
<td>B.M.</td>
<td>41.2</td>
<td>27.7</td>
<td>27.3</td>
<td>50.9</td>
</tr>
<tr>
<td>J.N.</td>
<td>46.2</td>
<td>24.8</td>
<td>25.8</td>
<td>36.1</td>
</tr>
<tr>
<td>C.W.</td>
<td>0</td>
<td>29.1</td>
<td>34.7</td>
<td>35.8</td>
</tr>
</tbody>
</table>

### Below-knee amputees:

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.W.</td>
<td>17</td>
<td>12</td>
<td>9.26</td>
<td>1.74</td>
</tr>
<tr>
<td>J.N.</td>
<td>3.9</td>
<td>3.67</td>
<td>3.52</td>
<td>1.49</td>
</tr>
<tr>
<td>B.M.</td>
<td>4.8</td>
<td>1.09</td>
<td>11.1</td>
<td>16.85</td>
</tr>
<tr>
<td>A.B.</td>
<td>98.1</td>
<td>61.54</td>
<td>9.82</td>
<td>5.3</td>
</tr>
</tbody>
</table>
Above-knee amputees:

<table>
<thead>
<tr>
<th>Subject</th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.B.</td>
<td>72.1</td>
<td>48.6</td>
<td>4</td>
<td>9.3</td>
</tr>
<tr>
<td>B.M.</td>
<td>51.3</td>
<td>1.8</td>
<td>26.2</td>
<td>3.2</td>
</tr>
<tr>
<td>J.N.</td>
<td>91.4</td>
<td>55</td>
<td>20.5</td>
<td>35.7</td>
</tr>
<tr>
<td>C.W.</td>
<td>86.8</td>
<td>69.1</td>
<td>75.2</td>
<td>139.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Subject</th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.B.</td>
<td>17.5</td>
<td>2.2</td>
<td>49.9</td>
<td>54</td>
</tr>
<tr>
<td>B.M.</td>
<td>47.8</td>
<td>8.6</td>
<td>8.6</td>
<td>46.8</td>
</tr>
<tr>
<td>J.N.</td>
<td>40</td>
<td>42</td>
<td>43</td>
<td>51.4</td>
</tr>
<tr>
<td>C.W.</td>
<td>18.6</td>
<td>5.2</td>
<td>14.7</td>
<td>63.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Subject</th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.B.</td>
<td>25.3</td>
<td>35.7</td>
<td>39.1</td>
<td>39.6</td>
</tr>
<tr>
<td>B.M.</td>
<td>41.2</td>
<td>27.7</td>
<td>27.3</td>
<td>50.9</td>
</tr>
<tr>
<td>J.N.</td>
<td>46.2</td>
<td>24.8</td>
<td>25.8</td>
<td>36.1</td>
</tr>
<tr>
<td>C.W.</td>
<td>0</td>
<td>29.1</td>
<td>34.7</td>
<td>35.8</td>
</tr>
</tbody>
</table>

Shock attenuation asymmetry (%).

Able-bodied subjects:

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>9.1</td>
<td>6.7</td>
<td>4.9</td>
<td>3.6</td>
</tr>
<tr>
<td>SD</td>
<td>3.1</td>
<td>4.5</td>
<td>4.1</td>
<td>2.3</td>
</tr>
<tr>
<td>Range</td>
<td>6-12.2</td>
<td>2.2-11.2</td>
<td>0.8-9.0</td>
<td>1.3-5.9</td>
</tr>
</tbody>
</table>

Below-knee amputees:

<table>
<thead>
<tr>
<th>Subject</th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.K.</td>
<td>61.8</td>
<td>56.8</td>
<td>44</td>
<td>43.1</td>
</tr>
<tr>
<td>R.B.</td>
<td>29.7</td>
<td>21.9</td>
<td>5.7</td>
<td>5.58</td>
</tr>
<tr>
<td>J.K.</td>
<td>72.6</td>
<td>19.9</td>
<td>14.1</td>
<td>10.26</td>
</tr>
<tr>
<td>N.L.</td>
<td>50.1</td>
<td>43.3</td>
<td>15.3</td>
<td>16.13</td>
</tr>
<tr>
<td>D.C.</td>
<td>39.9</td>
<td>39.3</td>
<td>24</td>
<td>13.2</td>
</tr>
</tbody>
</table>

Above-knee amputees:

<table>
<thead>
<tr>
<th>Subject</th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.W.</td>
<td>17</td>
<td>12</td>
<td>9.26</td>
<td>1.74</td>
</tr>
<tr>
<td>J.N.</td>
<td>3.9</td>
<td>3.67</td>
<td>3.52</td>
<td>1.49</td>
</tr>
<tr>
<td>B.M.</td>
<td>4.8</td>
<td>1.09</td>
<td>11.1</td>
<td>16.85</td>
</tr>
<tr>
<td>A.B.</td>
<td>98.1</td>
<td>61.54</td>
<td>9.82</td>
<td>5.3</td>
</tr>
</tbody>
</table>
Appendix 5

Correlation coefficient (r) for the correlation between the prosthetic and intact limbs of above- and below-knee amputees for joint kinematics (study 6.2). * denotes significant at p<0.05.

Table 10 - Correlation coefficient for below-knee amputees.

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle PF angle</td>
<td>0.165</td>
<td>-0.202</td>
<td>-0.366</td>
<td>0.616</td>
</tr>
<tr>
<td>Ankle DF angle</td>
<td>0.791</td>
<td>0.802</td>
<td>0.95</td>
<td>0.711</td>
</tr>
<tr>
<td>Knee HS</td>
<td>0.416</td>
<td>0.563</td>
<td>0.624</td>
<td>0.684</td>
</tr>
<tr>
<td>Knee MS</td>
<td>-0.517</td>
<td>0.350</td>
<td>0.688</td>
<td>0.539</td>
</tr>
<tr>
<td>Knee TO</td>
<td>0.732</td>
<td>0.654</td>
<td>0.803</td>
<td>0.672</td>
</tr>
<tr>
<td>Hip flex</td>
<td>-0.362</td>
<td>-0.658</td>
<td>0.031</td>
<td>0.714</td>
</tr>
<tr>
<td>Hip ext</td>
<td>0.077</td>
<td>-0.484</td>
<td>0.431</td>
<td>0.613</td>
</tr>
</tbody>
</table>

Table 11 - Correlation coefficient for above-knee amputees.

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle PF angle</td>
<td>-0.261</td>
<td>0.439</td>
<td>0.621</td>
<td>0.735</td>
</tr>
<tr>
<td>Ankle DF angle</td>
<td>-0.508</td>
<td>-0.611</td>
<td>-0.672</td>
<td>-0.712</td>
</tr>
<tr>
<td>Knee HS</td>
<td>0.412</td>
<td>0.605</td>
<td>0.525</td>
<td>0.273</td>
</tr>
<tr>
<td>Knee MS</td>
<td>-0.437</td>
<td>0.492</td>
<td>0.373</td>
<td>0.308</td>
</tr>
<tr>
<td>Knee TO</td>
<td>-0.559</td>
<td>-0.129</td>
<td>-0.603</td>
<td>-0.734</td>
</tr>
<tr>
<td>Hip flex</td>
<td>-0.763</td>
<td>-0.499</td>
<td>-0.143</td>
<td>-0.699</td>
</tr>
<tr>
<td>Hip ext</td>
<td>-0.406</td>
<td>-0.546</td>
<td>-0.723</td>
<td>-0.617</td>
</tr>
</tbody>
</table>

Correlation coefficient (r) for the correlation between the prosthetic and intact limbs of above- and below-knee amputees for joint moments (study 6.2). * denotes significant at p<0.05.

Table 12 - Correlation coefficient for below-knee amputees.

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle PF moment</td>
<td>0.798</td>
<td>0.698</td>
<td>0.261</td>
<td>0.629</td>
</tr>
<tr>
<td>Ankle DF moment</td>
<td>0.769</td>
<td>0.659</td>
<td>0.726</td>
<td>0.659</td>
</tr>
<tr>
<td>Knee flex moment</td>
<td>0.127</td>
<td>0.363</td>
<td>-0.017</td>
<td>0.421</td>
</tr>
<tr>
<td>Knee ext moment</td>
<td>-0.798</td>
<td>-0.613</td>
<td>0.469</td>
<td>0.731</td>
</tr>
<tr>
<td>Hip flex moment</td>
<td>0.389</td>
<td>0.483</td>
<td>0.539</td>
<td>0.649</td>
</tr>
<tr>
<td>Hip ext moment</td>
<td>0.286</td>
<td>0.132</td>
<td>0.591</td>
<td>-0.357</td>
</tr>
</tbody>
</table>

Table 13 - Correlation coefficient for above-knee amputees.

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle PF moment</td>
<td>0.169</td>
<td>-0.544</td>
<td>-0.316</td>
<td>-0.129</td>
</tr>
<tr>
<td>Ankle DF moment</td>
<td>-0.387</td>
<td>0.416</td>
<td>0.762</td>
<td>-0.139</td>
</tr>
<tr>
<td>Knee flex moment</td>
<td>0.079</td>
<td>0.553</td>
<td>0.772</td>
<td>0.126</td>
</tr>
<tr>
<td>Knee ext moment</td>
<td>0.171</td>
<td>-0.505</td>
<td>0.513</td>
<td>0.755</td>
</tr>
<tr>
<td>Hip flex moment</td>
<td>0.411</td>
<td>0.514</td>
<td>0.552</td>
<td>0.383</td>
</tr>
<tr>
<td>Hip ext moment</td>
<td>0.800</td>
<td>0.478</td>
<td>0.667</td>
<td>0.791</td>
</tr>
</tbody>
</table>
Correlation coefficient \((r)\) for the correlation between the prosthetic and intact limbs of above- and below-knee amputees for joint power output (study 6.2). * denotes significant at \(p<0.05\).

Table 14 - Correlation coefficient for below-knee amputees.

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle power gen</td>
<td>-0.674</td>
<td>-0.630</td>
<td>0.207</td>
<td>0.361</td>
</tr>
<tr>
<td>Ankle power abs</td>
<td>0.801</td>
<td>0.799</td>
<td>0.642</td>
<td>0.698</td>
</tr>
<tr>
<td>Knee power gen</td>
<td>-0.677</td>
<td>-0.798</td>
<td>-0.483</td>
<td>-0.621</td>
</tr>
<tr>
<td>Knee power abs</td>
<td>-0.056</td>
<td>0.288</td>
<td>0.009</td>
<td>0.421</td>
</tr>
<tr>
<td>Hip power gen</td>
<td>-0.566</td>
<td>-0.788</td>
<td>-0.684</td>
<td>-0.491</td>
</tr>
<tr>
<td>Hip power abs</td>
<td>0.304</td>
<td>0.452</td>
<td>0.674</td>
<td>0.216</td>
</tr>
</tbody>
</table>

Table 15 - Correlation coefficient for above-knee amputees.

<table>
<thead>
<tr>
<th></th>
<th>0.5</th>
<th>0.9</th>
<th>1.2</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle power gen</td>
<td>-0.784</td>
<td>-0.217</td>
<td>-0.338</td>
<td>-0.706</td>
</tr>
<tr>
<td>Ankle power abs</td>
<td>-0.491</td>
<td>0.780</td>
<td>0.402</td>
<td>-0.144</td>
</tr>
<tr>
<td>Knee power gen</td>
<td>-0.651</td>
<td>-0.769</td>
<td>0.346</td>
<td>0.558</td>
</tr>
<tr>
<td>Knee power abs</td>
<td>0.468</td>
<td>0.513</td>
<td>0.455</td>
<td>0.498</td>
</tr>
<tr>
<td>Hip power gen</td>
<td>0.148</td>
<td>-0.419</td>
<td>-0.369</td>
<td>-0.412</td>
</tr>
<tr>
<td>Hip power abs</td>
<td>0.344</td>
<td>0.351</td>
<td>-0.033</td>
<td>0.588</td>
</tr>
</tbody>
</table>