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ANALYSIS OF GROUND REACTION FORCES IN THE ANTERIOR CRUCIATE DEFICIENT KNEE

by

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Analysis of Ground Reaction Forces In the Anterior Cruciate Deficient Knee

Joe Glenn

Abstract--The purpose of the study was to evaluate ground reaction forces as a measure of ACL function. Data was collected, utilizing a force plate, for 14 subjects suffering at least a second degree sprain of the ACL. Fourier analysis was performed on the data collected and the Fourier coefficients for the injured limbs were compared (paired t-test) to those produced by the uninjured limb. Significant changes were observed for several coefficients. Reasons for the changes and possible uses for the instrumentation were discussed. Recommendations included reformulation of the force curves from the sanatized data and comparison to parametric analysis.

CHAPTER 1

Introduction

"Anatomically speaking, the anterior cruciate ligament connects the femur to the tibia, but most athletes know it as the part of the knee that breaks the heart." (Brant, 1989).

The anterior cruciate ligament (ACL) has been identified as the most frequently injured ligament involving the knee joint (Tibone, Antich, Fanton, Moynes, and Perry, 1986; Andrish, 1985; Johnson, 1983). Unfortunately, diagnosis of the injured ACL is often difficult and a great number of ACL tears go undectected. If and when the injury is correctly diagnosed treatment regimens are variable and are influenced by both situational and medical factors. The prognosis for return of function or return to sport following treatment is uncertain at best. Many athletes are advised to modify their activities and many others forced to by increasing degeneration of knee function. A test which could assist in diagnosis, provide input for treatment descisions, and reliably indicate the functional integrity of an injured joint would prove invaluable in the management of ACL injuries.

Boccardi, Chiesa and Pedotti (1977) have recommended measurement of ground reaction forces with a force plate as a test of knee function. They claimed that the technology is

suitable for clinical use and the results are both quantitative and reliable.

Statement of the Problem

The purpose of this paper is to evaluate the measurement of ground reaction forces by force plate, as a functional test for ACL insufficiency.

Delimitations

The suggested procedure is not recommended as a substitute for a thorough history and clinical examination but rather as a supplement which will allow the examiner to make a more complete assessment of the injured joint especially in cases of equivocal diagnosis.

Definition of terms

Force Platform. An electromechanical device that generates electrical signals proportional to the components of force acting upon it. The resultant force is resolved into three components; vertical, medial-lateral, and anterior-posterior (Rogers & Cavanagh, 1984).

Ground Reaction Forces. The forces that act on the body as a result of interaction with the supporting surface. Newton's third law asserts that ground reaction forces will be equal and opposite to those which the body exerts on the ground (Rogers & Cavanagh, 1984).

CHAPTER 2

Review of Literature

Introduction

The following review of literature will focus upon; functional anatomy of the knee, functional anatomy of the anterior cruciate ligament, injuries to the ACL, diagnosis of ACL injuries, and a rationale for the use of the proposed instrumentation.

The knee is the largest and most complex joint in the human body (Van De Graff, 1984; Moore, 1982) The joint actually consists of two osseous articulations; patello-femoral and tibio-femoral (Blackburn & Craig, 1980). Although the patello-femoral articulation is important for knee joint function, it is for the most part incidental for the topic in question. Because the ACL links tibia and femur, the tibio-femoral articulation is the structure of intrest. All subsequent use of the terms knee and knee joint shall refer to this articulation unless otherwise specified.

Functional Anatomy

The knee joint must reconcile two essentially mutually exclusive biomechanical functions: mobility and stability (Hay,

1982; Kapandji, 1970). Joint mobility has been described as the degree to which the articulation is allowed to move before being restricted, either by the bony configuration or surrounding tissues (Kreighbaum & Barthels, 1985). Knee joint mobility is essential to provide the range of movement needed for locomotion (Hay, 1982; Kapandji, 1970). Stability has been defined as the ability of the skeletal framework to absorb forces and withstand unwanted motion without injury to either the joint or the surrounding tissue (Kreighbaum & Barthels, 1985). Knee joint stability must be sufficient to withstand the large forces produced by the combination of body weight and relatively long lever arms (Hay ,1982; Kapandji, 1970). In the knee, stability has been sacrificed in the interest of mobility (McLeod & Hunter, 1980).

Joint Mobility

Description of joint mobility has referred to two distinct parameters; directions of movement or degrees of freedom and amount of movement or range of motion (ROM) (Hay, 1982). Factors that can influence these two parameters include; shape of the articulating surfaces, tension in soft tissue surrounding the joint, and compression of soft tissue adjacent to the joint (Kreighbaum & Barthels, 1985).

Kapandji (1970) described the knee as a joint which possesses one degree of freedom and one secondary or

accessory plane of movement. The principle motions of flexion and extension occur in the saggittal plane around a left-right axis (Moore, 1982). Flexion has been described as movement of the posterior aspect of the leg towards the posterior aspect of the thigh, while the reverse movement has been designated as extension (Kapandji, 1970). The accessory voluntary rotational movement occurs around the longitudinal axis of the leg in the transverse or horizontal plane only when the leg is flexed (Kapandji, 1970). Automatic rotation of the tibia in the transverse plane accompanies the principle flexion and extension movements: lateral rotation occurring in the final stages of extension and medial rotation accompanying the initiation of flexion (Kapandji, 1970). This involuntary movement is known as the screw home mechanism (Hay, 1982). The knee joint has been designated a modified hinge joint because it possesses the secondary rotational movements (Moore, 1982).

The ROM of the knee is measured from a position of reference in which the long axis of the thigh lines up with the long axis of the leg (Kapandji, 1970). This position, the so-called anatomical position is generally accepted as the limit of active extension although many individuals are able to extend the leg 10º past this point (Kapandji, 1970). Most others are able to passively extend the leg at least the same

amount (Kapandji, 1970). With the hip in a neutral position approximately 130º of flexion can be expected (Kapandji, 1970). This range can be increased roughly 10º (see Figure 1) by flexing the hip and decreased roughly the same amount by extending the hip (Kapandji, 1970). Passive flexion compresses the muscles of the calf and the thigh increasing the range to approximately 160º (Kapandji, 1970).

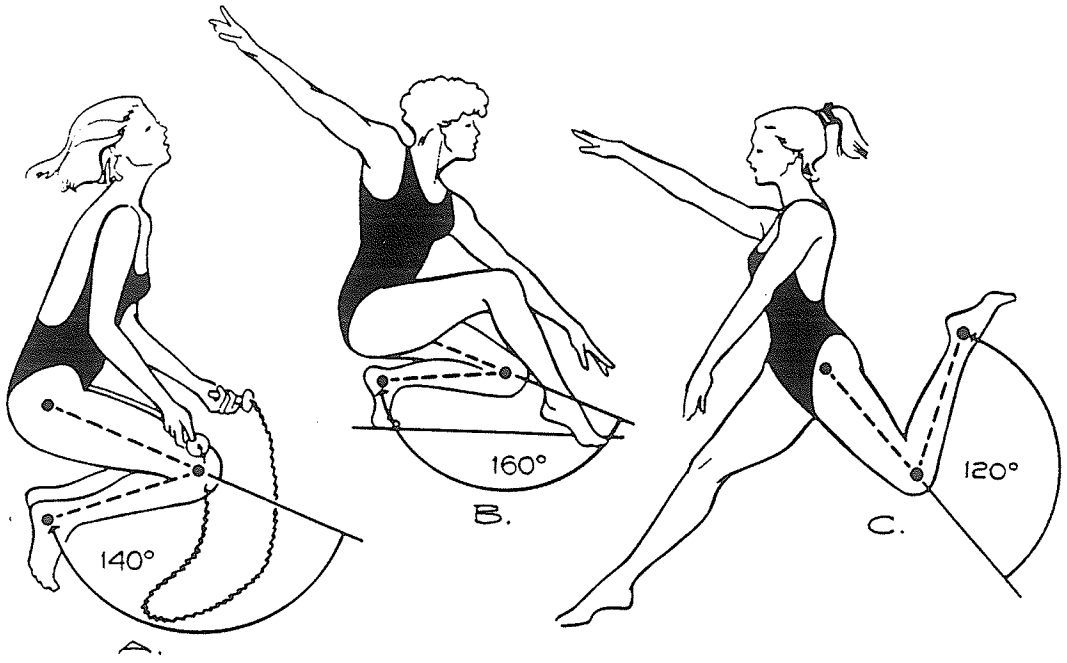

FIGURE 1 FLEXION OF THE KNEE
A. WITH HIP FLEXED B. PASSIVE FLEXION
C. WITH HIP EXTENDED
ADAPTED FROM KAPANDJI (1970)

When the knee is in the flexed position, the tibia can be rotated laterally approximately 40° and medially 30° from the position of reference (foot pointing straight ahead, see Figure 2) (Kapandji,1970). Passive rotation of the leg increases the ROM approximately 5-10° in each direction (Kapandji, 1970). Automatic rotation is limited to about 15° of tibal or femoral rotation depending upon which bone is most free to move (Müller, 1983).

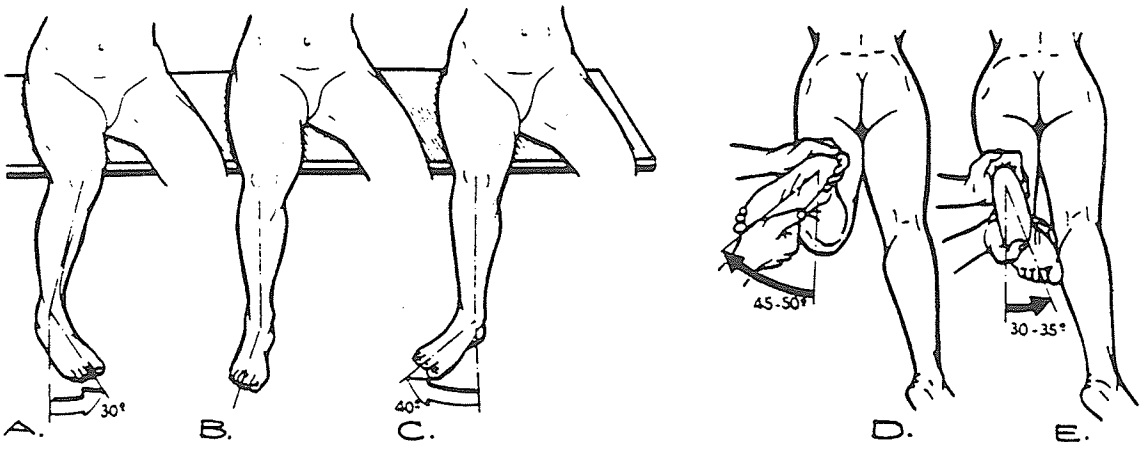

FIGURE 2 ROTATION AT THE KNEE
A. MEDIAL B. NEUTRAL OR REFERENCE C. LATERAL
D. PASSIVE LATERAL ROTATION E. PASSIVE MEDIAL ROTATION
ADAPTED FROM KAPANDJI (1970)

Joint Stability

Kreighbaum & Barthels (1985) defined joint stability as the capability of the joint to absorb and distribute forces and to withstand abnormal movements without injury to either the joint or the soft tissue surrounding it. Maintenance of knee joint stability is complicated because the joint is required to maintain stability under a wide range of circumstances (Welsh, 1980). The joint can be either loaded (weight-bearing) or unloaded (non-weight-bearing) and conditions either changing (dynamic) or constant (static) (McLeod, 1985; McLeod & Hunter, 1980; Noyes, Grood, Butler, & Malek, 1980). In addition the knee must remain stable throughout a large ROM while subjected to a range of forces which can be several times body weight (Nigg, 1985). Knee joint stability is provided by the interaction of joint geometry, compressive forces acting on the joint, ligaments, muscles, joint capsule, and menisci (Noyes, Grood, Butler & Raterman, 1980). Stability represents a balance of forces between stresses imposed on the knee joint by activity or movement and the internal forces generated by the stabilizing factors (Noyes, Grood, Butler, & Malek, 1980). Muscles have been identified as active stabilizers while the other structures have been described as passive stabilizers (Andrews & Axe, 1985; Noyes,

Grood, Butler & Raterman, 1980; Hughston , Andrews, Cross & Moschi, 1976).

Structures of the the knee joint will be discussed with emphasis on their respective relationships with joint mobility and stability.

Osteology of the knee joint

Structurally, the knee joint has been described as a double condyloid joint (Warwick & Williams, 1973). On the distal end of the femur the articulating surfaces consist of two cam-shaped condyles which are flattened on the inferior surfaces and separated by a "U" shaped intercondylar notch (Moore, 1982). The smaller lateral condyle is circular in shape while the larger, oval shaped medial condyle is elongated along the anterior-posterior axis (Warwick & Williams, 1973). The tibial articulating surface can be best described as a plateau on the superior tibia formed by flattening of the tibial condyles (Welsh, 1980). A narrow protruding tibial spine runs along an anterior-posterior axis separating the flattened medial and lateral tibial condyles (Blackburn & Craig, 1980). The lateral tibial plateau has a convex surface with a pronounced slope towards the posterior border and the medial surface is concave (Welsh, 1980).

The opposing articular surfaces are more or less reciprocally shaped; the medial tibial condyle is oval shaped, the lateral smaller and more nearly round, and the tibial spine fits into the intercondylar notch (Blackburn & Craig, 1980). As in all synovial joints the articulating surfaces are covered in hyaline cartilage (Moore, 1982).

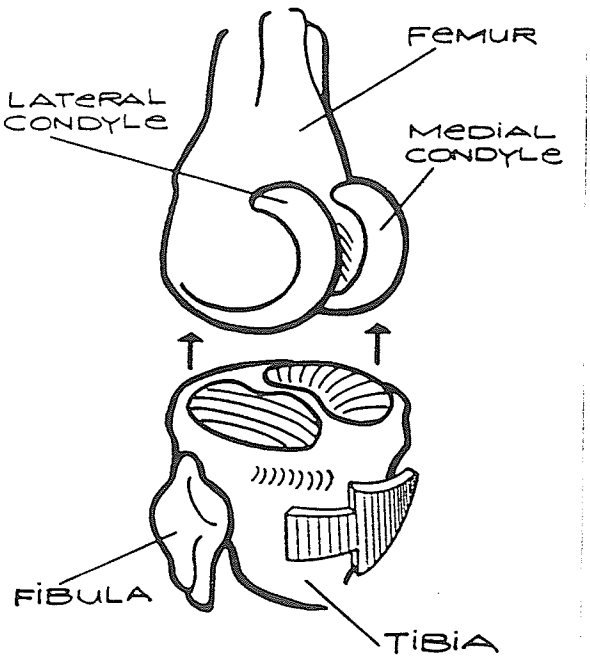

FIGURE 3 BONES OF THE KNEE ADAPTED FROM KAPANDJI (1970)

Movement of the joint is dictated to a great extent by joint geometry (ie the bony structure)(McLeod & Hunter, 1980). The anterior-posterior orientation of the tibial spine allows anterior-posterior movements in the saggittal plane but blocks medial-lateral movement in the frontal plane (Johnson & Pope,

1976). The sloping extremities of the intercondylar eminence allow tibial rotation only when the knee is flexed (Johnson & Pope, 1976). The greatest amount of rotation occurs when the joint is in a position of 35-50o of flexion (Abbot, Saunders, Bost, & Anderson, 1944) and the axis of rotation lies medial to the joint center (Müller, 1983). Automatic rotation, often referred to as the screw home mechanism, is a product of the inequalities of shape and size of the medial and lateral condyles (Ellison & Berg, 1985).

The flexion-extension mechanism combines rolling and gliding movements of the articulating surfaces (Müller, 1983; Ellison & Berg, 1985). The rolling component consists of rotation of the femoral
condyles on the tibial plateau causing translation in the saggittal plane of both the joint axis (point of contact) and the axis of the femoral condyles (Ellison & Berg, 1985). In the gliding motion the two articulating surfaces slide past one another and the two axis maintain their position (Ellison & Berg, 1985). If the mechanism of flexion-extension was comprised of a pure rolling motion, the femur would rotate off the top of the tibial plateau before the entire ROM had been achieved (Ellison & Berg, 1985). If on the other hand the mechanism consisted entirely of gliding, there would be premature impingement of the femoral shaft on the posterior

tibial plateau during flexion (Ellison & Berg, 1985). The ratio of rolling to gliding is variable; increasing from 1:2 in the early stages of flexion, to 1:4 as the knee approaches terminal flexion (Müller, 1983; Radin, 1976).

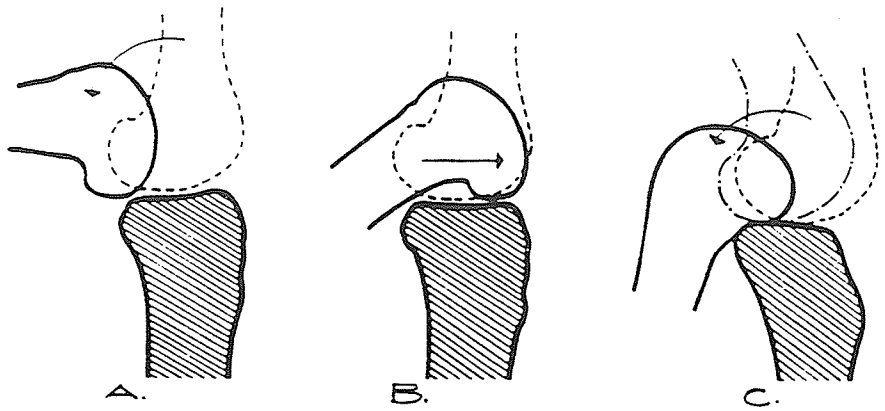

FIGURE 4
A. PURE ROLLING MOTION – FEMUR ROLLS OFF TIBIA
B. PURE GLIDING MOTION – TIBIA BLOCKS FULL ROM.
C. COMBINED ROLLING GLIDING ALLOWS INCREASED ROM WITH STABILITY.
ADAPTED FROM KAPANDJI (1970)

Automatic rotation occurs during the final 20º of extension (Müller, 1983). As the knee approaches full extension, the shorter lateral femoral condyle reaches the terminal position first, subsequently becoming the axis for the rotational movement (Müller, 1983). The longer medial condyle continues the rolling gliding movement resulting in external rotation of the tibia on the femur (Müller, 1983). If the foot is fixed (as in the weight-bearing position) the femur internally rotates

above the fixed tibia (Müller, 1983). The joint is then locked into full extension and must rotate in the opposite direction to initiate flexion (Ellison & Berg, 1985).

The bony configuration of the knee joint is not inherently stable. The convex inferior surfaces of the femur rest upon a convex superior surface of the lateral tibial plateau and an imperceptibly concave superior surface of the medial tibial condyle (McLeod & Hunter, 1980). The superior aspect of the tibia slopes posteriorly but there is no bony restraint to anterior-posterior motion (McLeod & Hunter, 1980). The two long articulating bones constitute relatively long levers which in turn produce large distracting forces, especially in the frontal plane (Warwick & Williams, 1973). There are some minor osseous concessions to stability; the slight concavity of the medial tibial plateau and the flattening of the tibial condyles add somewhat to stability (McLeod & Hunter,1980; Moore, 1982). The tibial spine resists medial-lateral shear, limits rotation, and self-centers the joint (Johnson & Pope, 1976). In spite of these stabilizing elements, the knee joint favors mobility at the expense of stability.

The Menisci

The menisci are dense plates of fibrocartilage located on the superior surface of the tibia, (see Figure 5) one lying on each side of the tibial spine filling the joint space between femur

and tibia (Moore, 1982). They are crescent shaped and triangular in cross-section; being thickest at the periphery (Kapandji, 1970). The extremities of the crescents are known as horns, thus each meniscus has an anterior and a posterior horn (Moore, 1982). The horns of the lateral meniscus lie very close to each other so that the meniscus approximates a circle (Kapandji, 1970). The medial meniscus is more oval shaped and open between the horns (Moore, 1982). Thus both menisci conform to the shape of the respective femoral condyles with the lateral meniscus being somewhat thicker than the medial (Moore, 1982).

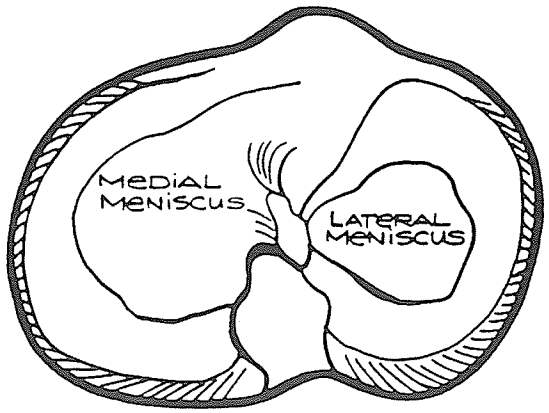

FIGURE 5 SUPERIOR VIEW OF TIBIA AND MENISCI. ADAPTED FROM ARNOCZKY (1983)

The menisci have a number of attachments which have significant functional implications (Kapandji, 1970). These include:

tibia : Both horns of both menisci are anchored to the superior tibial surface near the extremities of the intercondylar eminence while the anterior margins are attached to the anterior tibia via the coronary ligament (Welsh, 1980; Kapandji, 1970).

femur : The posterior horn of the lateral meniscus attaches to the medial femoral condyle via either the anterior menisco-femoral (ligament of Humphery) ligament or the posterior menisco-femoral ligament (ligament of Wrisberg) (Brantigan, 1941; Johnson & Pope, 1976).

capsule : Both menisci are attached to the respective deep capsular ligaments by menisco-femoral and menisco-tibial ligaments with the medial attachments being much stronger and more significant then the lateral (Welsh, 1980).

patella : Fibrous bands (menisco-patellar) connect both anterior horns to the posterior patella (Johnson & Pope, 1976; Kapandji, 1970). The transverse ligament joins the anterior horns and is in turn connected to the patella by strands of the infrapatellar fat pad (Kapandji, 1970).

medial collateral ligament : The medial collateral ligament (MCL) is attached to the medial meniscus through its intimate association with the medial capsular ligaments (Kapandji, 1970; Müller, 1983).

popliteus : A fibrous expansion of popliteus muscle attaches to the posterior border of the lateral meniscus (Kapandji, 1970).

semimembranosus : Semimembranosus muscle extends a fibrous expansion to the posterior horn of the medial meniscus (Johnson & Pope, 1976).

cruciate ligaments : Fibres joining the posterior cruciate ligament (PCL) and the posterior horn of the lateral meniscus constitute the bulk of the menisco-femoral ligament (Kapandji, 1970). ACL fibres insert at the anterior horn of the medial meniscus (Kapandji, 1970).

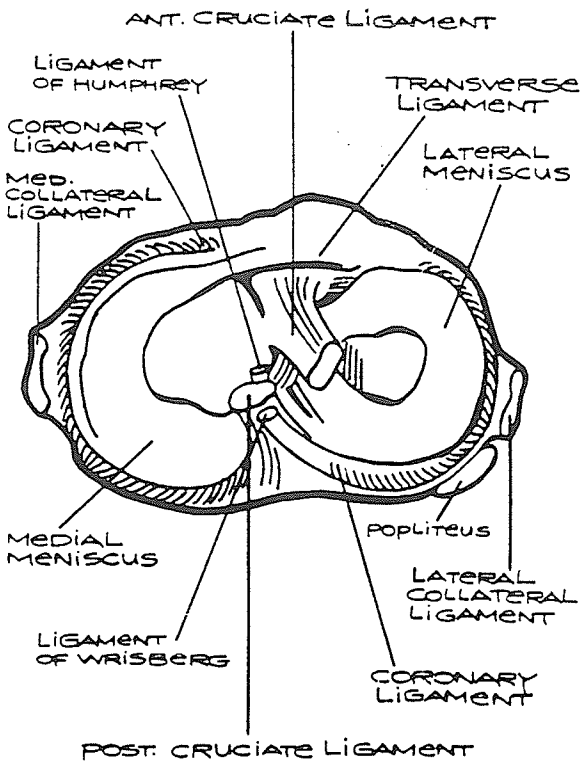

FIGURE 6 SUPERIOR VIEW OF TIBIAL PLATEAU. NOTE RELATIONSHIP OF MENISCI WITH LIGAMENTS.
ADAPTED FROM NORWOOD & CROSS (1977)

The menisci are avascular except for the tibial attachments and the periphery (Warren & Levy, 1983). A synovial fringe supplies nutrients to the superior and inferior surfaces but there is no penetration of the menisci (Warren & Levy, 1983).

Because the menisci are located between the articulating surfaces of the knee joint, they must behave as dynamic structures, altering position to accommodate the profile of the femoral condyles and avoid being trapped, to be crushed or torn (Brantigan, 1941). When the knee joint is moving from an

extended to a flexed position, both menisci move posteriorly (Johnson & Pope, 1976) . Retraction of the lateral meniscus is initiated by the popliteus muscle and is of a greater magnitude than that of the medial meniscus which is controlled by the tendinous expansions of semimembranosus (Kapandji, 1970). As the knee extends, the motion is reversed and both menisci slide forward to cushion the femoral condyles (Johnson & Pope, 1976). This motion has both active and passive components (Kapandji, 1970). The menisci are pushed anteriorly by the motion of the femoral condyles , and pulled forward by contraction of the quadriceps muscle by way of the patello-meniscal fibres and the transverse ligament (Johnson & Pope, 1976). The more mobile lateral meniscus is also pulled anteriorly by the tension developed in the menisco-femoral fibres which is initiated by increased tension in the PCL (Kapandji, 1970). If the knee is hyperextended increasing tension in the transverse ligament prevents excessive separation of the two anterior horns (Brantigan, 1941). Both menisci are displaced during rotational movements in the direction opposite to the movement (ie when the knee rotates laterally the medial meniscus moves posteriorly and the lateral meniscus moves anteriorly; when the knee rotates medially the opposite meniscal movement occurs) (Kapandji, 1970). This motion is mainly passive, although tension in the

menisco-patellar fibres draws one of the menisci forward (Kapandji, 1970). In all meniscal motion there is distortion of the structure due to the tibial attachments at the meniscal horns Kapandji, 1970).

Meniscal function not only includes contributions to mobility and stability but also transmission of loads and absorption of shock (Kapandji, 1970). The menisci increase the contact area between tibia and femur thus distributing the load over a greater area (Warren & Levy, 1983; Henning & Lynch, 1985). The dense fibrocartilage compresses under load, absorbing some of the force that would be transmitted through the joint (Johnson & Pope, 1976). The menisci aid in dispersion of synovial fluid within the joint (Welsh, 1980, Warren & Levy, 1983). Johnson & Pope (1976) have also suggested that the menisci prevent the joint capsule from being pinched in the joint space, but admitted that pinching of the capsule had never been observed following menisectomy. The menisci deepen the tibial articular surface and increase joint congruency (Moore, 1982; Johnson & Pope, 1976). They restrict rotary motion and act as a secondary restraint opposing anterior drawer (Hsieh & Walker, 1976; Wang & Walker, 1974; Warren & Levy, 1983). Brantigan and Voshell (1941) claimed that the menisci cushion hyperextension and hyperflexion of the joint.

Joint Capsule

Like all synovial joints the knee joint is surrounded by a fibrous articular capsule lined with a synovial membrane which secretes synovial fluid (Moore, 1982). The capsule attaches to the upper posterior margins of the femoral condyles and the intercondylar space, blending with the origins of gastrocnemius and reflecting laterally to allow popliteus to pass into the joint (Warwick & Williams, 1973). The posterior tibial attachment lies below the articular border of the tibial plateau, covering the head of the fibula and merging with the popliteal sheath on the lateral aspect (Warwick & Williams, 1973). The anterior capsule blends with the fibrous extensions of the vasti muscles to form patellar retinaculae which attach around the periphery of the patella and reflect inferiorly to the posterior-medial meniscal margins (Warwick & Williams, 1973). The capsule is generally loose and thin to allow a full range of motion but is extensively reinforced medially and laterally to increase stability (Moore, 1982).

Muscles Crossing the Knee Joint

Muscle function includes initiation of movement, maintenance of body position and support of joints (Van De Graff, 1984). The contractile properties of muscle tissue allow muscles to initiate and carry out movement (Van De Graff, 1984; Tortora & Anagnostakos, 1981). Muscles are

referred to as dynamic stabilizers because they are able to adjust tension in response to both cortically and proprioceptively initiated signals (McLuskey & Blackburn, 1980). Muscles crossing the knee joint are vital for both joint mobility and stability (Tortora & Anagnostakos, 1981).

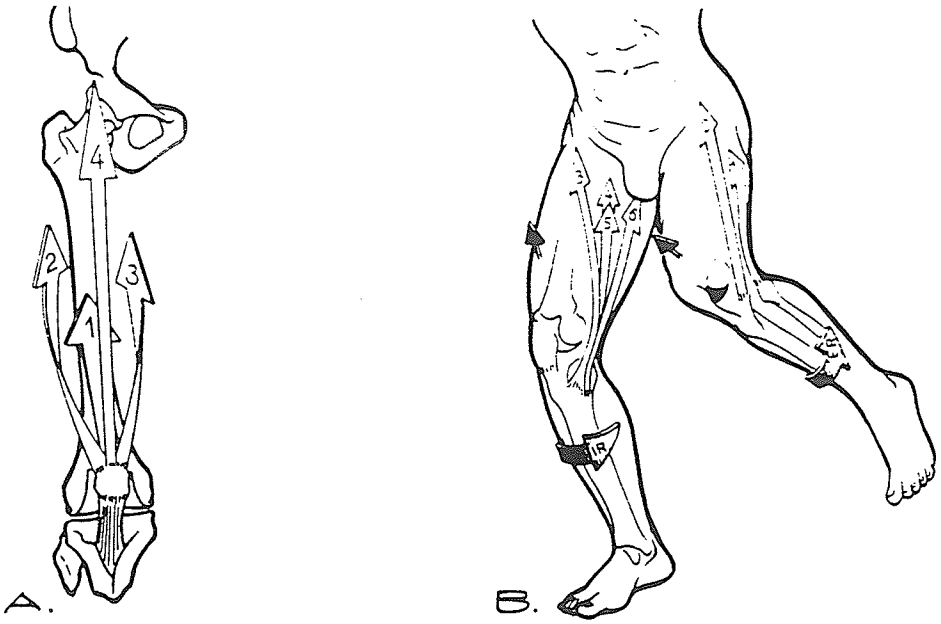

FIGURE 7

A. QUADRICEPS GROUP
1. VASTUS INTERMEDIUS
2. VASTUS LATERALIS
3. VASTUS MEDIALIS
4. RECTUS FEMORIS

B. MUSCLES CROSSING THE KNEE JOINT.
1. BICEPS FEMORIS
2. TENSOR FASCIA LATA
3. SARTORIUS
4. SEMITENDINOSUS
5. SEMIMEMBRANOSUS
6. GRACILIS

ADAPTED FROM KAPANDji (1970)

anterior aspect . Four individual muscles, rectus femoris, and three vasti; medialis, intermedius, and lateralis combine to form the quadriceps femoris (Tortora & Anagnostakos, 1981). The quadriceps attaches proximally on the pelvis (anterior inferior iliac spine) and the femoral shaft, crosses the knee joint anteriorly, and inserts on the tibial tuberosity of the proximal anterior tibia via the patellar tendon (Tortora & Anagnostakos, 1981). The principal actions of the quadriceps are flexion of the hip and extension of the knee (Tortora & Anagnostakos, 1981). The oblique portion of vastus medialis also contributes to internal rotation of the tibia (Müller, 1982). Tension in the quadriceps limits forward motion of the femur relative to the tibia and the elasticity of the muscle tissue helps distribute shock loads transmitted through the knee (McLeod & Hunter, 1980). The patella is a sesmoid bone enclosed in the quadriceps tendon which serves to increase the efficiency of the quadriceps by shifting the line of pull anteriorly (Kapandji, 1970).

medial aspect . The pes anserines group, comprised of sartorius, gracilis, and semitendinosus crosses the medial aspect of the knee joint (Tortora & Anagnostakos, 1981). Originating on the medial pelvis and inserting on the medial tibia, these muscles act as hip flexors, hip extensors, knee

flexors and internal rotators of the flexed knee (Johnson & Pope, 1976).

posterior-medial aspect . Semimembranosus stretches from an origin on the ischial tuberosity on the lower posterior pelvis to an insertion which spreads over the posterior tubercle of the tibia, the fascia of the medial head of gastrocnemius, the posterior horn of the medial meniscus, and the oblique popliteal ligament (Johnson & Pope, 1976). The principal actions of semimembranosus are hip extension, knee flexion and internal rotation of the tibia on the femur (Johnson & Pope, 1976). It is also responsible for retraction of the medial meniscus during knee flexion (Müller, 1982).

posterior aspect . Gastrocnemius, a two headed muscle, originates on the posterior femoral condyles and intercondylar notch, crosses the posterior knee joint, and inserts on the posterior calcaneous via the achilles tendon (Moore, 1982). This biarticular muscle acts as a secondary knee flexor, a principal plantarflexor, and supports the posterior capsule (Moore, 1982).

posterior-lateral aspect . The popliteus muscle originates on the medial border of the posterior tibia and runs laterally and superiorly to a diffuse insertion (Johnson & Pope, 1976). Fibres of the popliteus insert on the lateral condyle of the femur deep to the lateral collateral ligament and the joint

capsule (intracapsular insertion), the posterior horn of the lateral meniscus, and the arcuate ligament (Johnson & Pope, 1976). The primary function of popliteus is to medially rotate the tibia on the femur, thus unlocking the screw home mechanism in order to initiate knee flexion (Johnson & Pope, 1976). Popliteus also acts as a secondary knee flexor, retracts the lateral meniscus during knee flexion, and augments both the posterior capsule and the PCL (Johnson & Pope, 1976; Müller, 1982).

lateral aspect . The iliotibial band (ITB) originates as the tensor fascia lata at the anterior superior iliac spine, crosses the axis of the knee joint, and inserts on Gerdy's tubercle on the lateral tibia (Johnson & Pope, 1976). Because the ITB crosses the axis of the joint it functions as both a flexor (40-145o of flexion) and an extensor (0-30o of flexion) of the knee (Johnson & Pope, 1976). The ITB also augments the posterior-lateral capsule (Welsh, 1980). Originating on the ischial tuberosity and the posterior shaft of the femur, biceps femoris crosses the knee joint to insert on the fibular head, distal lateral collateral ligament and posterior capsule (Johnson & Pope, 1976). Principal functions of the biceps femoris are hip extension, knee flexion, and external rotation of the tibia when the knee is flexed (Moore, 1982). Biceps femoris reinforces the

lateral capsule, the lateral collateral ligament, and the anterior cruciate ligament (Johnson & Pope, 1976).

Ligaments

Ligaments are bundles of collagen fibres which connect bone to bone across joints (Tortora & Anagnostakos, 1981). They are usually described as passive stabilizers because they have no contractile properties and provide restraint only when stretched (McLeod & Hunter, 1980; Andrews & Axe, 1985). Ligaments tend to be pliant and flexible but only slightly elastic as the bundles of collagenous fibres are interspersed with only a few elastic fibres (approximately 10%) (Warwick & Williams, 1973).

The fibrous bundles are oriented to provide resistance to tensile stress (Akeson, Frank, Amiel, & Woo, 1982). In the relaxed state the fibres are arranged in a crimped fashion which progressively straightens and stretches under tensile load (Akeson, Frank, Amiel, & Woo, 1982). As the load increases more fibres are recruited, resulting in both early and increasing resistance to the tensile loading (Akeson, Frank, Amiel, & Woo, 1982). The collagen fibres either interdigitate directly with bony tissue or they connect to a transitional zone composed of fibrocartilage and mineralized fibrocartilage (Arnoczky, 1983; Akeson, Frank, Amiel, & Woo, 1982). The combination of attachments aids in the dissipation of forces

and reduces the incidence of ligament failure at the site of insertion (Akeson et al., 1982).

Innervation of ligaments is composed of sensory branches of the nerves supplying the muscles surrounding the joint in question (Akeson, Frank, Amiel, & Woo, 1982). Sensory receptors found in ligaments include pain receptors and proprioceptors (Akeson, Frank, Amiel, & Woo, 1982 ; Abbot, Saunders, Bost, & Anderson, 1944). Increased tension in the proprioceptors initiates a reflex arc with antagonistic muscles to prevent overloading, either by the agonists or external forces (Akeson, Frank, Amiel, & Woo, 1982). The splinting action of muscles surrounding a joint following ligamentous injuries suggests that increased activity of pain receptors can also initiate dynamic responses (Andrish, 1985). The activity of the sensory receptors and the role of the ligament as a link in a kinetic chain implies that the concept of the ligament as passive restraint may be an oversimplification; that the ligament behaves more as a dynamic structure even though it has no contractile properties (Andrish, 1985).

Ligaments have two basic functions; they guide the joint through its normal ROM and prevent unphysiologic (ie abnormal) motion (Abbot, Saunders, Bost, & Anderson, 1944; Cabaud, 1983). Lewis, Lew, Shybut, Jasty, and Hill (1982)

differentiated ligament function for low load and high load conditions. A low load situation requires maintenance of correct joint kinematics while the high load condition involves stabilization against loads which act too quickly or are too large for muscles to resist (Lewis, Lew, Shybut, Jasty, & Hill,1982). High load conditions are often traumatic situations (Lewis, Lew, Shybut, Jasty, & Hill,1982). Since ligaments can work only through tension, the amount of tension in a ligament indicates the contribution of that ligament to joint function in any given situation (Ellison & Berg, 1985).

intrinsic or capsular ligaments . The joint capsule is augmented medially and laterally by the intrinsic capsular ligaments which are simply localized thickenings of the capsule (McCluskey & Blackburn, 1980). The capsular ligaments are attached to the periphery of the meniscus and are divided by said structure into menisco-tibial and menisco-femoral ligaments (McCluskey & Blackburn,1980). The medial capsular ligaments are larger, stronger, and more securely attached to the meniscus (Hughston , Andrews, Cross & Moschi, 1976; McCluskey & Blackburn, 1980). Fibres of the medial capsular ligaments blend with those of the medial collateral ligament (MCL) and are often identified as the deep layer of the MCL (Hughston , Andrews, Cross & Moschi, 1976; McCluskey & Blackburn, 1980). The posterior third of the medial capsular ligament forms the posterior oblique ligament while the lateral counterpart blends with the deep portion of the arcuate complex (McCluskey & Blackburn, 1980). The coronary ligament, comprised of capsular folds, loosely attaches the remainder of the meniscal boundaries to the tibia (McCluskey & Blackburn, 1980).

external or extrinsic ligaments . Extrinsic ligaments are separate structures which reinforce the capsule on all sides (Moore, 1982).

Anteriorly, the ligamentum patellum, which can also be described as an extension of the patellar tendon, stretches from inferior patella to the anterior tibia (tibial tuberosity) and limits forward movement of the femur on the tibia (Mcleod & Hunter, 1980).

The tibial or medial collateral ligament (MCL) is a broad flat band passing inferiorly from the medial epicondyle of the femur to the medial tibial shaft (Moore, 1982). This phylogenic analogue of the adductor magnus tendon blends superiorly with the medial capsular ligaments and inferiorly with the insertion of the pes anserinus, superficial to the insertion of semimembranosus (Welsh, 1980; Abbot, Saunders, Bost, & Anderson, 1944). The MCL has a triangular shape that causes the anterior portion to become taut in flexion and the posterior segment to become taut as the joint extends (Abbot, Saunders, Bost, & Anderson, 1944). The entire ligament is slack at midflexion (Abbot, Saunders, Bost, & Anderson, 1944). The MCL acts as the prime medial stabilizer resisting valgus stress, a secondary restraint of anterior drawer, and a restraint to anteromedial rotation of the tibia on the femur (Andrish, 1985). The relationship of the MCL to the medial meniscus is of clinical importance (Moore, 1982).

A cord like structure provides the major ligamentous support to the lateral aspect of the knee joint (Abbot,

Saunders, Bost, & Anderson, 1944; Welsh, 1980). The lateral collateral ligament (LCL) runs inferiorly from the lateral epicondyle of the femur to the posterior head of the fibula where it splits the insertion of the biceps femoris tendon (Welsh, 1980). The ligament is slack in all positions of flexion and taut only when the leg is fully extended (Abbot, Saunders, Bost, & Anderson, 1944). The joint capsule and the lateral meniscus are not attached to but in fact are separated from the LCL by the popliteus tendon (Welsh, 1980). Functions of the LCL include resistance to varus stress, restraint of anterior drawer, and prevention of hyperextension (Mcleod & Hunter, 1980; Abbot, Saunders, Bost, & Anderson, 1944; Brantigan & Voshell, 1941).

The posterior third of the medial capsular and medial collateral ligaments comprise the posterior oblique ligament which supports the posteromedial aspect of the joint (Hughston , Andrews, Cross & Moschi, 1976; Blackburn & Craig, 1980; McCluskey & Blackburn, 1980). An expansion of the semimembranosus tendon serves as the ligament of Winslow (Welsh, 1980). Stretching superiorly from the posterior tibia to the popliteal surface of the femur above the lateral condyle ,this ligament reinforces the posterior capsule (Welsh, 1980). Linking the posterior head of the fibula and the posterior surface of the lateral femoral condyle, the arcuate ligament is

a Y-shaped band of fibres superficial to the popliteal tendon (Moore, 1982; Hughston , Andrews, Cross & Moschi, 1976). The arcuate complex augments the posterolateral capsule (McCluskey & Blackburn, 1980).

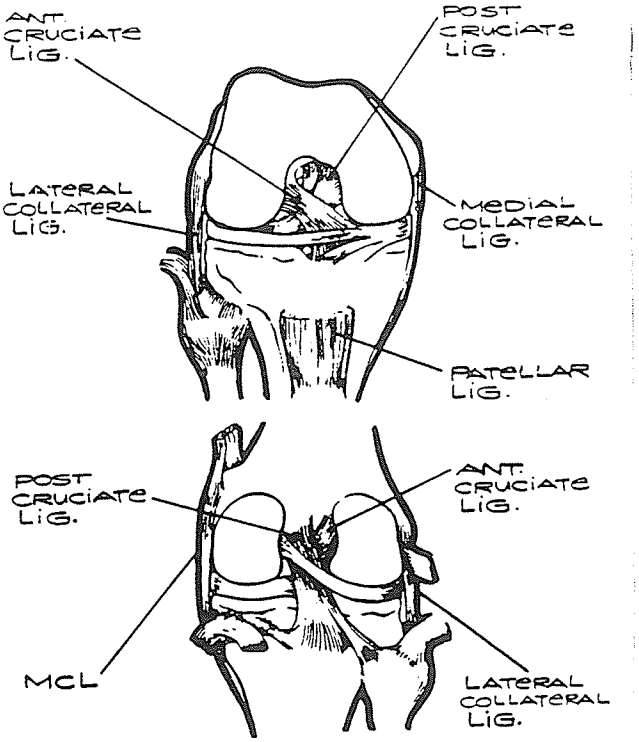

FIGURE 8
THE LIGAMENTS OF THE KNEE JOINT.
ADAPTED FROM GRANT'S ATLAS
(ANDERSON, 1978)

Intra-articular ligaments . The anterior and posterior cruciate ligaments link the femur and tibia between the medial and lateral condylar articulations (Moore, 1982). The ligaments cross near the center of the joint (cruciate = cross), and are differentiated by their tibial attachment (Moore, 1982). The cruciates are intra-articular but extrasynovial, ie

they lie inside the joint but outside the joint capsule (Moore, 1982).

The ACL measures roughly three to four cm. in length by one cm. in diameter and its fibres run posteriorly, superiorly, and laterally from the tibial insertion (Kennedy, Weinberg, & Wilson, 1974; Norwood & Cross, 1977). The site of attachment is a fossa on the tibial plateau, anterior and lateral to the tibial spine (Arnoczky, 1983, Ellison & Berg, 1985). The ACL attaches to the femur in a fossa on the medial surface of the lateral condyle (Arnoczky, 1983; Marshall, Rubin, Wang, Fetto, and Arnoczky, 1978; Girgis et al., 1975). The functional anatomy of the anterior cruciate ligament will be discussed in appropriate detail in a separate section.

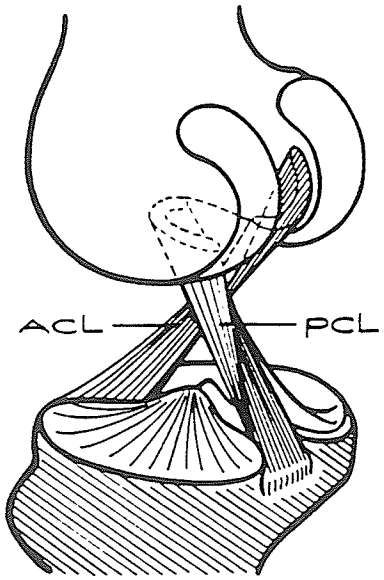

FIGURE 9
THE CRUCIATE LIGAMENTS
ADAPTED FROM KAPANDJI (1970)

The posterior cruciate ligament (PCL) which is the largest and the strongest ligament in the knee, consists of a broad flat band stretching superiorly, anteriorly, and medially from the posterior intercondylararea of the tibia and the posterior horn of the lateral meniscus to the lateral surface of the medial femoral condyle (Welsh, 1980). The attachment sites are curved; the concave surface facing the midline of the joint (Abbot, Saunders, Bost, & Anderson, 1944; Girgis, Marshall, & Monajem, 1975).

The ligament is divided longitudinally into two discrete functional bundles; the anterolateral (ALB) and the posteromedial bundles (PMB) (Andrews & Axe, 1985; Girgis et al., 1975). The two bundle structure and the curved shape of the insertions insure that some part of the ligament remains taut throughout the entire ROM of the joint (Andrews & Axe, 1985; Abbot, Saunders, Bost, & Anderson, 1944). The anterolateral bundle is taut during knee flexion and tension increases in the posteromedial bundle as the leg extends (Andrews & Axe, 1985).

The function of the PCL is not totally understood. The position of the ligament and the orientation of its fibres dictate that the PCL prevents forward subluxation of the femur on the tibia and conversely, posterior movement of the tibia relative to the femur (Butler, Noyes, & Grood, 1980; Mains,

Andrews, & Stonecipher, 1977). Hughston et al. (1976) and Detenbeck (1974) claimed that the PCL is the principal stabilizer of the knee and therefore the key to joint integrity. As such, the PCL acts as the axis for rotary motion and also guides the screw home mechanism (Hughston, Andrews, Cross & Moschi, 1976; Detenbeck, 1974; Mains, Andrews, & Stonecipher, 1977). Müller (1983) noted that the cruciate ligaments worked in concert to maintain spatial relationships between the tibia and femur, especially to control the rolling/gliding mechanism.

In the knee joint normal motion and stability depend on complex interactions between ligamentous structures (Gollehon, Torzilli, & Warren, 1987). The ligaments and capsule act as a system in which one structure serves as a primary stabilizer for each plane of motion, while some of the others act as secondary stabilizers and contribute to a lesser degree (Noyes, Grood, Butler, & Malek, 1980). Consequently few ligaments bear sole responsibility for a particular function and conversely, each ligament generally serves more than one function (Müller, 1982).

Nerve Supply to the Knee Joint

The posterior joint is innervated by branches of the posterior tibial nerve and a terminal branch of the obturator nerve (Kennedy, Alexander, & Hayes, 1982). The anterior portion of the joint is innervated superiorly by articular branches of the nerve to the vasti and inferiorly by the lateral articular recurrent peroneal nerve and the infrapatellar branch of the saphenous nerve (Kennedy, Alexander, & Hayes, 1982). Kennedy et al. (1982) hypothesized that loss of knee joint innervation compromises neuromuscular control of knee joint stability which can lead to repetitive injury and degeneration of the joint.

Other Structures in the Knee Joint

There are a number of bursae and fat pads surrounding the knee joint whose function is to separate and protect various elements of the joint (Blackburn & Craig, 1980). Although they are not directly involved with joint function, damage to one of these structures and subsequent loss of protection can compromise function (McLeod & Hunter, 1980).

The Anterior Cruciate Ligament

Structure

The ACL measures roughly three to four cm. by one cm. and its fibres run posteriorly, superiorly, and laterally from the tibial insertion (Kennedy, Weinberg, & Wilson, 1974; Norwood & Cross, 1977). Description of ACL structure varies considerably. Ellison and Berg (1985), Blackburn and Craig (1980), Marshall, Rubin, Wang, Fetto, and Arnoczky (1978), and Furman, Marshall, and Girgis, (1976) described two distinct bundles of fibres, a small anteromedial band (AMB) and a larger posterolateral bundle (PLB). The two segments of the ligament behave as two fibrous plates joined by a flexible material which allows each plate to act independently (Marshall, Rubin, Wang, Fetto, & Arnoczky, 1978; Furman et al., 1976). Norwood and Cross (1979, 1977) and Blackburn and Craig (1980) claimed that the the ACL is composed of three distinct segments; the AMB, the PLB, and an intermediate bundle. The AMB is the longest of the three while the PLB is the shortest and most vertical (Norwood & Cross, 1979). These observations are not so much conflicting as they are confusing. The flexible fascia between the two bundles might in fact be equivalent to a third bundle. Norwood and Cross

(1979) also noted that the synovial folds must be reflected to observe all the bundles. Consequently the differences observed are likely due to either interpretation of observations or quality of dissection. Arnoczky (1983) and Welsh (1980) suggested that the concept of independent functional units in the ACL is an oversimplification, the ACL is a continuum of fibres passing from a cord-like origin to a broad flat insertion.

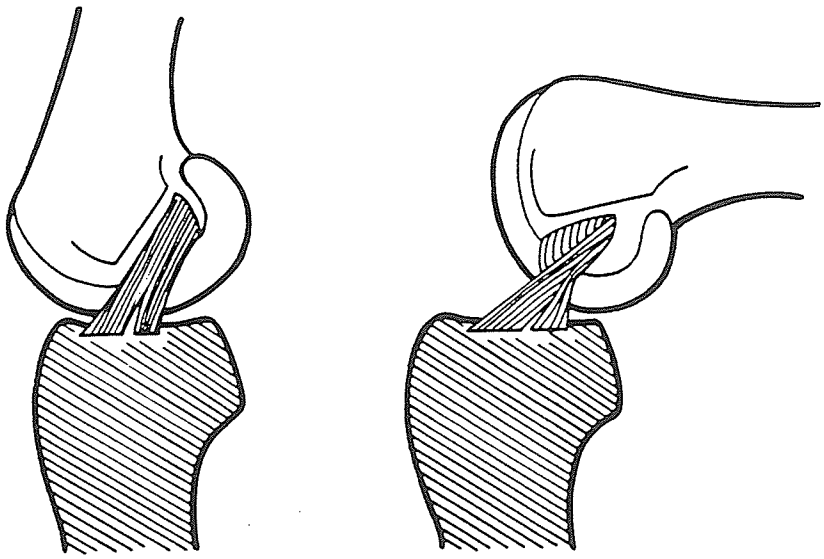

FIGURE 10
THE ACL SHOWING ATTACHMENTS, ORIENTATION AND THE TWISTING ACTION AS THE KNEE FLEXES.
ADAPTED FROM KAPANDji (1970)

The microanatomy of the anterior cruciate ligament (ACL) is identical to that described for ligaments in the previous section.

Femoral Attachment

The ACL attaches to the femur in a fossa on the medial surface of the lateral condyle (Arnoczky, 1983; Marshall, Rubin, Wang, Fetto, and Arnoczky, 1978; Girgis et al., 1975). The attachment is crescent shaped with a straight anterior and a curved posterior border (Arnoczky, 1983; Ellison & Berg, 1985; Girgis et al., 1975). Norwood and Cross (1979) described the attachments of the various bundles as follows; AMB posterior and superior, PLB anterior and inferior, and the intermediate bundle lying in between.

Tibial Attachment

Forming a triangle or wedge pointing posteriorly, the tibial attachment is the larger and stronger of the two (Arnoczky, 1983, Ellison & Berg, 1985). The site of attachment is a fossa on the tibial plateau, anterior and lateral to the tibial spine (Arnoczky, 1983, Ellison & Berg, 1985). Arnoczky (1983) reported that the AMB attaches near the anterior medial base of the tibial spine with some fibres passing inferior to the transverse meniscal ligament and blending with the anterior attachment of the lateral meniscus. The PLB attaches anterior and lateral to the tibial spine while the intermediate bundle

attaches between AMB and PLB (Arnoczky, 1983). Norwood and Cross (1979), Girgis et al. (1975), and Welsh (1980), disregarded the existence of discrete bundles and claimed that the ligament attaches as a collection of fascicles which fans out over a broad area.

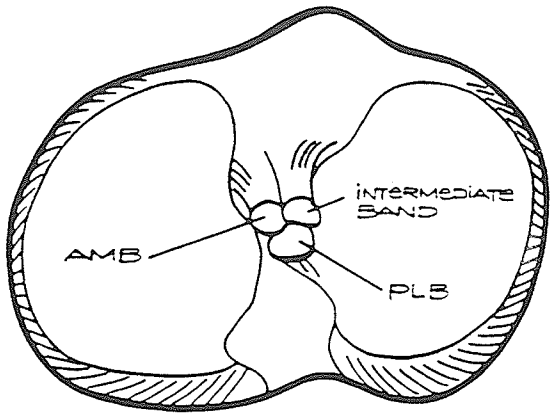

FIGURE 11
TIBIAL ATTACHMENT OF ACL SHOWING LOCATION OF SEPERATE BUNDLES.
ADAPTED FROM ARNOCZKY (1983)

Nerve Supply

The ACL is innervated by branches of the tibial nerve which penetrate the posterior joint capsule (Arnoczky, 1983). Kennedy et al. (1974) observed neural elements in all regions of the ACL, especially in proximity to the tibial attachments and in the vascular synovial coverings. These included proprioceptors, free nerve endings resembling pain fibres, and paravascular fibres which likely control vasomotor function

(Kennedy, Weinberg & Wilson, 1974). Kennedy et al. (1982) reported Golgi-like receptors on the ligament surface. Schutte, Dabezies, Zimny, and Happel (1987) identified a number of mechanoreceptors found throughout the substance of the ACL. Two types of slow adapting Ruffini endings which respond with prolonged discharge to changes in tension, motion, joint position, and joint angle, signal the proximity of the joint to its functional limits (Schutte, Dabezies, Zimny, & Happel, 1987). Rapid adapting Pacinian corpuscles respond to joint movement (Schutte, Dabezies, Zimny, & Happel, 1987). Schutte et al. (1987) also observed the free nerve endings which respond to pain stimuli. The presence of the neural structures indicates that the ACL is not simply a passive restraint, it also possesses dynamic properties in the form of feedback loops involving the muscles surrounding the knee joint (Andrish, 1985).

Blood Supply to ACL

The vascular supply to the ACL is of soft tissue origin rather than through the osseous attachments (Arnoczky, 1985; 1983). Arnoczky (1985; 1983) stated that branches of the popliteal, middle genicular, and lateral inferior genicular arteries descend along the the dorsal surface of the ligament to penetrate the synovial folds covering the ligament and anastamoze with endoligamentous vessels. Kennedy et al.

(1974) claimed that the blood supply is adequate to support healing of damaged tissues.

Function of ACL

Introduction

The function of the ACL is neither clearly defined nor totally understood (Welsh, 1980; Norwood & Cross, 1979; Kennedy, Weinberg, & Wilson, 1974). Early studies attributed function to the ACL on the basis of the gross anatomy of the ligament (Arnoczky, 1983). Brantigan and Voshell (1941) exemplified this approach claiming that the position and spatial orientation of the ACL dictated that the ligament could not help but oppose anterior displacement of the tibia on the femur. However, the knee joint and the ACL are components in an intricate biomechanical system. Norwood and Cross (1977) and Detenbeck (1974) observed that as the joint passes from extension to flexion the distance between tibial and femoral attachments of the ACL decreases. Girgis et al. (1975) partially agreed, reporting that the distance between the posterior attachments decreased but, that the distance between anterior insertions increased. Simultaneously, the spiral arrangement of the fibres and the curvature of the insertions cause the ligament to wind as the joint flexes (Girgis, 1975; Arnoczky, 1983; Ellison & Berg, 1985). Detenbeck (1974) concluded that prediction of ligament function based solely on anatomy or spatial orientation is

difficult and often leads to inconsistency if not outright contradiction. However, anatomical description leads to speculation and the formation of testable hypotheses.

Procedures used to identify ACL function

Ellison & Berg (1985) identified a number of procedures used to test ACL function. These included:

a. observation of tension in ACL under different conditions

b. observation of ACL damage as the joint moves past normal ROM

c. observation of changes in joint stability after severing the ACl

d. correlation of ACL injury and loss of function

Tension in ACL. Because ligaments do not have contractile elements they can only apply forces through tension (McLeod & Hunter, 1980). The tensile state of a ligament should reflect the contribution of that ligament to joint function in a given situation (Ellison & Berg, 1985). Consequently, the tensile state of the ACL fibres throughout the repertoire of joint excursions has been used to infer ACL function (Ellison & Berg, 1985). Kennedy, Hawkins, and Willis (1977) noted that a significant portion of the research (see table 1) in this area is of a qualitative nature, utilizing subjective interpretation of tension in the ACL based on observation and palpation rather than objective measurement (Kennedy, Hawkins, & Wilson,

1977). Qualitative data is difficult to compare, resulting in inconsistency and contradiction.

Methodological differences in the tension analysis have also contributed to the confusion. Several researchers have reported that the discrete bundles of fibres which comprise the ACL are oriented differently and can function as independent structures (Arnoczky, 1983; Welsh, 1980; Furman et al.,1976). Others (Ellison, 1980; Kennedy, Weinberg, & Wilson, 1974) have approached the ligament as a single unified structure. These approaches inevitably contribute conflicting or contradictory results

Table 1
Tension Observed in ACL

AUTHOR	LIMB POSITION	LIG SEGMENT	ROTATION	TENSION
Brantigan & Voshell (1941)	all	all	0	+
	all	all	internal	++
	hyperext	all	0	+
Abbot et al. (1944)	ext	all	0	+
Kennedy et al. (1974)	ext	all	0	+
	5-20°	all	0	+
	20-70°	all	0	-
	40°	all	0	- -
	70-90°	all	0	+
	all flexion	all	internal	+
Girgis et al.(1975)	flex	AMB	0	+
	ext	PLB	0	+
	ext	all	internal	+
	ext	all	external	±
	flex	all	internal	-
	flex	all	external	-
Norwood & Cross (1977)	flex	AMB	0	-
	flex	IB	0	-
	all	PLB	0	+
Marshall et al. (1978)	flex	AMB	0	+
	ext	PLB	0	+
Ellison (1980)	35°	all	0	- -
	ext	all	0	+

In light of the above, some researchers have attempted to measure tension in the ACL. Detenbeck (1974) employed a strain gauge to measure, ACL tension at different joint positions in amputated specimens. The instrument was mounted so as to measure strain in the whole structure (Detenbeck, 1974). Kennedy et al. (1977) measured tension as the joint moved through its ROM using a less intrusive strain gauge placed in the midsubstance of the ligament. Findings were related to various mechanisms of injury rather than to function but the measured results were similar to those of Detenbeck. Lewis et al. (1982) installed force transducers on all major ligaments, applied external forces to the joint and measured the changes in tension. The buckle type strain gauge used was unable to accommodate the entire ACL, therefore the authors installed a pair; the first on the AMB and a second on the PLB (Lewis, Lew, Shybut, Jasty, & Hill,1982). Although the experimental design involving two ligamentous segments was used due to instrument limitation, the results suggest that it is a valid approach since there appear to be functional differences between the two bands.

The three studies cited utilized cadaver specimens and this methodology raises questions of test validity. Nicholas (1977) noted that generalization of function from cadaver specimens to the living knee joint is a dubious process due to loss of

dynamic properties, loss of proprioceptive function, and changes in tissue properties. Henning, Lynch, and Glick (1985) conducted an in vivo study secondary to exploratory arthroscopic surgery. Strain gauges were implanted in the AMB to measure tension in the ligament during clinical stress tests and activities which would be included in a typical rehabilitation program (Henning, Lynch, & Glick, 1985). They reported that the ACL was instrumental in preventing anterior translation of the tibia. In retrospect, the true value of this study may lie with the protocol and instrumentation which allowed the authors to take measurements in vivo.

Tension studies invite questions with regard to external validity. ACL function can only be inferred from the measures of tension because increases in tension may not always equate with increased function. Optimal function in one situation is often sacrificed to insure adequate function in others. Consequently increased tension might in some cases detract from optimal function rather than enhance it.

Ligament failure when normal ROM exceeded. Rather than measure the tension of the ligament under various conditions, other studies have subjected the structure to excessive or abnormal forces so as to increase tension to pathological levels. Ultimately ligament failure occurred, and the structure that failed was deemed to have opposed the applied force.

Kennedy et al. (1974) and Norwood and Cross (1979) forced cadaver joints into extreme hyperextension and observed the structural damage. If the distraction forces were large enough the joint was totally dislocated and all structures were disrupted (Kennedy, Weinberg, &Wilson, 1974). In such cases, one assumes the order of failure is as important as the structures damaged. The sequential disruption of structures illustrates the concept of primary and secondary stabilizers which must be differentiated. This methodology involves pathological forces, thus raising questions as to whether ligament function under these conditions differs from normal function. The results of these tests have been used to suggest mechanisms of injury as well as ligament function (Norwood and Cross, 1979).

Changes in joint laxity after severing the ACL. Tension studies fail to establish a cause and effect relationship between ligament tension and function as function can only be inferred from changes in tension. A cause and effect relationship between the ligament and a functional variable would identify ligament function with a greater degree of certainty. The most commonly used protocol involves measurement of joint laxity (displacement of the joint resulting from an applied force), bisection of the structure in question (the ACL) and remeasurement of joint laxity. Note

that laxity refers to any joint motion, normal or abnormal (Markolf, Kochan, & Amstutz, 1984). There are many variations of this protocol. Brantigan and Voshell (1941), Markolf, Mensch, and Amstutz (1976), Girgis (1975), Mains et al. (1977), and Sullivan, Levy, Sheskier, Torzilli, and Warren (1984), treated the ACL as a single structure. Furman et al. (1977) on the other hand examined the fibrous bundles which comprise the ACL, individually and varied the sequence of cutting the ligaments. Conversely, the ACL was the final cut in the tests conducted by Gollehon et al. (1987). Although everyone tested the extended knee, the test positions in flexion varied and the forces used to demonstrate joint laxity were also varied.

All the studies cited to this point tested the ACL in an unloaded state, but Wang and Walker (1974), Hsieh and Walker (1976), Lipke, Janecki, Nelson, McLeod, Thompson, Thompson, and Haynes (1981), and Markolf, Bargar, Shoemaker, and Amstutz (1981) attempted to simulate the compressive forces due to gravity and to muscle contraction in their respective tests.

Butler et al (1980) claimed that the order of cutting the ligaments often determined the outcome of the tests. Stability of the joint in each test was partially dependent on interaction of the remaining ligaments. Changing the order of

cutting the ligaments changed the nature of the interaction and influenced test results. In order to avoid this problem Butler et al. (1980) first measured the force required to produce a given laxity in a given direction, severed the ACL and remeasured the force required to produce an identical laxity. The difference between the two forces represented the contribution of the ligament to joint stability for the selected motion.

Whether or not one subscribes to Butler's hypothesis one must concede that studies of this nature must be performed in vitro. Nichols, Lipke, and Markolf (1981) conceded that cadaver specimens lack the contributions of dynamic tissues, and consequently the interaction between joint structures is significantly altered. Furthermore, normal joint kinematics have been altered by the dissections and the instrumentation (Lipke, Janecki, Nelson, McLeod, Thompson, Thompson, & Haynes, 1981). Differences in protocol make comparison of results difficult, and in some instances there was a failure to distinguish between directions of movement in the plane of motion in question (Markolf, Mensch, and Amstutz, 1976). Small sample sizes were the rule rather than the exception and statistics were poorly explained, incomplete or absent in a number of papers.

Correlation of loss of function and ACL injury. Norwood and Cross (1979) alleged that the only way to evaluate the contribution of the ACL to functional stability is to correlate ACL lesions (confirmed by surgery) with clinical evaluations. Unfortunately, combined injuries may confound the results and isolated injuries to the ACL are relatively rare (Markolf, Kochan, & Amstutz, 1984). Consequently, comparison of disimilar injuries and small sample sizes are common. Even if the methodology is sound and the sample size adequate, causal factors for functional changes can only be inferred.

Review of the test procedures used to determine ACL function has revealed a number of deficiencies, some of which are inherent to research involving human subjects and some of which are due to error. Correction of these deficiencies is beyond the scope of this paper, the intent being to demonstrate the difficulty faced in defining ACL function.

Function

Ellison and Berg (1985) and Marshall et al. (1978) reviewed the available literature and attributed the following functions to the ACL:

a. prevents anterior displacement of the tibia relative to the femur

b. limits hyperextension

c. opposes tibial rotation (internal and external)

d. secondary stabilizer in varus and valgus stresses

e. controls screw home and rolling gliding mechanisms

Opposition to anterior displacement of the tibia. Henning, Lynch and Glick (1985) reported that measured tension in the AMB increased in response to isometric contraction of the quadriceps at various joint angles. Tension in the ACL also increased when a Lachman test (see page 57) was performed (Henning, Lynch, & Glick, 1985). The ACL apparently resisted anterior translation of the tibia in relation to the femur in response to both intrinsic and extrinsic forces. Lewis et al. (1982) also observed increased tension in the AMB of cadaver specimens when anterior force was applied to the tibia. This effect was reduced as the joint was moved through progressive increments of flexion.

After severing the ACL, Girgis et al. (1975) Hsieh & Walker (1976) Markolf et al. (1976) Mains et al. (1977) Brantigan and

Voshell (1941) Furman et al. (1976) and Sullivan et al. (1984) observed increasedanterior translation of the tibia in both flexed and extended positions. Brantigan and Voshell (1941) claimed that the greatest change occurred during flexion. Sullivan et al. (1984) agreed, specifying a position of 30° flexion. Markolf et al. (1976) asserted that the greatest change occurred when the knee was extended but they did not distinguish between anterior and posterior motion. Furman et al. (1976) observed an increase in anterior drawer in flexed positions (45°, 90°) if the AMB were cut. Anterior drawer increased in the extended position if the PLB was severed. Hsieh & Walker (1976) claimed that the ACL provided greater anterior stability than any other structure. Butler et al. (1980) concurred having measured the contribution of the ACL to be 85% of the forces restraining anterior drawer. There were no changes in anterior drawer until some part of ACL was cut (Furman, Marshall, & Girgis, 1976) and the medial structures had limited capability to restrain anterior displacement of tibia on femur only after ACL was cut (Sullivan, Levy, Sheskier, Torzilli, & Warren, 1984). Mains et al. (1977) observed that severing the anterior cruciate increased anterior drawer only if the joint was in a position of neutral rotation. If the knee was rotated there was no change after the ligament was cut (Mains, Andrews, & Stonecipher, 1977).

Markolf et al. (1984) compared ACL deficient knees to the normal contralateral joints and observed an increase in anterior-posterior laxity. The magnitude of the increase was greatest at 20º flexion. Daniel, Stone, Sachs, and Malcom (1985) observed similar results at 30º. Hughston et al. (1976) disagreed alleging that anterior laxity correlated with injuries to the medial capsule.

Note that by opposing anterior drawer the ACL acts in an antagonistic role to the quadriceps action which tends to pull the tibia forward during leg extension (Feagin & Lambert, 1985; Feagin, 1979).

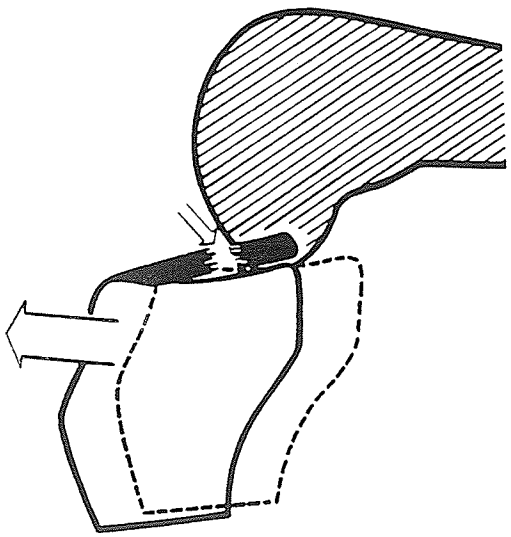

FIGURE 12
ANTERIOR TRANSLATION OF THE TIBIA FOLLOWING BISECTION OF THE ACL.
ADAPTED FROM KAPANDJI (1970)

hyperextension. The simplest approach has been to estimate the tensile state of the ACL when the knee joint is in various positions (see table 1). Such observations are less than reliable due to their subjective nature and therefore measurement of tension is desirable.

Detenbeck (1974) recorded maximal tension at terminal extension, decreasing tension as the knee flexed, and increasing tension at approximately 135o and beyond. The decrease in tension as the joint flexed was attributed to unwinding of the ligament fibres and reduction of the distance between the osseous attachments (Detenbeck, 1974). Detenbeck (1974) concluded that the ACL inhibits hyperextension, but that the PCL controls the screw home mechanism even though the ACL exhibits more tension in the extended position. Kennedy et al. (1977) obtained similar results claiming that the ligament was most lax at 35º flexion and exhibited maximal tension at the extremes of flexion and extension. Lewis et al. (1982) observed that applied forces increased tension in the AMB as the joint approached the extended position and that neither portion of the ligament exhibited any tension in the flexed position. Although the differences in instrumentation and protocol in these three studies render direct comparison of measured values unproductive, similar trends can be observed. All reported

increased tension in some portion of the ligament at similar positions of extreme flexion and extension.

Norwood and Cross (1977) forced the knee joint past anatomical limits into hyperextension and reported that the PLB of the ACL failed first, followed by the AMB and the intermediate band. They suggested that the ACL resisted hyperextension. However in similar tests the ACL was not damaged until both the the posterior capsule and PCL had torn (Kennedy, Weinberg & Wilson,1974). The latter suggests that the ACL function is secondary to that of the PCL and posterior capsule in resisting hyperextension. Norwood and Cross (1977) disagreed stating that the PCL did not fail until the joint was extended 10o past the point of ACL failure.

Mains et al. (1977) and Girgis et al. (1975) recorded an increased range of hyperextension following bisection of the ACL. Furman reported similar results, noting that severing the entire ligament resulted in a larger increase than cutting the PLB only, which in turn yielded a larger change than cutting only the AMB. Furman's results are similar to those reported by Norwood and Cross (1977).

Norwood and Cross (1979; 1977) argued that radiographic analysis of the extended knee revealed that the AMB and intermediate band of the ACL support the intercondylar shelf of the femur and must consequently prevent hyperextension.

Note that the radiographic analysis consisted of inspecting the location and orientation of the ACL fibres in different positions.

Tibial rotation. Brantigan and Voshell (1941) observed increased tension in the ACL when the the tibia was internally rotated. Kennedy et al. (1974) described similar results except that tension increased only when the tibia was rotated in the flexed position. Girgis et al. (1975) claimed that the ligament tension increased when the joint was rotated in extension. Lewis et al. (1982), Kennedy et al. (1977) and Detenbeck (1974) measured increased ACL tension subsequent to internal rotation of the tibia.

Markolf et al. (1976) observed an increased rotary laxity in the extended knee after severing the ACL. Girgis et al. (1975), Mains et al. (1977), and Lipke et al. (1981) reported similar results but noted that the increased laxity was limited to internal rotation of the tibia. Furman et al. specified that cutting the PLB increased internal rotation while the joint was extended, but severing the AMB increased internal rotation in all positions. Norwood and Cross (1979) observed that bisection of the AMB increased anterolateral instability of the knee joint(internal rotation of the tibia).

Markolf et al. (1984) reported increased internal rotation of the tibia in subjects with a history of ACL deficiency. Internal

rotation increased in all joint positions but the position of greatest change was 20° flexion (Markolf, Kochan, & Amstutz, 1984).

Lewis et al. (1982) observed that when the joint was flexed to 90°, tension in the posterior portion of the ligament increased as external rotation forces were applied.

Girgis et al. (1975) demonstrated that cutting the ACL increased external rotation while the knee was in a extended position. Furman et al. (1976) severed the AMB and produced identical results. However Furman et al. (1976) also reported that if the PLB was cut external rotation occurred in all positions.

Lipke et al. (1981), Mains et al. (1977), and Hsieh and Walker (1976) alleged that cutting the ACL did not significantly increase rotary laxity. Wang and Walker (1974) claimed that excess rotary laxity was prevented by the collaterals with the assistance of the mensci. Gollehon et al. (1987) observed no change in rotary movement after the ACL was cut unless the LCL and the deep posterolateral structures were also cut.

Varus-valgus laxity. Markolf et al (1976) reported that cutting the ACL increased varus-valgus laxity if the knee was extended. Grood et al. (1981) observed similar results at 5° and 25° of flexion if collateral ligaments and capsular

structures were already severed. When comparing ACL deficient to normal knees Markolf et al. (1984) observed increased varus-valgus laxity in the damaged knees. The increase was greatest at 20° flexion.

Control of screw home and rolling gliding mechanisms. The screw home mechanism is simply the name given to the automatic rotation (internal rotation of femur or external rotation of the tibia) which occurs in the lower limb as the knee joint approaches terminal extension (Ellison & Berg, 1985). The geometry of the two articulating surfaces dictates that rotation accompanies saggittal motion but tension in the ACL initiates and sustains the rotation (Ellison & Berg, 1985).

The rolling gliding mechanism is the process by which the flexion -extension axis is shifted by combining rotation (rolling) with translation (gliding) in order to either prevent premature impingement of the tibia and femur or restrain the femur from rolling off the supporting tibia (Ellison & Berg, 1985; Müller, 1983; Radin, 1976; Kapandji, 1970). The path of the axis can be simulated by the concept of a crossed four bar link system: the cruciates being fixed at their end-points act as the cross links of the system in the knee joint. Tension in the cruciates controls the ratio of rotation to translation and direction of each movement (rolling and gliding act in opposing

directions) insuring that the normal ROM is facilitated (Kapandji, 1970).

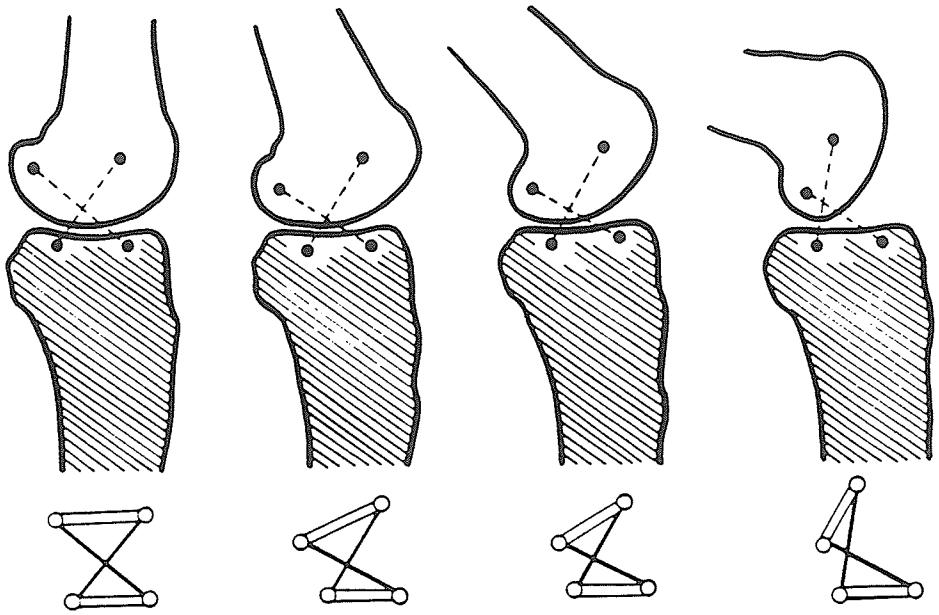

FIGURE 13 THE CRUCIATE LIGAMENTS ACTING IN A FOUR BAR LINK. ADAPTED FROM RADIN (1976)

The hypothesis suggested by Ellison & Berg (1985), Müller (1983), Radin (1976), and Kapandji (1970) advocating ACL control of joint kinematics has not been extensively tested. Indirect evidence was presented by Gerber and Matter (1983) who demonstrated, using an instant center analysis, that both flexion and extension patterns differ in normal and ACL deficient knees. Instant center analysis locates the rotation axis for the movements being tested. All ACL deficient knees, even those exhibiting minimal clinical signs, produced a

characteristic abnormal movement pattern (Gerber & Matter, 1983). Abnormal patterns indicate that the normal rolling gliding mechanism has been altered.

Conclusions regarding ACL function.

The ACL is the primary restraint opposing anterior translation of the tibia, a secondary resistance to hyperextension, rotation of the knee, a secondary stabilzer for abduction and adduction at the knee, and contributes to the control of the rolling gliding mechanism. However, the role of the ACL in the control of the joint dynamics is only indirectly supported.

Test results also indicate that different portions of the ligament may have different functions (Lewis, Lew, Shybut, Jasty, & Hill,1982) and that function changes as the joint passes through its ROM (Furman, Marshall & Girgis, 1976; Girgis, Marshall, & Monajem, 1975).

Joint synergy and ACL function

The function of ACL cannot be defined without acknowledging the synergisms that exist between all the structures in the knee joint. Grood et al. (1980), and Hsieh and Walker (1976) stated that knee joint stability is a product of the interaction of static stabilizers (ligaments, capsular structures, and menisci), dynamic muscular control, joint

geometry, and contact forces between the femur and the tibia. Ellison and Berg (1985) added that ACL function cannot be separated from synergisms with these other factors. To do so, ignores the interrelationships of function between them (Welsh, 1980).

Hsieh and Walker (1976) termed ligaments, joint capsule, muscles, and menisci intrinsic stabilizing factors while the compressive forces generated by gravity were designated as an extrinsic factor.

Intrinsic stabilizers were deemed most important in nonload-bearing situations and extrinsic in load-bearing situations. When compressive forces equivalent to those experienced in vivo were applied in an in vitro test, joint laxity in all directions of movement was reduced in all positions, even after stabilizing structures had been bisected (Markolf, Bargar, Shoemaker, & Amstutz 1981; Hsieh & Walker, 1976).

Joint shape was also identified as a factor in weight-bearing situations since the femur must move uphill on the tibia to either displace anteriorly or rotate (Hsieh & Walker, 1976).

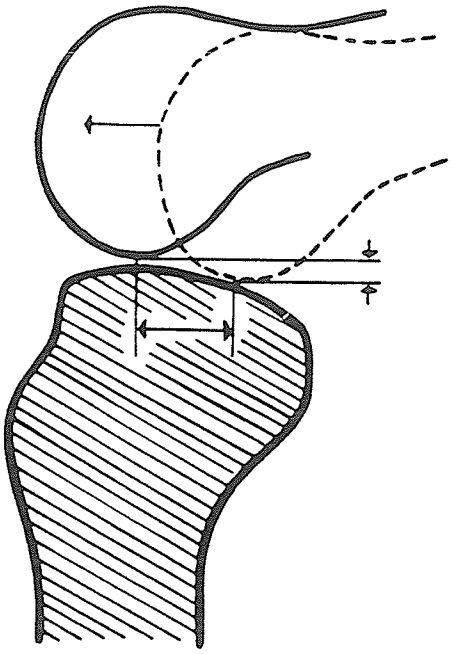

FIGURE 14
UPHILL MOVEMENT OF THE FEMUR ON THE TIBIA AS THE FEMUR DISPLACES ANTERIORLY.
ADAPTED FROM KAPANDJI (1970)

Intrinsic factors maintain the correct tibio-femoral relationship allowing the joint geometry to be effective (Hsieh & Walker, 1976). For example, the two cruciates twist around each other when the tibia is externally rotated thus resisting the motion and maintaining proper relationship between the two articulating surfaces (Kapandji, 1971; Brantigan & Voshell, 1941).

Further evidence of the interaction of structures can be inferred from the increased laxity in all directions when the

ACL was cut in conjunction with other structures (Markolf et al., 1976)

The true function of the ACL or any other structure cannot be defined without acknowledging these synergisms that exist between them. This complexity can lead to problems of diagnosis, treatment, and prognosis in the case of injury.

ACL INJURY

Introduction.

Johnson (1983) described the ACL as the most frequently torn ligament in the knee with frequency of injury surpassing that of the MCL. Andrish (1985) claimed that injury to the ACL is the most frequent severe ligamentous injury to the knee. Tibone, Antich, Fanton, Moynes, and Perry (1986) concurred stating that the ACL had replaced the MCL as the most injured knee ligament, although part of this trend was due to better recognition of ACL injuries. McConkey (1986) reported isolated or principal injury to ACL comprised one third of all knee injuries incurred while skiing. Howe and Johnson (1985) observed that only 7% of knee injuries to skiers were isolated ACL ruptures but failed to include ACL injuries which occurred in conjunction with MCL injuries. They also suggested that the number of ACL injuries appears to be increasing. A recent survey of sports injuries indicated that 10% of knee injuries in males and 7% in females were sprains of the ACL (DeHaven & Lintner, 1986). However this last study included a separate injury category " internal derangement of the knee " which would have included ACL injuries and consequently ACL injuries were likely underreported. Jensen, Conn, Hazelrigg, and Hewett (1985) noted that 23% of sports related acute knee

injuries were tears of the ACL. Clancy (1983) claimed that ACL insufficiency is the most common cause of knee instability.

Mechanism of injury

Angle of knee flexion, direction of applied forces, compressive load, and external contact are the critical parameters for description of the injury mechanism (Zarins & Nemeth, 1985). Intrinsic forces are generated within the subject's body, while extrinsic forces are are generated by interaction with the environment (McConkey,1986). Contact injuries are caused by the transfer of force after a collision with another body (Zarins & Nemeth,1985). Feagin and Lambert (1985), Zarins and Nemeth (1985), Derscheid and Malone (1980) implicated deceleration forces in many non-contact injuries.

McConkey (1986), Howe & Johnson (1985), Zarins and Nemeth (1985), King (1983), Wang, Rubin, and Marshall (1975), and Norwood and Cross (1977) suggested that hyperextension of the knee joint was the mechanism which would result in an isolated injury of the ACL. Abbot et al. (1944) agreed with the mechanism but argued that the PCL and the posterior capsule were also injured. Zarins and Nemeth (1985), King (1983), Fetto and Marshall (1980), Marshall et al. (1978), and Furman et al. (1976) reported that hyperextension combined with internal tibial rotation resulted in tearing of the ACL. Mariani

(1982) reported similar injuries when varus forces were combined with hyperextension. Combined hyperextension, varus forces, and internal tibial rotation resulted in both meniscal and ACL tears (Warren & Marshall, 1978). Warren & Marshall (1978) also observed ACL injury attributed to hyperflexion of the knee.

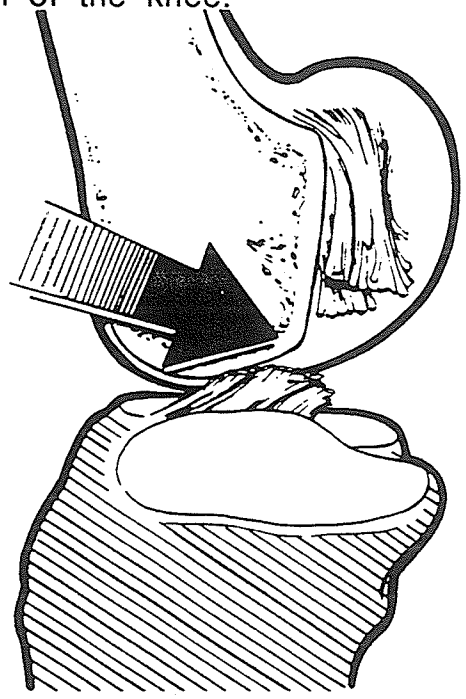

FIGURE 15 ACL INJURY DUE TO HYPEREXTENSION MECHANISM. ADAPTED FROM NORWOOD & CROSS (1977)

Howe and Johnston (1985), Feagin and Lambert (1985), Derscheid and Malone (1980), and Kennedy et al. (1974) suggested that excessive internal rotation resulted in an isolated ACL lesion. Feagin and Lambert (1985) described how the ACL was stretched over the PCL until it finally gave way.

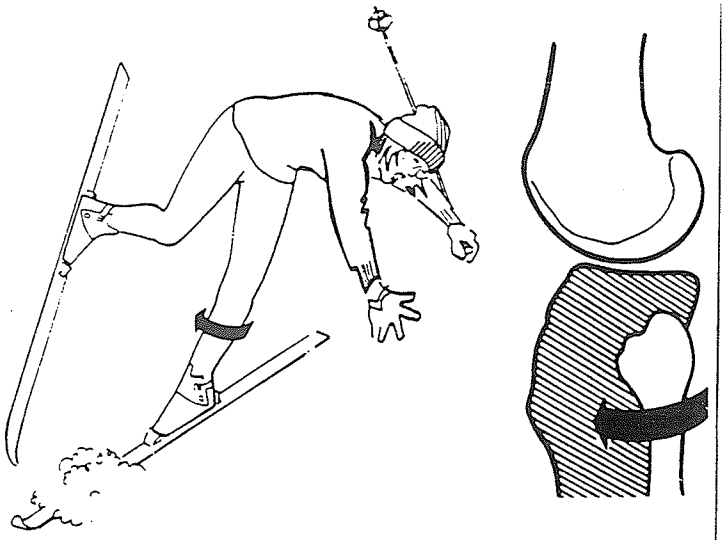

FIGURE 16 EXCESSIVE INTERNAL ROTATION MECHANISM COMMONLY ASSOCIATED WITH ACL INJURY. ADAPTED FROM KENNEDY ETAL (1974)

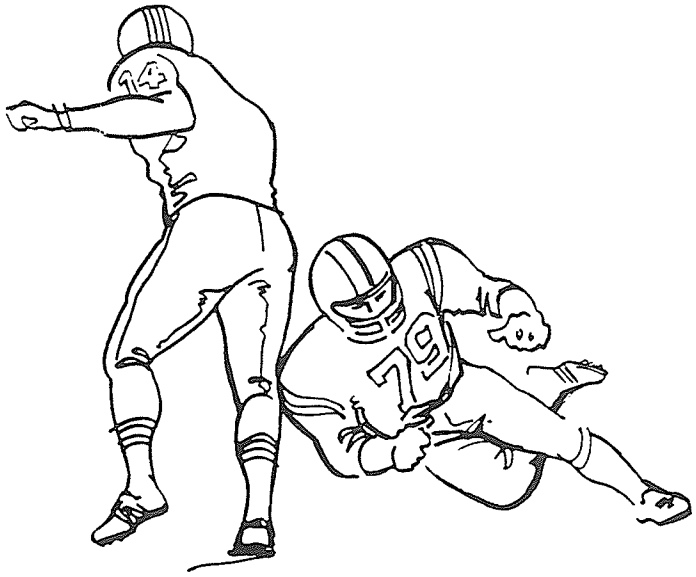

FIGURE 17 CONTACT INJURY ASSOCIATED WITH "TRIAD" PATTERN OF INJURY. ADAPTED FROM WANG, RUBIN & MARSHALL (1975)

Derscheid and Malone (1980), and Warren and Marshall (1978) reported excessive valgus stress caused damage to the ACL but only after the MCL had been ruptured. Such injuries are usually caused by external forces applied to the lateral aspect of the thigh or leg (contact injuries). Zarins and Nemeth (1985) described a similar type of contact injury, but the leg was also externally rotated and the knee was flexed to some degree. Fetto and Marshall (1980) and Norwood and Cross (1979) concurred, adding that MCL lesions always accompanied ACL damage and often lesions to the medial meniscus. This combination of of lesions has been called the "triad" (O' Donoghue, 1981).

Contact varus injuries are rare because the medial aspect of the joint is protected by the contralateral limb (Zarins & Nemeth, 1985). Abbot et al. (1944) alleged that valgus forces combined with internal rotation while the knee is fixed in a flexed position result in the triad pattern of injury.

McConkey (1986) described a non-contact mechanism, that of catching an inside edge while skiing, which involves valgus stress and external rotation of the tibia in a semi-flexed position. The resulting lesions to the ACL and MCL resemble those of the contact injury (McConkey, 1986; Warren & Marshall, 1978; Kennedy, Weinberg, & Wilson, 1974). Zarins and Nemeth (1985) described the same mechanism with the added stresses of deceleration with the leg externally rotated and the knee flexed. Abbot et al. (1944) reported a mechanism involving external tibial rotation and varus forces in the flexed position, which resulted in ACL and MCL lesions.

Feagin & Lambert (1985) claimed that excessive external tibial rotation stretches the ACL across the lateral femoral condyle resulting in an isolated lesion of the ACL. Howe & Johnson (1985) agreed with the mechanism but argued that the medial collateral and medial capsular ligaments were also damaged.

McConkey (1986) described two skiing related mechanisms in which the tibia was subjected to excessive anterior forces

producing an isolated ACL lesion. In one instance the anterior force was generated by the long lever arm of the ski and transmitted to the knee by the stiff boot during a flat landing after "taking air" (McConkey, 1986). In the other violent contraction of the quadriceps muscles during recovery from falling back caused an anterior drawer stress sufficient to rupture the ACL (McConkey, 1986). Derscheid and Malone (1980) and Kennedy et al. (1974) reported the same outcome for direct posterior violence to the tibia in non-skiing incidents.

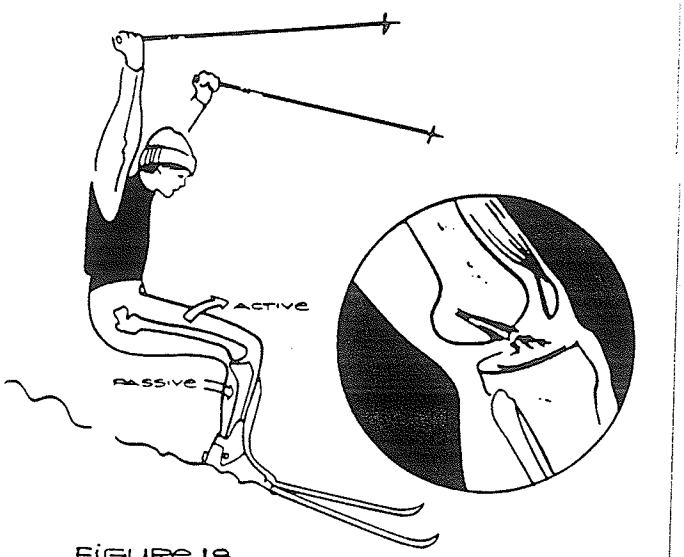

FIGURE 18
VIOLENT CONTRACTION OF QUADRICEPS RESULTING IN ACL INJURY.
ADAPTED FROM MCKONKEY (1986)

Feagin and Lambert (1985) listed a number of other factors which contributed to ACL injury. These included conditioning, fatigue, cyclic loading, strength, and ageing. Weisman, Pope and Johnson (1980) reported that cyclic loading results in

softening and a reduction of strength in the ligament. Skinner, Wyatt, Stones, Hogdon, and Barrack (1986) and Steiner, Chillag, and Schelberg-Karnes (1986) observed increased anterior-posterior laxity in the knee following exercise. The exercise protocol involved both repetitive movement and muscular fatigue. Although both factors appeared to have some effect, the relative contribution of each was not established.

The injury mechanisms described have generally been collected by reconstructing the traumatic event from individual case histories. Often the mechanisms and the resulting injuries were not identical so the resulting data consists of a number of similar incidents resulting in similar injuries. This variance plus the variety of combinations of injuries makes classification of injuries difficult if not impossible (Derscheid & Malone, 1980). McConkey (1986) conceded that forces of sufficient magnitude applied from any direction will eventually cause ACL disruption. Reproduction of injuries in vitro is difficult due to the loss of dynamic functions and joint synergisms, while in vivo reproduction is clearly impossible.

Classification of sprains

An injury to a ligament is defined as a sprain (Andrish, 1985). Sprains occur when sufficient force is transmitted through a joint so that the joint in question exceeds its normal range of motion (Derscheid & Malone, 1980). All sprains can be

classified as either first, second, or third degree according to the severity of the injury (Andrish, 1985).

A first degree sprain exhibits an intact ligament with only microscopic tears and uninterrupted function (Andrish, 1985; Baugher & White, 1985). Pain or tenderness may be present but there is no instability (Andrish, 1985; Baugher & White, 1985). Partial tearing of the ligament with some loss of function and a mild instability indicates a second degree sprain (Andrish, 1985; Baugher & White, 1985). Considerable pain and swelling accompany a second degree sprain but there is a firm end-point at the limits of the range of motion (Andrish, 1985; Baugher & White, 1985).

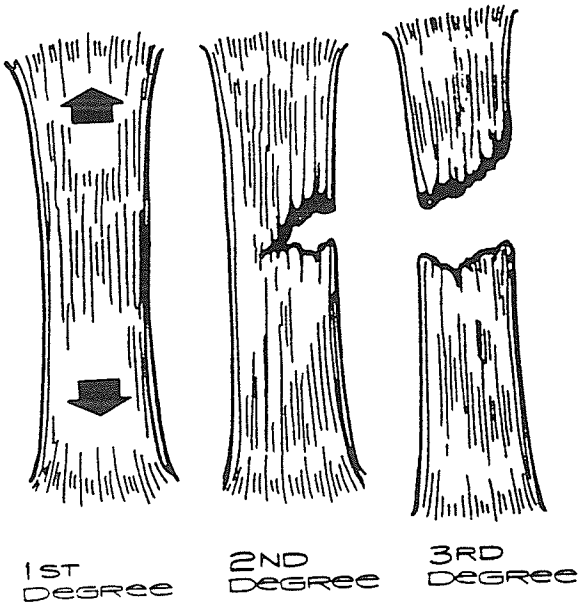

FIGURE 19
CLASSIFICATION OF SPRAINS.
ADAPTED FROM ANDRISH (1985)

If the ligament is completely torn the injury is designated as a third degree sprain (Andrish, 1985; Baugher & White, 1985).

Third degree sprains result in severe degradation of function, copious swelling, and acute instability (Andrish, 1985; Baugher & White, 1985). Pain is often less severe than in a second degree injury due to the lack of tension in the disrupted structure (Andrish, 1985; Baugher & White, 1985). The end-point at the limit of the ROM is soft and indistinct; some describe it as "mushy" (Andrish, 1985; Baugher & White, 1985).

Classification of Instability

By definition, instability implies unsteadiness and is a pathological condition indicative of a clinical syndrome (Losee, 1985). Instability is not synonymous with laxity, which refers only to the amount of motion in the joint as a response to an applied force (Losee, 1985; Clancy, 1983). Laxity describes the static looseness of the knee while instability refers to the functional status of the joint (Losee, 1985; Clancy, 1983). However, one must be aware that many authors have tended to ignore the distinction between the two terms and have used the term instability to describe excess or abnormal laxity.

Classification of instability based on the pathology is difficult because instability can vary significantly for similar injuries (Clancy, 1983). Therefore classification has been based upon the direction of the abnormal motion (Nicholas, 1978). Three general types of instability have been identified;

one-plane, rotary and combined rotary (Nicholas, 1978). Specific instabilities are identified by referring to the movement of the tibia in relation to the fixed femur (Andrews & Axe, 1985; Hughston & Barrett, 1983; DeHaven, 1976).

straight instabilities. Straight instabilities involve movement in one plane and described as follows:

medial: abduction of the tibia

lateral: adduction of the tibia

anterior: anterior translation of the tibia

posterior: posterior translation of the tibia

(Andrews & Axe, 1985; Hughston & Barrett, 1983; Fowler,1980; DeHaven, 1976).

Anterior instability often accompanies isolated ACL injuries, while medial and lateral instabilities are present only if the primary medial and lateral stabilizers are also damaged (Andrews & Axe, 1985; Jensen, Conn, Hazelrigg, & Hewitt, 1985; Fowler, 1980; Marshall & Rubin, 1977; Butler, Noyes, & Grood, 1980).

rotary instabilities. Rotary instabilities exhibit simultaneous movement in two or more planes (DeHaven, 1976). Three types of rotary instability have been commonly identified; anteromedial (AMRI), anterolateral (ALRI), and posterolateral (PLRI) (Andrews & Axe, 1985; Hughston & Barrett, 1983; DeHaven, 1976). Rotary instabilities require a

pivot around which the tibia can rotate, normally an intact PCL (Andrews & Axe, 1985; Hughston & Barrett, 1983; Norwood & Hughston 1980). A rotary instability is a combination of straight one plane instabilities (Marshall & Baugher,1980; Marshall & Rubin, 1977; Norwood & Cross, 1979). Identification of the rotary instability can best be accomplished by identifying the straight plane instabilities individually (Marshall & Baugher,1980; Marshall & Rubin, 1977; Norwood & Cross, 1979). Rotary instability is an expression of ligament injury but not specifically of one structure (Norwood & Hughston, 1980). Consequently rotary instabilities which involve the ACL generally indicate damage to other structures (Terry & Hughston, 1985).

Anteromedial instability is demonstrated by an anterior shift of the medial tibial plateau and simultaneous external rotation of the tibia (Andrews & Axe, 1985; Hughston & Barrett, 1983; DeHaven, 1976). Suggested mechanisms of the pathology include application of excessive external rotation and abduction forces to the knee, or deterioration of secondary stabilizers in the anterior cruciate deficient knee (Ellison, 1980). Many believe that AMRI is the inevitable product of anterior cruciate pathology (Jensen, Conn, Hazelrigg, and Hewett, 1985; 1985; Terry & Hughston, 1985; Hughston & Barrett, 1983; Larson,1982; Norwood & Hughston, 1980;

Marshall & Rubin, 1977; Fowler, 1980). Others (Andrews & Axe,1985) suggested that only severe cases of AMRI involved ACL injury. Lipke et al. (1981) reported that bisecting the ACL in cadavers did not produce any signs of AMRI.

Anterolateral instability has been described as a mirror image of AMRI (Andrews & Axe,1985) in which the lateral tibial plateau subluxes anteriorly with concomitant internal rotation of the tibia (Andrews & Axe,1985; Hughston & Barrett, 1983; Fowler, 1980; Ellison 1980; De Haven, 1976). Fowler (1980) also differentiates between ALRI as the knee approaches extension and ALRI while the knee is flexed. ACL pathology has been identified as an underlying cause of ALRI (Jensen, Conn, Hazelrigg, and Hewett,1985; Noyes, McGinniss, & Grood, 1985; Fowler, 1980; Galway & Macintosh, 1980; Norwood & Hughston, 1980; Marshall & Rubin, 1977). Andrews & Axe (1985) suggested that individuals suffering isolated ACL injuries can exhibit ALRI. Lipke et al. (1981) observed that severing the ACL in cadavers produced ALRI, but Gollehon et al. (1987) reported that bisecting the ACL had no effect on ALRI.

Posterolateral instability involves posterior movement of the lateral tibial plateau combined with external or lateral rotation of the tibia (Andrews & Axe, 1985; Hughston & Barrett, 1983; Fowler, 1980; De Haven, 1976). Terry & Hughston (1985) linked ACL pathology and PLRI. DeHaven

(1976) also described posteromedial instability, a rare condition which does not appear to involve the ACL.

Rotary instabilities may be combined in any fashion (Andrews & Axe, 1985; Hughston & Barrett, 1983; De Haven, 1976). Combined rotary instabilities are the expression of either severe (Fowler; 1980) or chronic (Andrews & Axe, 1985) injuries. Damage to the ACL has been implicated in combined ALRI-AMRI, the most common combined rotary instability (Terry & Hughston, 1985; Fowler, 1980; Norwood & Hughston, 1980)

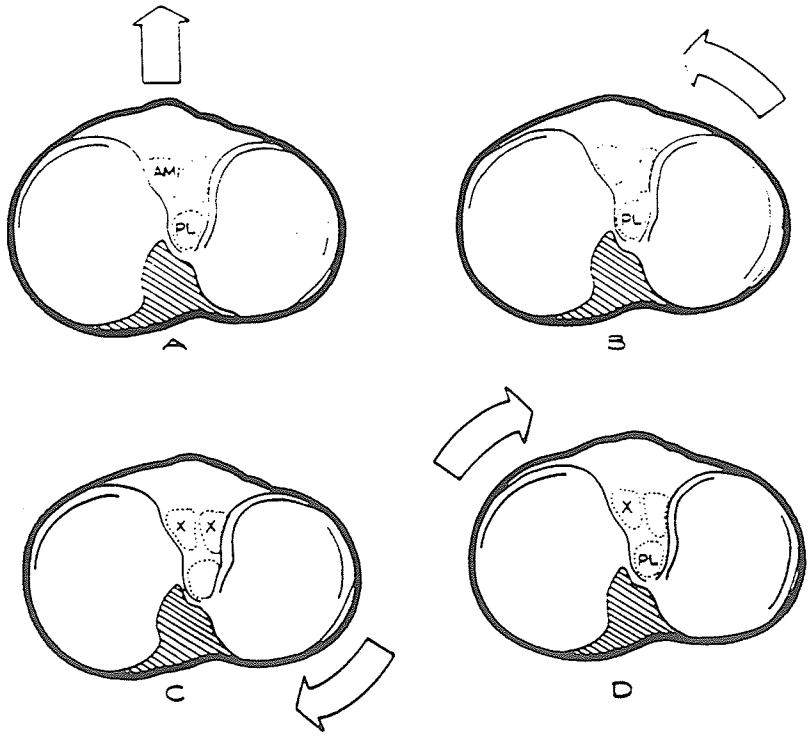

FIGURE 20
INSTABILITIES AND ASSOCIATED PATTERNS OF PATHOLOGY.
A. STRAIGHT ANTERIOR
B. ANTEROLATERAL
C. POSTEROLATERAL
D. ANTEROMEDIAL

ADAPTED FROM NORWOOD & CROSS (1977)

Diagnosis of injury

Introduction

The most important factor in treatment of ACL injuries is an accurate diagnosis (O' Donoghue, 1981). Diagnosis of a tear of the ACL is a difficult task which is made even more so if the tear is an isolated injury (Andrish, 1985; Johnson, 1983; King, 1983; O'Donoghue, 1981). Gross disruptions of joint integrity are relatively easy to assess but more subtle injuries can be underestimated (Noyes, Paulos, Mooar, & Signer, 1980). Retrospective studies have reported that at the time of the initial examination 80-93% of ACL injuries were either misdiagnosed or under-diagnosed (Fowler & Regan, 1987; Noyes, Mooar, Matthews, & Butler, 1983; Mariani,1982; Dehaven, 1980; Noyes, Paulos, Mooar, & Signer, 1980). Marshall, Rubin, Wang, Fetto, and Arnoczky (1978) reported that 58% of initial diagnosis were correct. These figures may be subject to dispute (Kannus & Järvinen, 1987; Simonsen, Jensen, Mouritsen, & Lauritzen, 1984) but even error figures half as large indicate a substantial problem.

Diagnosis and subsequent treatment of a ligamentous injury to the knee are based upon a thorough history and a clinical evaluation (Baugher & White, 1985; Losee, 1985; Zarins and Nemeth, 1985; Larson, 1983). Losee (1985), and Zarins and

Nemeth (1985) claimed that diagnosis is suggested by history, confirmed by clinical tests and verified by arthroscopy.

history

An adequate history includes a description of; the mechanism of the injury, pain, swelling, unusual sensations, and prior injuries (Marshall & Baugher, 1985; King, 1983; Marshall & Rubin, 1977).

mechanism. A number of characteristic mechanisms for ACL injury have been described in a previous section. Zarins and Nemeth (1985) and Fetto and Marshall (1979) claimed that the mechanism of the injury provides a strong "index of suspicion" as to the nature of the injury.

Note however, that many mechanisms for ACL injuries have been described and that McConkey (1986) has conceded that forces of sufficient magnitude applied from any direction will eventually cause ACL disruption. Consequently, mechanism of injury may be somewhat less valuable when dealing with ACL injury. In addition, the patient's recollections of the mechanism can be less than perfect.

hemarthrosis. A tense bloody effusion of the joint capsule has been described as a reliable indicator of ACL pathology (Andrish,1985; Losee, 1985; Zarins & Nemeth, 1985). Marshall, Rubin, Wang, Fetto, and Arnoczky (1978) and Marshall and Rubin (1977) stated that injury to the ACL is the most common

reason for hemarthrosis of the knee joint. Jensen et al. (1985) and Noyes, Bassett, Grood, and Butler (1980) observed 73% of cases involving hemarthrosis of the knee joint also involved ACL injury. Bell (1984) suggested that the figure was 63%.

popping. A popping sensation in the knee that is either heard or felt is suggestive of injury to the ACL (Andrish, 1985; Losee, 1985; Zarins & Nemeth, 1985; Fetto & Marshall, 1980). Marshall et al. (1978) and Marshall and Rubin (1977) reported that 90% of those who experience the popping sensation have suffered ACL damage. Daniel, Stone, Sachs, and Malcom (1985) claimed that 81% of ACL injuries had been accompanied by a popping sensation. Marshall et al. (1978) did not agree stating that 65% of individuals presenting with ACL injuries had not experienced the popping sensation. Noyes, Bassett, Grood and Butler (1980) claimed identical results, while Warren and Marshall (1978) observed that the "pop" was present in 21% of ACL injuries. One might conclude that the "pop" when present is a reasonably accurate indicator of an ACL injury but its absence does not preclude injury to the ligament.

locking. Locking is not a reliable indicator of ACL pathology because there are conditions, such as torn menisci and loose bodies which are as apt to cause the joint to lock as a torn ACL stump (Marshall & Rubin, 1977). True locking can

also be confused with the pseudo-locking effects of pain, joint effusion and muscle spasm (Farquharson-Roberts & Osborne, 1983; Marshall & Rubin, 1977).

others. Losee (1985) and Fetto and Marshall (1985) suggested that recurrent episodes of "giving away" were indicative of ACL pathology. In light of the previous section on instability one might find it difficult to disagree. Losee (1985) also suggested that guarding the joint by internally rotating the femur or externally rotating the tibia was often a positive indicator of ACL injury.

clinical tests.

Clinical laxity tests attempt to isolate the passive restraints provided by the ligaments (Noyes, Grood, Butler, & Malek, 1980) because the status of a ligament correlates highly with its functional integrity (Marshall & Baugher, 1977; Marshall & Rubin, 1977). Consequently, ligament pathology can be inferred from the laxity tests (Marshall & Baugher, 1980; Marshall & Rubin, 1977). If, following injury, the ligament shows no abnormal laxity, it is functionally intact and the diagnosis must be a first degree sprain (Marshall & Baugher, 1980; Marshall & Rubin, 1977). If there is some abnormal laxity with a firm endpoint the diagnosis is a second degree sprain (Marshall & Baugher, 1980; Marshall & Rubin, 1977). Gross instability with a soft or indistinct endpoint indicates a

third degree sprain (Marshall & Baugher, 1980; Marshall & Rubin, 1977). Note that the quality of the "endfeel" is an important aspect of the test. Marshall & Baugher (1980) and Marshall & Rubin (1977) suggested that accurate grading of ACL sprains is not practical.

Anatomic location and severity of injury are best described in straight planes unless the condition is of a chronic nature in which single plane testing can be confounded by partial healing and fatigue failures (Marshall & Baugher, 1980).

general limitations of clinical tests. Noyes, Paulos, Mooar, and Signer (1980) suggested that clinical tests may have limitations. Oliver and Coughlin (1987) described a number of other factors which could effect the outcome of clinical tests. These included the size of the limb in question, the ability of the patient to cooperate, the elapsed time between injury and examination, and a history of prior injury (Oliver and Coughlin, 1987; Johnson,1983). Stauffer, Chao, and Györy (1977) observed that clinical signs might be subtle or misleading when dealing with subtle or incomplete ligamentous injuries.

Clinical laxity tests are highly subjective (Oliver and Coughlin, 1987; Noyes, Grood, Butler, & Malek, 1980; Crowinshield, Pope, Johnson, & Miller, 1976). Laxity tests are estimated rather than measured and the other critical parameter, endfeel, is strictly a qualitative property (Marshall

& Baugher, 1980; Marshall & Rubin, 1977). Bargar, Moreland, & Markolf (1980) observed that the expectations of the examiner influenced the outcome of laxity tests. Examiners tended to overestimate joint laxity if they had been lead to believe that the joint in question was unstable (Bargar, Moreland, & Markolf, 1980).

Because clinical tests are subjective, the skill and experience of the examiner also have a bearing on the outcome (Oliver and Coughlin, 1987; Katz and Fingeroth, 1986; Daniel, Stone, Sachs, and Malcom, 1985). An examiner must detect changes in laxity as small as three millimeters simply by feel, (Daniel, Stone, Sachs, and Malcom, 1985) and differences of endfeel are often subtle (Marshall & Baugher, 1980; Marshall & Rubin, 1977; Stauffer, Chao, and Györy, 1977).

Reliability of clinical tests is an issue because subjective data is very difficult to compare accurately and accurate reproduction of test results is a questionable process (Stauffer, Chao, and Györy, 1977). Bargar, Moreland, Markolf, & Shoemaker (1983) observed that slightly changing the position of the foot during an anterior drawer test had significant effects on the test results and Markolf, Graff-Radford, and Amstutz (1978) suggested that the amount of laxity is a function of the force used in the test. Exact replication of foot position and test forces in every

application would seem to be a formidable task. Markolf et al. (1978) also observed significant differences between measured anterior-posterior laxities of left and right knees in normal subjects. Since the injured joint is compared to the contralateral limb, the findings of Markolf et al. (1978) raise questions about test reliability.

Noyes, Grood, Butler, and Malek (1980) suggested that clinical laxity tests may sometimes prove invalid because the forces generated during the tests are insufficient to overcome the restraining forces provided by secondary stabilizers. Consequently, clinical signs will be absent even though the patient reports functional disability (Jensen, Conn, Hazelrigg, & Hewitt, 1985; Butler, Noyes, & Grood, 1980; Noyes, Grood, Butler, and Raterman, 1980; Noyes, Grood, Butler, and Malek, 1980). When activity is resumed, the secondary stabilizers are unable to resist the larger forces and reinjury (usually more severe) or damage to uninjured structures occurs (Noyes, Grood, Butler, and Malek, 1980).

The validity of clinical tests is also questionable because the clinical evaluation does not define or predict the functional status of the injured knee (Noyes, Grood, Butler, and Malek, 1980; Hughston, 1980; Stauffer, Chao, and Györy, 1977). Only the passive restraints provided by the ligaments are tested while the other factors which contribute to joint

stability are ignored (Noyes, Grood, Butler, and Malek, 1980; Hughston, 1980; Stauffer, Chao, and Györy, 1977). Consequently disruption of function does not always follow disruption of the ACL (Johnson, 1983).

The preceding limitations apply in some measure to all clinical laxity tests although perhaps not to the same degree. Individual tests, including those specific to the ACL may also exhibit characteristic deficiencies. These inadequacies will be discussed with the tests in question.

anterior drawer test. Jensen et al. (1985), Larsen, 1983; Marshall and Rubin (1977), and Hughston et al. (1976) described the test as follows. The patient is either supine or seated on the examining table with the limb in question flexed 45º, the knee flexed 90º, and the foot firmly fixed on the table in a neutral position. Conducting the tests with the foot in external or internal rotation also tests the medial and lateral structures of the knee, but the test becomes one of rotary instability (Katz and Fingeroth, 1986; Jensen, Conn, Hazelrigg, & Hewett, 1985; Losee,1985; Larson, 1983).

The examiner places hands on the upper tibia, palpating the hamstring tendons with the fingers and resting the thumbs on the tibial plateaus. He/she then pulls forward on the tibia in an attempt to slide it anteriorly on the femur. Tension in the hamstrings should be monitored to prevent the patient from

resisting the movement. A positive test is indicated by anterior translation of the tibia in excess of that found in the uninjured leg, assuming no previous injuries (Jensen, Conn, Hazelrigg, & Hewett, 1985; Marshall and Rubin, 1977; Hughston, Andrews, Cross, & Moschi, 1976). Movement of less than 5 mm is classified as a grade 1 laxity, 5-10 mm of translation is a grade 2 laxity and anything greater than 10 mm is designated as a grade three laxity (Jensen, Conn, Hazelrigg, & Hewett, 1985; Marshall and Rubin, 1977; Hughston, Andrews, Cross, & Moschi, 1976).

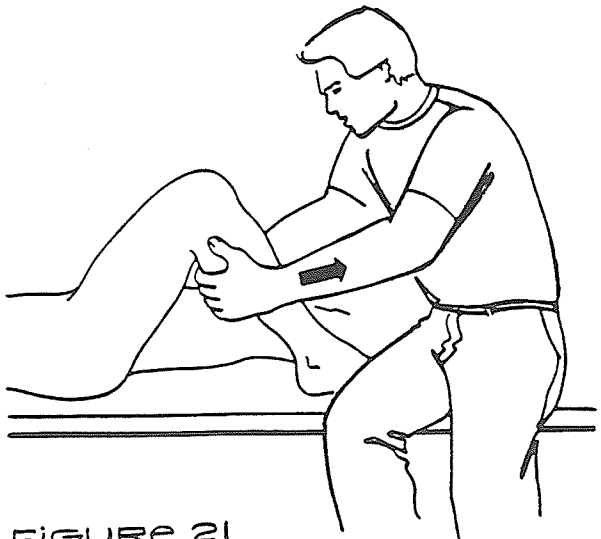

FIGURE 21
ANTERIOR DRAWER TEST.
ADAPTED FROM FOWLER (1980)

Since the ACL provides the bulk of the resistance to anterior movement of the tibia a positive anterior drawer test has been interpreted as the sign of a torn ACL . Marshall et al. (1978), described the anterior drawer test as the most reliable and consistent test of ACL integrity. Baugher and White (1985), King (1983), O'Donoghue (1981), Fetto and Marshall (1980), Marshall and Baugher (1980), and Marshall and Rubin (1977) reported that there is a high positive correlation between positive anterior drawer and ACL pathology, in other words the positive anterior drawer test was highly specific to ACL injury.

Others have suggested that this is an overly simplistic interpretation of the test. Katz and Fingeroth (1986), King (1983) and Larsen (1983) suggested that because the posterolateral band (PLB) of the ACL is taut only while the joint is in an extended position, and the anterior drawer test is performed at 90º flexion only the the anteromedial band (AMB) of the ligament is tested. Consequently a negative test does not eliminate an injury to the PLB. Clancy (1983) claimed that a positive test indicated only that other structures had been damaged along with the ACL. Hughston et al. (1976) partially agreed stating that a positive drawer test is a manifestation of anterolateral instability and as such indicates injury to the medial structures.

DeHaven (1983) also suggested that the anterior drawer test is not always a true indicator of ACL status. Howe and Johnson, (1985) proposed a number of conditions which might prevent a positive test in an injured knee. Increasing the likelihood of a false-negative test effectively decreases test sensitivity. Hamstring spasm caused by pain can effectively resist the anterior movement of the tibia, giving the effect of an intact ligament (Donaldson, Warren, & Wickiewicz, 1985; Howe and Johnson,1985; Jonsson, Althoff, Peterson, & Renstrow, 1982). The posterior horns of the menisci can wedge under the femoral condyles to block anterior tibial translation (Katz and Fingeroth, 1986; Howe and Johnson,1985; Zarins & Nemeth, 1985). Ligament fragments, torn menisci, or loose bodies can also block anterior subluxation of the tibia (Marshall, Rubin, Wang, Fetto, & Arnoczky,1978; Marshall & Rubin, 1977). Joint effusion or hemarthrosis may mask positive results (Katz and Fingeroth, 1986; Donaldson, Warren, & Wickiewicz, 1985; Howe and Johnson,1985; Jonsson, Althoff, Peterson, & Renstrow, 1982).

The anterior drawer test also appears to be susceptible to the effects of secondary stabilizers. The forces exerted by the secondary restraints (principally the MCL) are sufficient to resist the distraction forces of the test (Katz and Fingeroth, 1986; Baugher & White, 1985; Howe and Johnson,1985). The

apparent sensitivity of the anterior drawer test increases if the secondary stabilizers are injured or stretched because the anterior movement of the tibia is unresisted (Katz and Fingeroth, 1986; Warren, 1982; O'Donoghue, 1981).

False-positive results occur far less often, but misinterpretation of other conditions is possible. Howe and Johnson (1985) and Marshall and Rubin, (1977) reported instances in which posterior instability was identified as anterior laxity or the external rotation, characteristic of AMRI was misinterpreted. Marshall, Rubin, Wang, Fetto, and Arnoczky (1978) suggested that generalized laxity present before the injury might be misinterpreted as positive anterior drawer following an injury.

Howe and Johnson (1985) and Warren (1982) suggested that the anterior drawer test might be unsuitable in many cases because swelling and pain resulting from the injury prevent patients from flexing the the knee to 90º.

Lachman test. The Lachman test has been described as an anterior drawer test performed with the knee joint in a position of 20-30º of flexion (Losee, 1985; Clancy, 1982). The examiner stabilizes the thigh with one hand, and applies an anterior force to the tibia with the other (Andrish, 1985; Baugher & White, 1985; Daniel, Stone, Sachs, & Malcom, 1985; O'Donoghue, 1981). Both the amount and the endfeel of the

tibial movement are monitored and compared to that of the contralateral limb (Baugher & White, 1985; Daniel, Stone, Sachs, & Malcom).

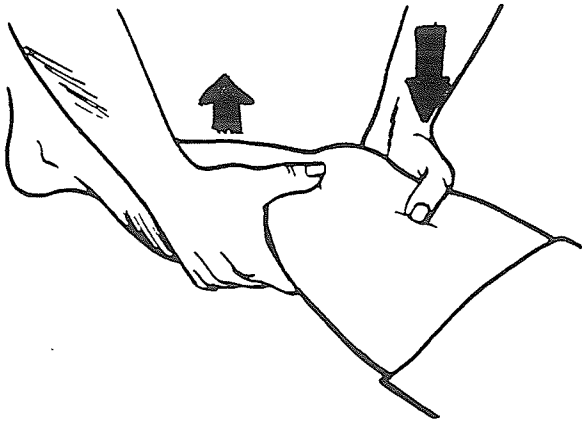

FIGURE 22 LACHMAN TEST FOR ACL INSUFFICIENCY. ADAPTED FROM FOWLER (1980)

Excessive anterior translation of the tibia and/or a soft indistinct endfeel, sometimes described as mushy indicate a positive test (Baugher & White, 1985; Daniel, Stone, Sachs, & Malcom, 1985; Warren, 1982). Larsen (1983) added that positive test is also indicated if the normal concavity of the patellar tendon becomes convex. Baugher & White (1985) suggested that the quality of endfeel is the most important aspect of the test.

A positive Lachman test indicates injury to the ACL (Andrish, 1985; Baugher & White, 1985; Daniel, Stone, Sachs, & Malcom, 1985; O'Donoghue, 1981). Zarins and Nemeth (1985)

stated that the entire ligament is tested while Jensen et al. (1985) and King (1983) claimed that only the PLB is tested since only the PLB is taut as the knee approaches extension. Farquharson-Roberts and Osborne (1983) suggested that a positive Lachman combined with a negative anterior drawer was indicative of a lesion of the PLB while a negative Lachman combined with a positive anterior drawer is indicative of a AMB lesion.

When the Lachman test is compared to the anterior drawer test some advantages are evident. Katz and Fingeroth (1986) and Howe and Johnson (1985) claimed that the wedging effects of the posterior horns of the menisci and the guarding effect resulting from pain are both minimized because the joint is flexed only 20°. Donaldson et al. (1985) reported that the test is not affected by injuries to other structures.

The Lachman test also exhibits some flaws. A large discrepancy between the hand size of the examiner and the size of the patients thigh often causes difficulty in grasping the thigh (Donaldson, Warren, & Wickiewicz, 1985; Howe and Johnson,1985; Larsen, 1983; Jonsson, Althoff, Peterson, & Renstrow, 1982; O'Donoghue, 1981). Obviously all patients with large limbs and considerable muscle mass have the potential to create this problem. If the patient is very large or very strong the examiner might also experience difficulty

when he/she attempts to move the tibia anteriorly (Donaldson, Warren, & Wickiewicz, 1985; Howe and Johnson,1985; Larsen, 1983; Jonsson, Althoff, Peterson, & Renstrow, 1982; O'Donoghue, 1981).

Although the Lachman test is less affected by pain and spasm than the anterior drawer, Clancy (1982) noted that hamstring spasm could evoke a false negative result even though the ACL was damaged. Pain, induced by the required hand position close to the injured joint, often increased guarding and obscured test results (Donaldson, Warren, & Wickiewicz, 1985). Baugher & White (1985) suggested that pain and spasm can block the end point of tibial motion yielding a false negative or inconclusive test result. Donaldson et al. (1985) and Clancy (1983) claimed that a torn medial meniscus combined with a torn posterior capsule could mimic the anterior tibial motion characteristic of a positive Lachman.

pivot shift. The term pivot shift refers both to the clinical sign which is indicative of ACL insufficiency and to the clinical test used to elicit this sign (Larsen, 1983; Fetto and Marshall, 1980; Galway & Macintosh, 1980). Losee (1983) reported that the pivot shift most often occurs during twisting, jumping and deceleration movements. As the leg moves towards an extended position, the tibia shifts

anteriorly so that the lateral femoral condyle rides onto the posterior slope of the lateral tibial plateau (Larson,1983; Losee, 1983; Fetto and Marshall, 1980; Galway & Macintosh, 1980). Tibial motion is initiated passively by external forces or actively by a slingshot effect generated by the contracting quadriceps and patella and increased tension in the ITB (Losee, 1983). If the pivot is of sufficient magnitude the femur can become wedged against the posterior aspect of the tibial plateau (Larson,1983; Losee, 1983; Fetto and Marshall, 1980; Galway & Macintosh, 1980).

Reduction of the pivot shift occurs either when the knee is fully extended or flexed. As the joint reaches full extension the posterior capsule pulls the tibia posteriorly causing reduction (Losee, 1983). When the knee is flexed past 40o the ITB shifts posterior to the axis of the joint and causing a posterior reduction of the tibia (Losee, 1983).

Various tests are employed to reproduce the pivot shift in a clinical setting. These include the pivot shift, Losee, and Macintosh tests (Larson,1983; Losee, 1983). To perform the pivot shift test, the examiner holds the leg by the heel in an extended position (Larson,1983; Losee, 1983). An upward and valgus force is applied at the fibular head as the the knee is passively flexed (Larson,1983; Losee, 1983). A positive test is indicated by subluxation and subsequent reduction of the

tibia at 40o flexion, accompanied by a clunking sensation (Larson,1983; Losee, 1983). Other tests designed to elicit a pivot shift employ similar strategies which yield similar outcomes.

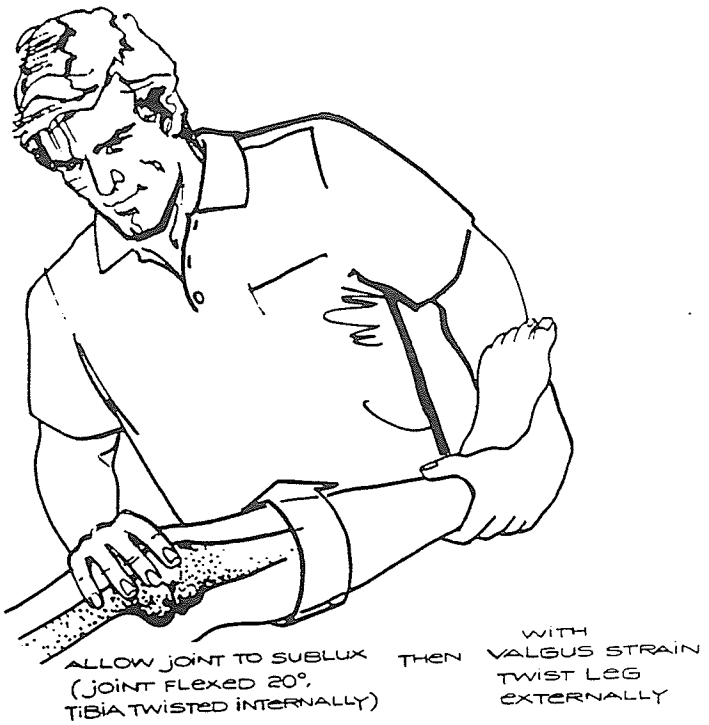

FIGURE 23 PIVOT SHIFT TEST. ADAPTED FROM LOSEE (1983)

Fetto and Marshall (1980) and Galway and Macintosh (1980) reported that a positive pivot shift test indicated ACL insufficiency. Marshall and Rubin (1977) claimed that an isolated ACL injury was sufficient cause for a positive test and that no other structures need be involved. Losee (1985) on

the other hand suggested that a positive pivot shift was indicative of an ALRI, suggesting damage to other structures.

Execution of pivot shift tests is complicated by a number of factors. Patient apprehension and guarding obstruct performance of the tests and obscure test results (Jensen, Conn, Hazelrigg, and Hewett, 1985; Losee,1985; Johnson, 1983; Jonsson, Althoff, Peterson, & Renstrow, 1982). Pivot shift tests like other clinical tests, are subjective and as such are dependent upon the skill and experience of the examiner for effectiveness (Jakob, Stäubli, Deland, 1987).

accuracy of clinical tests. Accuracy rates for clinical tests have been frequently calculated and compared. The procedure most often used has involved comparison of the results of clinical tests with the findings at surgery (arthroscopy or arthrotomy). Accuracy rates for the different tests vary throughout the different studies. These differences exist generally because different criteria have been used to select subjects, to group them and to define success. Consequently, comparison of accuracy rates is at best a tenuous exercise and determination of the appropriate rate for each test is beyond the scope of this paper.

However trends were observed which allowed some general conclusions:

a. The tests were consistently ranked in order of accuracy as follows; Lachman, pivot shift, and anterior drawer (Donaldson, Warren, & Wickiewicz, 1985; Johnson, 1983; Jonsson, Althoff, Peterson, & Renstrow, 1982; Dehaven, 1980).

b. Accuracy rates increased for all of the tests if they were administered while the patient was anesthetized (Daniel, Stone, Sachs, & Malcom, 1985; Donaldson, Warren, & Wickiewicz, 1985; Howe and Johnson,1985; Simonsen, Jensen, Mouritsen, & Lauritzen, 1984; Larsen, 1983; Warren, 1982).

objective laxity tests. Objective testing procedures were developed in response to claims that existing clinical tests were subjective and the results were nonreproducible (Oliver & Coughlin, 1987; Daniel, Stone, Sachs, & Malcom, 1985). The procedures employed mechanical devices which reproduced the stresses of the anterior drawer and Lachman tests using predetermined forces and measured the resulting tibial moverment. Markolf et al. (1978) measured anterio-posterior laxity in normal subjects. Daniel et al. (1985) utilized a MEDmetric KT 1000 arthrometer to establish limits of anterior-posterior laxity in a normal population and compared these values to those generated by injured subjects. Oliver and Coughlin (1987) and Boniface, Fu, and Ilkanipour (1986)

adopted a similar protocal utilizing the Stryker Knee Laxity Tester and the Genucom Knee Analysis System respectively.

Results from the studies in question are inconsistent. Markolf et al. (1978) observed anterior displacement of less than 3mm at both 20° and 90° in the normal subjects, with the larger values at 20°. Subjects were able to reduce the amount of laxity substantially by contracting the thigh muscles. There were also significant differences between left and right limbs in the same subjects. Boniface et al. (1986) produced similar values for anterior laxity in normal joints at 20° but detected significant left-right differences (ie > 2mm) in only 10% of the normal group. In the injury group a difference of 2mm or more was highly suggestive of an ACL tear. Daniel et al. (1984) reported approximately twice as much anterior laxity using a similar force at 30° flexion. The mean difference in laxity between right and left was determined to be less than 2mm and a difference of more than 3mm was strongly correlated with ACL damage. Oliver and Coughlin (1987) reported similar results to those of Daniel et al. and observed that the lateral tibial condyle was displaced more than the medial in male subjects. Oliver and Coughlin also suggested that rotation of the leg decreased the amount of anterior tibial translation.

The studies infer that the Genucom, the MEDmetric, and the Stryker systems may prove useful in the diagnosis of ACL pathology. Objective results can be compared not only to normal populations but to subsequent tests on the same limb in order to monitor the status of the ACL over a period of time. However, objective tests of knee laxity improve only upon the reliability of clinical tests and do not change many of the factors that compromise the effectivness of clinical test. Daniel et al. (1984) identified starting position, amount of force applied, muscle tone of the patient, and rotation of the joint as factors which influenced the degree of laxity. The studies also testify to the importance of the skill and experience of the examiner in interpreting the results of clinical tests. Differences in laxity of 2-3mm between legs were established as grounds for a positive diagnosis. Three millimeters is very small measure and subjective interpretation of this standard requires a very subtle touch. ancillary tests.

A number of tests are used to supplement clinical tests. These include radiography (x-ray), arthrography (both single and double contrast), arthroscopy, and exploratory arthrotomy (Jonsson, Althoff, Peterson, & Renstrow, 1982).

radiography. Standard x-rays are generally used to rule out fractures in cases of knee injury (Tongue & Larson, 1980) but

there are some radiological signs indicative of ACL injury. Second's fracture, a fleck of bone avulsed from the lateral articular margin of the tibia near the joint capsule can denote an acute ACL injury (Losee, 1985). A notch defect in the lateral femoral condyle, especially if accompanied by a positive pivot shift test, signifies a chronic ACL condition (Losee, 1985). Jensen, Conn, Hazelrigg, and Hewett (1985) claimed that stress x-rays are a valuable diagnostic tool for the identification of ligament damage.

Mooar, Gregg, and Jacobstein (1987) claimed on the other hand that x-rays reveal little about soft tissue pathology and are insensitive to mild or moderate degenerative changes. Stress x-rays are technically difficult, time consuming, painful to the patient and provide information of limited value (Mooar, Gregg, and Jacobstein,1987).

Gerber and Matter (1983) employed sucessive frames of a series of lateral view radiographs to calculate the instant-centres of movement throuhout the ROM. When compared to normal joints ACL deficient knees consistently exhibited a characteristic pattern (Gerber & Matter, 1983). The procedure produced reproducible objective results but was apparently effective only for limited well defined lesions (Gerber & Matter, 1983). However the instant centre analysis is highly

technical, time consuming, requires a series of x-rays, and consequently has limited application as a clinical tool.

arthrography. Jensen et al. (1985), Pavlov (1983), Pavlov, Warren, Sherman, and Cayea (1983) and Tongue and Larsen (1980) identified arthrography as a helpful technique for evaluation of the ACL when pain and spasm prevent or limit clinical evaluation. The technique involves injecting a contrast medium either in conjunction with air (double contrast) or without (single contrast), into the joint and taking x-rays (Reider, Clancy, & Langer, 1984; Pavlov, Warren, Sherman, & Cayea,1983; Tongue and Larsen, 1980; Nicholas, Freiberger, & Killoran, 1970). The x-rays may be either regular view or stress (Pavlov, Warren, Sherman, & Cayea,1983; Tongue and Larsen, 1980; Nicholas, Freiberger, & Killoran, 1970).

Pavlov et al. (1983) claimed that the appearance of the synovial surface of the ligament denoted the status of that structure. If the edge of the synovium was straight the ligament was intact. If the edge was not visible, acutely angled, or the contrast medium was pooled the ligament was torn. A lumpy or wavy edge signified a torn but intact ligament, while a bowed edge indicated a lax ligament (Pavlov, Warren, Sherman, & Cayea,1983).

Unfortunately interpretation of results is not always straightforward. Pavlov et al. (1983) noted that irregularities of the synovium have been misinterpreted as ACL pathology. Tongue and Larson (1980) and Dalinka and Garofola (1976) on the other hand argued that either the infrapatellar synovial fold, the infrapatellar fat pad, or the ligamentum mucosa could be mistaken for an intact ACL. Kaye and Frieberger (1975) suggested that partial tears of the ligament might pass unnoticed because the arthrogram only reveals the margins of the structure. Overlapping structures or insufficient coating of the contrast material can obscure portions of the ACL especially the middle third (Jackson, Jennings,, Maywoods, & Berger, 1988; Nicholas, Freiberger, & Killoran, 1970).

Arthrography is an invasive procedure which poses certain risks. Complications can include infection, transient synovitis, and reaction to the contrast material (Jackson, Jennings,, Maywoods, & Berger, 1988; Kaye & Frieberger, 1975; Nicholas, Freiberger, & Killoran, 1970). Patient discomfort has been frequently reported (Jackson, Jennings,, Maywoods, & Berger, 1988; Kaye & Frieberger, 1975).

computed tomography (CT). Dalinka, Gohel, and Rancier (1973) employed lateral tomography in conjunction with normal arthrographic techniques in an attempt to augment diagnostic accuracy in cases of ACL injury. Passariello,

Trecco, Masciocchi, and Beomonte-Zobel (1986) adopted a similar strategy using direct CT of the knee joint. In both instances the authors were able to accurately differentiate leisions of ACL although there were a number of false positives. Regretably, the technology is expensive and of limited availability, often necessitating substantial waiting periods.

magnetic resonance imaging (MRI). Jackson, Jennings, Maywoods, and Berger (1988) reported that a noninvasive procedure, MRI afforded high resolution images of the knee, from which tears of the ACL could be accurately detected. Again, the technology is expensive and of limited availability.

arthroscopy. Arthroscopy has been advocated as a valuable diagnostic procedure in occurrences of hemarthrosis which present equivocal results from clinical tests and/or arthrography (Simonsen, Jensen, Mouritsen, & Lauritzen, 1984; Curren & Woodward, 1980; Jackson & Dehaven, 1975). Johnson (1976) and Watanabe (1975) explained that arthroscopic findings represented direct observation of pathology while all other tests represented circumstantial evidence at best. Mariani (1982) argued that the procedure was especially beneficial for athletes because the "wait and see" period could be eliminated.

The procedure involves the introduction of a fibre optic viewing telescope into the affected joint via a large bore needle (Mariani, 1982; Watanabe, 1976). The joint is irrigated with a sterile saline solution to wash away hemarthrosis and distend the joint for better viewing (Dehaven, 1983; Jackson and Dehaven, 1975; Watanabe, 1975). Dehaven (1983) also specified that palpation via probe is necessary to assess the integrity of the cruciates.

The arthroscopic procedure affords the examiner some important advantages. Because the arthroscope allows direct visualization of the affected joint, accuracy rates approaching 100% have been reported (Selsnick, Bates Noble, Bachman, & Steinberg, 1985; Curran & Woodward, 1980; Noyes, Bassett, Grood, & Butler, 1980; Dehaven & Collins, 1975). When compared to open arthrotomy, the other means of direct visualization, arthroscopy results in minimal tissue morbidity and a shorter healing period (Watanabe, 1975).

Although arthroscopy is a valuable tool in the diagnosis of ACL injuries, the process is not without shortcomings. It is an invasive procedure with the inherent risks of infection, nerve damage, and instrument breakage common to all such procedures (Mooar, Gregg, & Jacobstein, 1988; Sherman, Fox, Synder, Del Pizzo, Freidman, Ferkel, & Lawley, 1986; Peek & Haynes, 1984). Sherman et al. (1986) described complications

which included, adhesions, hemarthrosis, ecchymosis, delayed healing, and compartment syndrome in the lower leg caused by extraversion of the irrigation fluid. Sherman et al. also reported that pure diagnostic arthroscopy caused the least complications but that complications increased with the use of a tourniquet and were more frequent in older patients. Arthroscopy is a surgical procedure with all the attendant surgical/anesthetic risks and also may involve considerable expense and prolonged waiting periods. The quality of results is dependent upon the skill and experience of the surgeon.

functional tests.

Davies, Malone, and Basset (1980) proposed that in cases of uncertain diagnosis, since the test forces are too small to elicit symptoms the only way to reproduce said symptoms is by functional testing. Typical functional tests mimic the patient's normal activities and include; squatting, kneeling, jumping, hopping, running in a straight line, running figure eights, running up and down stairs and slopes, cutting, duck walking, deccelerating, cross over tests, and apprehension tests (Shields, Silva, Lincoln, & Brewster, 1987; Jensen, Conn, Hazelrigg, & Hewett, 1985; Losee, 1985; Larsen, 1983; Losee, 1983).

Often when the clinical tests for ACL insufficiency are equivocal, a "wait and see approach" is adopted (Feagin, 1983; Dehaven, 1980; Noyes, Paulos, Mooar, & Signer, 1980). King (1983) described this period of observation and evaluation of function as "trial of function", a process which involves aspects of both diagnosis and treatment. The treatment is symptomatic and activity is gradually resumed as the acute stage is resolved (King,1983). Resumption of activity serves as a gross functional test. Unfortunately, in this scenario a positive result is indicated by reoccurrence of the injury or exaggeration of symptoms, both undesirable outcomes which suggest that the pathology has progressed to sub-acute chronic stage (Noyes, Paulos, Mooar, & Signer, 1980).

Results of many functional tests are based upon the patients subjective interpretation of discomfort and instability experienced during the test and do not involve the measurement of performance variables. Such tests can confirm the presence of injury in the absence of other symptoms but they remain subjective and nonreproducable since there is no measurement of the performance variables (Boccardi, Chiesa, & Pedotti, 1977; Stauffer, Chao, & Györy, 1977). Tegner, Lysholm, Lysholm, & Gillquist (1986) addressed this problem by measuring time and distance variables generated in tests of straght running, figure of eight running,

stair running, slope running, and hopping off one leg. Comparison of test results for a group of athletes with ACL injuries with those generated by an injured group revealed significant differences in running around the curve in the figure eight test, in stair running, and running up a slope (Tegner, Lysholm, Lysholm, & Gillquist, 1986). Performance deficits correlated highly with injury to the ACL but other contributing variables could not be eliminated. Tegner et al. (1986) suggested that although these functional tests had no predictive power they would serve to monitor patient progress during a course of treatment.

For the most part, the aforementioned movements are of a vigorous nature and include the potential for aggrevation of symptoms or furthur damage (Davies, Malone, & Basset 1980). Patients have often been reluctant to perform such vigorous tests on already unstable joints (Tibone, Antich, Fanton, Moynes, & Perry, 1986; Davis, 1980). Tibone et al. (1986) evaluated the gait characteristics at moderate activity levels (ie normal walking speed) as well as at higher levels, of a group of patients prior to ACL reconstruction. Patient compliance was increased in the less vigorous tests. Parameters evaluated included free walking velocity, cadence, stride length, joint angles, and neuro-muscular activity. In the less strenuous tests all parameters fell within the normal

limits for age and sex and exhibited no differences between the injured and the unaffected limbs.

A percentage of patients do not exhibit disability following ACL injury (Shields, Silva, Yee, & Brewster, 1987; Walla, Albright, McAuley, Martin, Eldridge, & El-Khoury, 1985; Clancy, 1982; Mariani, 1982; Noyes, Paulos, Mooar, & Signer, 1980; Hughston, 1980). Warren (1982) and Mariani (1982) claimed that 20% of ACL patients fit this description but identification of these individuals prior to a "trial of function" is not yet possible (Walla, Albright, McAuley, Martin, Eldridge, & El-Khoury, 1985). King (1983) and Müller (1983) stated that clinical stability is not end in itself but rather a condition of weight bearing function and therefore the pertinant test of an injured knee may well be long-term function. Some researchers (Andrish, 1985; Cabaud & Rodkey 1985; Noyes, Mooar, Matthews, & Butler, 1983; McDaniel & Dameron, 1980; Marshall, Rubin, Wang, Fetto, and Arnoczky, 1978) have argued that the lack of functional degradation represents only a temporary respite and that the joint will eventually degenerate into a state of chronic instability, exhibiting increasing loss of function, meniscal tears, and eventual osteoarthritic changes, while others have suggested that the long term prognosis for these patients is still unresolved. (Noyes, McGinniss, & Grood, 1985; Noyes & McGinniss, 1985;

Odensten, Hamberg, Nordin, Lysholm, & Gillquist, 1985; Walla, Albright, McAuley, Martin, Eldridge, & El-Khoury, 1985; 1985; Larson, 1983).

Functional tests have uses beyond diagnostic application. Once the the acute stage of the injury has passed, the functional status of the knee joint in question becomes the overriding factor for decisions involving treatment and rehabilitation (King, 1983: Müller, 1983). Changes of functional status indicate patient progression or regression and serve as input for treatment descisions (Tegner, Lysholm, Lysholm, & Gillquist, 1986).

Regardless of the diagnosis or the course of treatment followed, the goal of the athlete following injury to the ACL is to return to preinjury performance levels (Tegner, Lysholm, Lysholm, & Gillquist, 1986). A convenient rule of thumb advocated by Arnhiem (1985) is no return to sport until normal range of motion (ROM), strength and coordination are regained. Unfortunately, following ACL rehabilitation, patients often exhibit normal ROM and quadriceps and hamstring strength but are still unable to function at the desired level (Tegner, Lysholm, Lysholm, & Gillquist, 1986). Although strength and ROM are convenient yardsticks(Tegner, Lysholm, Lysholm, & Gillquist, 1986). evaluation of knee function is most essential

to determine return to sport(Tegner, Lysholm, Lysholm, & Gillquist, 1986).

Unfortunately, functional tests for ACL integrity have been generally lacking in both specificity and sensitivity while at the same time maintaining significant potential for exacerbating the pathology they are intended to detect.

Ground Reaction Forces Generated via Force Plate

Introduction

Each foot exerts pressure on the ground during the stance phase of a stride and this force, known as the ground reaction, is the resultant of all forces acting on the body (Boccardi, Chiesa, & Pedotti, 1977). A force plate is an electro-mechanical device which measures the ground reaction force in three dimensions; the vertical plane, the anterior-posterior plane, and medial-lateral plane (Rogers & Cavanagh, 1984). These forces can be plotted as a function of time, to produce a force pattern for the stance phase in question (Boccardi, Chiesa, & Pedotti, 1977). Bocarrdi et al. (1977) claimed that the qualitative data thus produced was unlike other forms of biomechanical measurements, appropriate for clinical use. Harrington (1974) added that analysis of ground reaction forces is a direct, simple, noninvasive technique which permits detection of abnormal loading patterns and correlation of these patterns with clinical findings.

Force plate analysis of pathological gait

Boccardi et al. (1977) compared vectors compiled from vertical and anterior-posterior force plate measurements of normal subjects to vectors generated by subjects afflicted with a pathological gait. The latter subjects generated

patterns that were characteristic for each pathology (poliomyelitis and muscular dystrophy) and significantly different from those of the normal group. The normal group produced patterns which were consistently characteristic for each individual and symmetrical with regard to the contralateral limb (Boccardi, Chiesa, & Pedotti, 1977). Boccardi et al. (1977) suggested that the instrumentation was sufficiently sensitive to register subtle changes in gait as well as the gross dysfunctions recorded in this study.

Stauffer et al. (1977) described a gait analysis project in which the pathological gait group suffered from either unspecified degenerative changes or rheumatoid arthritis of the knee. These subjects exhibited consistent changes in force patterns while the normal group again manifested individual patterns which included left-right symmetry.

Schneider and Chao (1983) reported that subjects afflicted with degenerative knee joint disease generated significantly different ground reaction forces with the affected limb than normal subjects. There were no differences in the unaffected limb of the pathological group, when compared to either the affected limb or the normal group.

Tibone et al. (1986) examined instability in subjects awaiting reconstruction of the ACL and observed that the most meaningful data was collected using a force plate. The

subjects displayed significant force pattern changes in the affected limb as compared to the sound limb, during fast walking, running, and cutting. When subjects were retested two years post reconstruction the deficits recorded in the cutting test had disappeared (Tibone & Antich, 1988). Fast walking and running were not tested in the post operative test. Tibone, Antich, Perry, and Moynes (1988) duplicated the test procedures used by Tibone et al. (1986) to compare function in reconstructed and untreated posterior cruciate injuries. Analysis of force plate data revealed significant changes in gait patterns during walking and runnning.

Proposed use of force plate data for ACL deficient subjects

The proposed test involves measuring ground reaction forces as subjects who have sustained ACL damage walk across a force plate. Smidt (1974) described walking as a natural, automatic function, which presents an ideal means for testing of lower joint integrity. The walking gait also produces forces which can be tolerated by the target group and do not threaten to exacerbate the original injury. Consequently, walking is an easily administered functional test.

The force plate is a simple, direct, non-invasive method of dectecting abnormal loading patterns (Harrington, 1974). Gerber and Matter (1983) observed characteristic abnormal

movement patterns in ACL deficient joints. Abnormal movement patterns should result in irregular loading patterns which can be measured by the force plate. If the loading patterns remain characteristic the test becomes a valid test of ACL function. Neither Peters (1988) nor Tibone et al. (1986) observed these changes in walking patterns but this was most likley due to insensitive analysis of the data rather than failure of the instrumentation.

Conclusions

A major portion of the preceeding review of literature was devoted to the functional anatomy of the knee with emphasis on the ACL. The complexity of both joint and ligament do not allow a definative description of either form or function. This ambiguity extends predictably to the diagnosis and management of ACL injuries.

ACL function cannot be separated from synergisms with other structures. Yet, the clinician attempts to isolate the ACL when testing for ligament integrity. Naturally, the complexity of the joint and interdependency of function thwart attempts to assign a single function to a specific ligament. Consequently, clinical tests are often inconclusive. Testing the joint as a single entity (as in functional tests) offers a solution to the problem posed by joint synergy but can create problems of test specificity. The walking test proposed results from a compromise between exhibiting the level of function and reducing the potential for exacerbation of the original injury.

CHAPTER 3

Methods and Procedures

Introduction

The purpose of this investigation was to evaluate ground reaction forces generated during normal walking as measured by force plate as a functional test for ACL integrity.

Subjects

Subjects (n=14) were selected from patients of the Athletic Injuries Clinic at the University of Manitoba and the Pan Am Sports Medicine Clinic. All subjects had sustained an arthroscopically confirmed injury (second degree or greater) to the ACL in one leg only. The group consisted of 8 females and 6 males and involved ten left and four right limbs. The average age of the subjects was 23 years and the mean weight was 160 lbs. All subjects reported high levels of activity previous to the injury. Seven subjects had been forced to modify their activity levels to accommodate the injury while seven had continued to participate at their previous level. Post injury levels ranged from recreational and fitness activity, through the varsity level to the professional level. Ten subjects employed an orthopedic brace to enhance joint stability during strenuous activity but the effectiveness of the

brace was not an issue and therefore was not worn for the test.

The subjects selected for this study consisted for the most part of an athletic group who maintained high levels of activity even after their injuries. Those competing at higher levels appeared less willing to modify their activities in deference to the injury. Even though this outlook places these individuals at risk for further injury, they appeared to be more successful at coping with the instabilities resulting from the injury. There was no attempt to objectively assess the functional status of knee or to relate the subjects recovery to severity of injury, rehabilitation programs, strength, or coordination.

Since the normal or uninjured leg for each subject was to be used as the basis of comparison no control group was tested to establish normal values. Pre-injury there is no difference between left and right limbs (Hammil, Bates, & Walker, 1984). Changes are expected in the injured leg, which should be accompanied by compensatory changes in the uninjured leg, although Schneider and Chao (1983) observed no statistical difference between values for the normal leg of knee joint disease patients and the values for normal controls. There is a wide variation in the normal population but the potential differences in the present investigation were expected to be

subtle, since no subject exhibited visually dectectable pathological gait. Therefore it is quite conceivable that a pathological gait for one individual (normal clinical situation) might fall within the bounds of the average normal or a distinct normal gait for one individual might fall outside the normal bounds. Since left-right symmetry is normal, side to side difference is an appropriate indicator of dysfunction.

Comparison of the contralateral limb also allowed the inclusion of both sexes in the same group since sex differences could be eliminated as a confounding variable. Chao, Schneider, Laughman, and Stauffer (1983) observed no significant male-female differences in the shape of force curves.

Data Collection

The Mark 4 force board system employed for this study was located in the Biomedical Engineering Laboratory at the University of Manitoba. The triangular shaped plate (1.22m x 1.22m x 2.44m) is suspended by force transducers and cable between and flush with two 6m walkways. The configuration of the transducers allows measurement of forces in three directions: vertical, medial-lateral or across the board, and posterior-anterior or along the length of the board. The low-voltage signals generated by the transducers are amplified by

a DC amplifier, converted from analog to digital form by the A/D board and stored by an IBM-PC. The software employed, written by staff and students employed in the facility, included

CALDRY4 - calibrates the amplifier gain.

GAITRM2 - collects the data.

EDITRM4 - edits the the output from GAITRM2 using the calibration factors calculated by CALDRY4.

MADPLOT - controls a Hewlett Packard 7470A digital plotter that prints the edited output.

Test Protocol

Each subject made 18 traverses over the force plate. Alternating the direction of the traverses enabled a left-right alternation of footstrike on the plate. The first six traverses were designated as a learning process and were discarded. The remaining traverses (six right, six left) were retained for analysis. The subjects walked at a self-regulated comfortable walking speed to avoid artifacts that might be generated by artificially imposed gait parameters.

Data Analysis

Six traverses (3 left, 3 right) were plotted on the same axis (Appendix A) for each of the three directions; vertical, medial-lateral, and posterior-anterior. Consequently the output from

each subject was represented by two sets of axes. Cursory examination of these plots revealed apparent differences between injured and normal legs. Although the plots constituted an objective record of the forces generated, measurement of these differences was difficult due to the curvilinear nature of the data and to the imprecise nature of the plotted scales. Quanitative data is needed to statistically demonstrate that the differences are real.

Software (JOE) was developed in an attempt to describe the shape of the curve by extracting the force values at the critical points as specified by Chao, Laughman, Schneider, and Stauffer (1983) and calculating parameters such as impulse (the product of the force and the time of application) and the slope of the initial force application. Not only was this procedure crude, but the force curves were extremely ragged, rendering the data extraction process ineffective. Consequently this approach was abandoned.

Schneider and Chao (1983) advocated Fourier analysis of force plate generated data claiming that the procedure facilitated data storage, and allowed comparisons and evaluations of normal and pathological gaits. Fourier analysis involves treating the force curve as a periodic signal which can be represented as the sum of a number of sine and cosine waves. The sums of these sine and cosine waves define by

their magnitude, the shape of the curve. Data points along the curve are computed and described in terms of harmonic coefficients (A_n = cosine and B_n = sine) (Penner, 1989).

Penner (1989) attributed the ragged appearance of the force curves to vibration of the board precipitated by the subject's foot striking the board. Manning (1985) identified the inherent resonant frequencies of the forceplate used for this study as follows: 30 hz for the vertical direction, 23 hz for the medial-lateral dimension and 17 hz in the anterior-posterior direction. Thornton-Trump (1989) described a forth natural resonant frequency at 14 hz caused by a bowstring mechanism in the supporting cables. The noise is particularly troublesome in the horizontal planes in which the low amplitude of forces combined with the low resonant frequencies result in a small signal to noise ratio (Penner,1989).

Penner (1989) suggested that Fourier analysis was an ideal means of eliminating vibrational noise from the force data since the offending frequencies, if they are known to be outside the range of intrest, can simply be eliminated. The elimination of the noise causing frequencies results in no loss of information since the motions involved in walking are of a low frequency (ie below 10hz) (Penner, 1989). Consequently only the first few (2-4) harmonics are the dominant harmonics required to accurately describe a force record (Schneider &

Chao, 1983). However increasing the number of harmonics increases the resolution of the plot (Chao, Laughman, Schneider, & Stauffer, 1983; Schneider & Chao, 1983). Since the noise inducing frequencies of the force plate in question are 14 hz or greater, 14 hz was chosen as the cutoff point. This frequency first appeared in the 12th harmonic. In order to produce the greatest resolution with the least noise the first 11 harmonics for each subject were analyzed.

A modified version of Penner's program SMURFT was employed to execute the discrete Fourier transform (dft) on the edited data (EDITRM4) producing a sequence of Fourier coefficients for the first 15 harmonics. The coefficients generated were as follows:

a. Real (A_n) -- the cosine function

b. Imaginary (B_n) -- the sine function

c. the magnitude of the coefficients

$$mag = \sqrt{A_n^2 + B_n^2}$$

d. the phase shift in the harmonic

phase = the angle formed by the magnitude and the horizontal axis

In addition to calculating the above, the program also normalized these values as a percentage of the subject's body weight and eliminated the superfluous zeros at the beginning and end of each plot.

Each traverse (14 sub x 12) was processed three times using the SMURFT program so as to generate coefficients for three directions of force application. The output from SMURFT was then grouped according to the force dimension resulting in three data sets each containing 168 tables.

The data was translated from the MS-DOS format used in the IBM-PC and processed using Apple Macintosh hardware and the STATVEIW 512 SE software package. The 504 (168x3) tables containing the FOURIER coefficients were categorized as injured and normal. Adopting the procedure tested and validated by Chao et al. (1983), typical curves for each limb were calculated by finding the means of the six traverses. Although this procedure involved combining different frequencies it was invoked to minimize variability within subject. Note that frequency variability increases in the higher harmonics, but this occurs because the harmonics are multiples of the fundamental frequencies. Also note that the Fourier analysis describes the curve as a function of the harmonics not the frequency. The data set for each direction of force application now consisted of 14 typical curves for each of the injured and normal legs. At this time all harmonics beyond the eleventh were deleted.

The 14 typical curves for each leg, each containing data for 11 harmonics, were combined and sorted by harmonic number

to produce 11 subsets containing the data for the respective harmonic for each of the fourteen subjects . The corresponding harmonics for injured and normal legs were merged producing eleven subsets of paired data in each force direction . Variables; real, imaginary, magnitude, and phase were compared using a paired t-test for the 11 harmonics. Frequency was compared only for the first harmonic since differences in the higher harmonics would have only been multiples of the fundamental difference.

CHAPTER 4

Results and Discussion

Force curves for 12 traverses for each of the 14 subjects were plotted resulting in a total of 504 separate curves (Appendix A). Figures 24, 25, and 26 are typical curves for three subjects reproduced to serve as examples. Each figure contains three sets of axes representing the vertical, medial-lateral, and posterior-anterior forces measured by the force plate. Two traverses, one of the injured leg and one for the normal leg are plotted in contrasting colors on each axis (injured-red, normal-blue). Force (y axis) is plotted as a function of time which has been normalized on the x axis so as to facilitate comparisons between the curves.

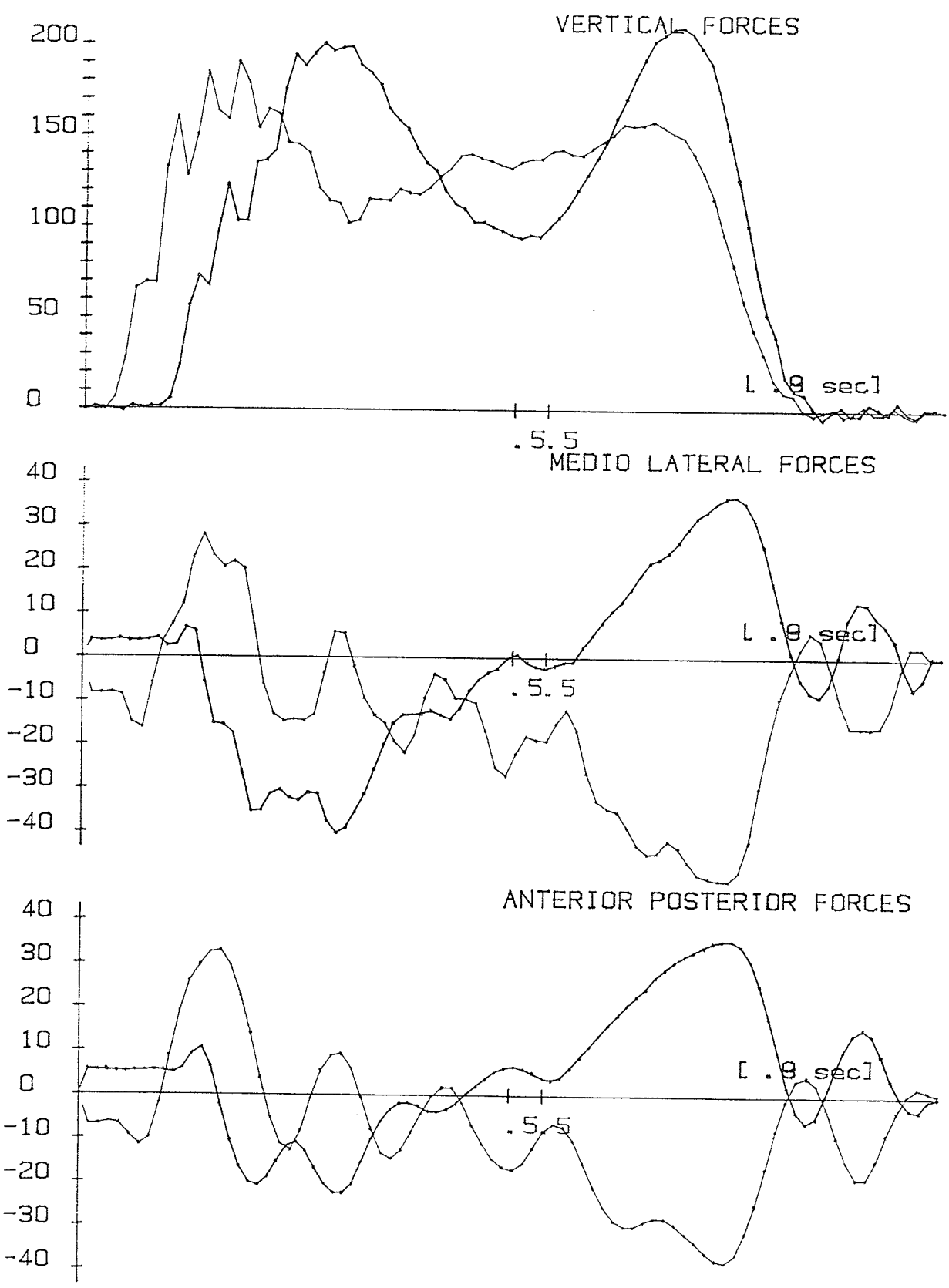

Figure 24 left and right traverses for subject AL
red = left = injured

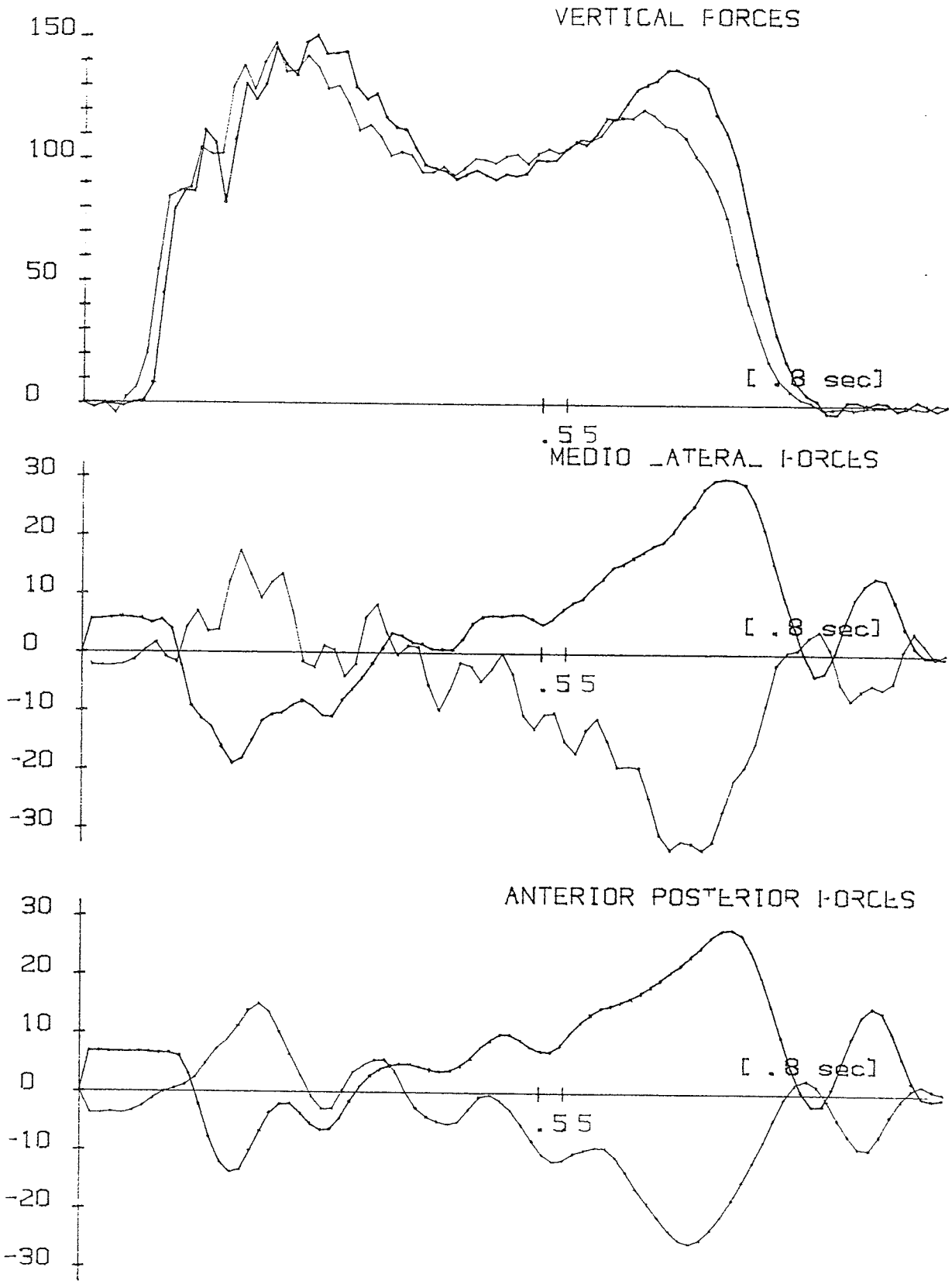

Figure 25 left and right traverses for subject SH
red = left = injured

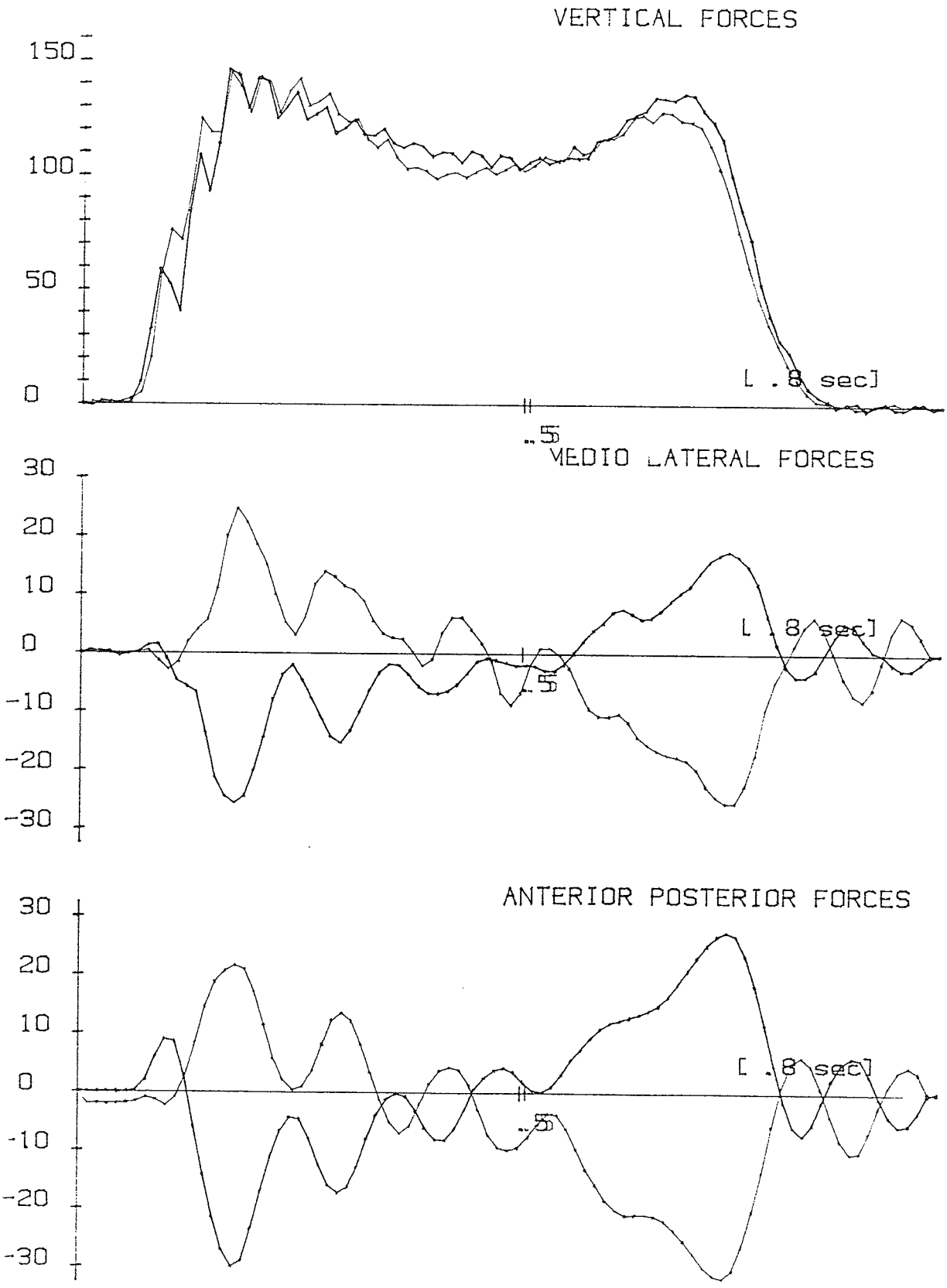

Figure 26 left and right traverses for subject TA
red = left = injured

Not all the curves in figures 24-26 begin in the at 0 time on the plots due to an artifact in the data collection software (GAITRM2). The program constantly samples 10 points at a time. If the recording mechanism is triggered at any point during a sample points preceding the first actual force input are recorded as zeros. These irrelevant zeros were eliminated in the Fourier transform performed subsequent to the plotting routine.

Visual inspection of the force curves revealed a high degree of similarity between curves for the same leg in each subject. Inter-subject variability was quite extensive while variability between limbs for the same subject was spread over a wide spectrum. The curves were ragged and uneven, especially immediately following heelstrike. Vibration appeared to diminish as the traverse progressed but reappeared after the subject's foot had left the board.

Vertical Forces

Vertical force curves for the normal or uninjured limbs of all subjects resembled the normal bimodal curves described by Hammil et al. (1984), Chao et al. (1983), Chao and Schneider (1983) and Stauffer et al. (1977). Although the curves for the injured limbs (figures 24-26) are similar in shape they tend to be flatter and the forces appear to be smaller. Note that vertical forces are in the same direction regardless of limb or

direction of travel rendering them indistinguishable as to limb or direction of travel unless there is prior knowledge. Because forces generated in the vertical direction involve support of the entire body weight they are significantly larger than those generated in other directions.

Medial-Lateral Forces

The medial-lateral forces generated by the contralateral limbs are opposite in direction and consequently force curves for left and right form mirror images. In figures 24-26, the left leg was injured in each case and was represented by a curve which changed from positive (medial force) to negative (lateral force). Irregularities in shape due to the effects of vibration were more significant due to the smaller forces generated in the medial and lateral directions. There appeared to be a large vibrational component after the subject's foot had left the board.

The large vibrational component in the medial-lateral curves obscures some of the differences but the injured leg appears to generate slightly larger forces. The curves also appear to be less regular for the injured limb.

Posterior-Anterior Forces

Force curves for the posterior-anterior direction resemble medial-lateral curves in both shape and magnitude. They appear as mirror images because the direction of travel was alternated in the test protocol. A large vibrational component was observed after the subject's foot had left the board. The visable differences between injured and normal limbs (figures 24-26) appear to be differences of shape. The curve for the injured leg appears broader and more irregular than that of the normal leg.

Fourier Analysis

Although there are apparent differences between force curves for injured and normal limbs the significance of the differences is dependent on the judgement of the observer. In terms of functional change, force curve interpretation is of limited value unless the changes can be measured. In the present format, measurement of forces was improbable if not impossible. The Fourier transform was employed to resolve this situation by converting the data to a quantifiable state. The transform was performed on all of the 504 force curves generated by the subjects.

Tables 2, 3, and 4 contain the the Fourier coefficients which represent the vertical, the medial-lateral and the posterior-anterior force curves generated by one subject (SQ) during one traverse (normal leg) after the frequencies above 14 hz have been eliminated. The frequency listed for harmonic #1 is the fundamental frequency and all subsequent frequencies are multiples of this fundamental frequency. Values for the coefficients exhibit a number of characteristics. The magnitude of coefficients (mag) is always positive and tends to be largest in lower harmonics and tapers to values which are less than 10% of the initial values by the tenth harmonic. Vertical magnitudes (like vertical forces) are larger than those in the other directions. Sine (imag) and cosine (real) coefficients can either be negative or positive and large values in one are matched by small values in the other. Phase shift appears to alternate between series of negative and positive values.

Table 2 Fourier transforms for vertical forces of 1 traverse

	harm	freq	real	imag	mag	phase
1	1	.980	-29.064	2.298	29.155	-.079
2	2	1.961	-21.175	.309	21.177	-.015
3	3	2.941	6.037	1.007	6.121	.165
4	4	3.922	10.600	-2.728	10.945	-.252
5	5	4.902	.311	-.745	.807	-1.176
6	6	5.882	-2.046	1.711	2.667	-.696
7	7	6.863	-1.918	1.261	2.295	-.582
8	8	7.843	-.552	-.537	.771	.772
9	9	8.824	.652	-1.228	1.390	-1.083
10	10	9.804	.623	-.086	.629	-.137
11	11	10.784	.031	.361	.362	1.485
12	12	11.765	.014	-.006	.016	-.426
13	13	12.745	-.278	-.073	.288	.256
14	14	13.725	.206	-.169	.267	-.687

Table 3 Fourier transforms for medial-lateral forces of 1 traverse

	harm	freq	real	imag	mag	phase
1	1	.980	.398	7.590	7.600	1.518
2	2	1.961	1.904	-1.561	2.462	-.687
3	3	2.941	.181	-4.126	4.130	-1.527
4	4	3.922	-.594	-.261	.649	.413
5	5	4.902	-.213	2.269	2.279	-1.477
6	6	5.882	-.136	1.278	1.286	-1.464
7	7	6.863	.592	-.664	.890	-.843
8	8	7.843	.999	-1.743	2.009	-1.050
9	9	8.824	.192	.437	.478	1.157
10	10	9.804	-.500	-.489	.700	.774
11	11	10.784	-.066	.105	.124	-1.010
12	12	11.765	-.343	-.142	.371	.391
13	13	12.745	-.078	-.196	.211	1.191
14	14	13.725	-.016	-.124	.125	1.440

Table 4 Fourier transforms for posterior-anterior forces of 1 traverse

	harm	freq	real	imag	mag	phase
1	1	.980	-.322	6.455	6.464	-1.521
2	2	1.961	.824	-1.392	1.618	-1.037
3	3	2.941	.263	-3.402	3.413	-1.494
4	4	3.922	-.117	-.140	.182	.877
5	5	4.902	-.111	1.991	1.994	-1.515
6	6	5.882	-.220	1.114	1.135	-1.376
7	7	6.863	.404	-.721	.827	-1.060
8	8	7.843	1.063	-1.658	1.970	-1.001
9	9	8.824	.274	.609	.668	1.148
10	10	9.804	-.565	-.482	.743	.706
11	11	10.784	-.098	-.062	.116	.568
12	12	11.765	-.231	-.168	.285	.629
13	13	12.745	-.050	-.228	.234	1.356
14	14	13.725	.011	-.013	.018	-.860

Typical curves for each leg of each subject were produced by averaging the coefficients for the six traverses for each leg. Tables 5, 6, and 7 contain the typical curves for both injured and normal legs for one subject (SQ). Coefficients for typical curves display the same types of relationships as the individual curves. In order to facilitate comparison of injured to normal, the corresponding values for each the four variables (real, imag, mag, phase) were plotted as a function of the harmonic. Comparison of the plots (figures 27-38) reveals relationships similar to those presented by the force curves. Coefficients (real, imag) for vertical forces (figures 27,30) of contralateral legs are indistinguishable without prior knowledge because vertical forces are in the same direction for both limbs. Medial-lateral and posterior-anterior coefficients (real, imag) (figures 28,29,31,32) suggest mirror images for contralateral limbs. The magnitude of the coefficients (figures 33,34,35) is always positive and the values are similar display a similar range. The phase shift plots (figures 36,37,38) reveal similar patterns with differences of amplitudes and timing.

Table 5 Fourier transforms for subject SQ - vertical forces

	i-freq	n-freq	i-real	n-real	i-imag	n-imag	i-mag	n-mag	i-phase	n-phase
1	1.151	1.153	-.971	1.290	-4.475	4.840	4.659	5.144	.834	.769
2	2.303	2.326	-.222	.144	.559	.069	.701	.160	-.252	.445
3	3.453	3.458	1.012	-1.160	1.605	-1.364	2.284	2.343	-.053	-.284
4	4.605	4.610	.712	-1.048	-.203	.155	.862	1.150	-.171	.138
5	5.756	5.763	-.422	-.131	-.586	.710	1.038	.964	-.387	-.637
6	6.907	6.915	-.735	.402	.291	-.077	1.016	1.026	-.299	-.170
7	8.058	8.068	-.660	1.038	.837	-.848	1.115	1.506	-.845	-.779
8	9.209	9.220	-.001	.328	.401	-.527	.664	.752	.056	-.570
9	10.361	10.373	.235	.207	.048	-.062	.441	.598	-.299	.608
10	11.512	11.525	.247	-.142	-.038	-.189	.296	.342	-.484	-.398
11	12.663	12.393	.058	-.078	-.027	-.071	.102	.151	.160	-.187
12	12.988	12.654	.042	-.192	-.012	-.160	.112	.256	.425	.667

Table 6 Fourier transforms for subject SQ - medial-lateral forces

	i-freq	n-freq	i-real	n-real	i-imag	n-imag	i-mag	n-mag	i-phase	n-phase
1	1.151	1.138	-.157	1.917	-5.543	5.782	5.722	6.179	.492	1.238
2	2.303	2.276	.517	1.259	.210	-.697	1.283	1.158	-.054	-.451
3	3.061	3.414	.982	-1.337	1.991	-2.052	2.810	2.892	.023	-.150
4	4.605	4.552	.790	-1.341	.012	-.022	1.017	1.436	-.241	.113
5	5.756	5.102	-.326	-.403	-.665	1.810	1.117	1.855	-.397	-1.352
6	6.907	6.828	-.699	.647	.283	.096	1.102	1.204	-.322	-.062
7	8.058	7.966	-.648	1.046	.782	-.607	1.072	1.374	-.785	-.588
8	9.209	9.104	-.009	.155	.410	-.632	.677	.784	.087	-.127
9	10.361	10.242	.205	.135	.126	-.159	.403	.592	.089	.493
10	11.512	11.380	.195	-.134	.018	-.149	.244	.291	.207	-.144
11	12.663	12.518	.067	.067	-.021	-.069	.085	.196	-.186	-.715
12	12.988	12.645	.041	-.240	-.016	-.105	.085	.306	.404	.360

Table 7 Fourier transforms for subject SQ - posterior anterior forces

	i-freq	n-freq	i-real	n-real	i-imag	n-imag	i-mag	n-mag	i-phase	n-phase
1	1.151	1.152	-23.285	-20.834	4.796	6.081	24.326	22.353	-.240	-.336
2	2.303	2.305	-13.343	-13.185	10.849	11.475	18.555	19.672	-.652	-.703
3	3.453	3.458	4.683	3.259	-.119	1.685	5.035	4.116	-.063	.533
4	4.605	4.610	1.405	1.727	-3.751	-2.888	5.303	5.473	-.256	.626
5	5.319	5.763	-.171	-1.517	-1.901	-.611	1.908	2.144	1.481	-.321
6	6.907	6.915	-.814	-1.539	.705	.631	1.143	1.778	-.350	-.335
7	8.058	8.068	-.143	-.616	.197	.256	.542	1.048	.077	-.492
8	9.209	9.220	.170	.009	-.174	-.371	.401	.613	-.442	.386
9	10.361	10.373	-.008	.276	.080	-.379	.248	.545	.043	-.560
10	11.512	11.525	-.132	-.020	.153	.077	.431	.343	-.862	.161
11	12.663	12.678	-.025	.087	.001	.060	.495	.286	-.185	.526
12	12.988	12.645	.304	-.004	-.100	-.083	.583	.137	.659	.214
13	13.548	13.245	.081	.047	-.127	-.130	.441	.272	-.035	-.347

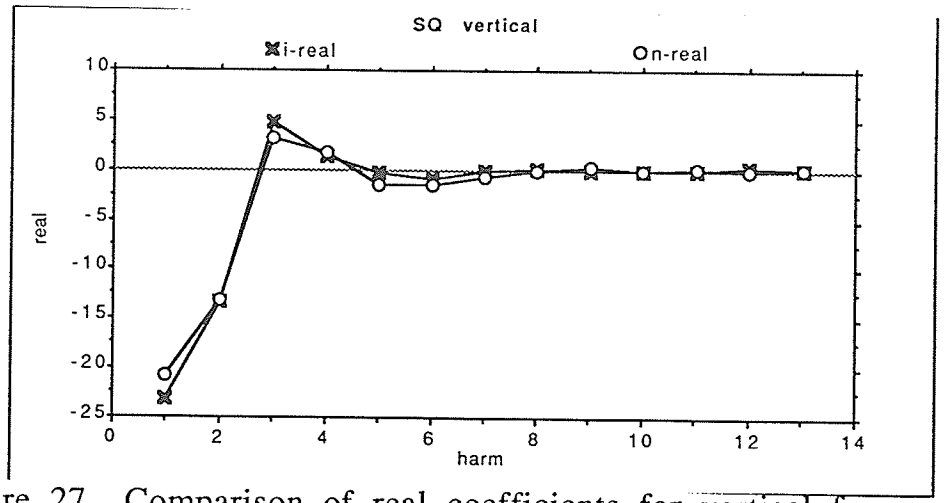

Figure 27 Comparison of real coefficients for vertical forces - injured vs normal subject SQ

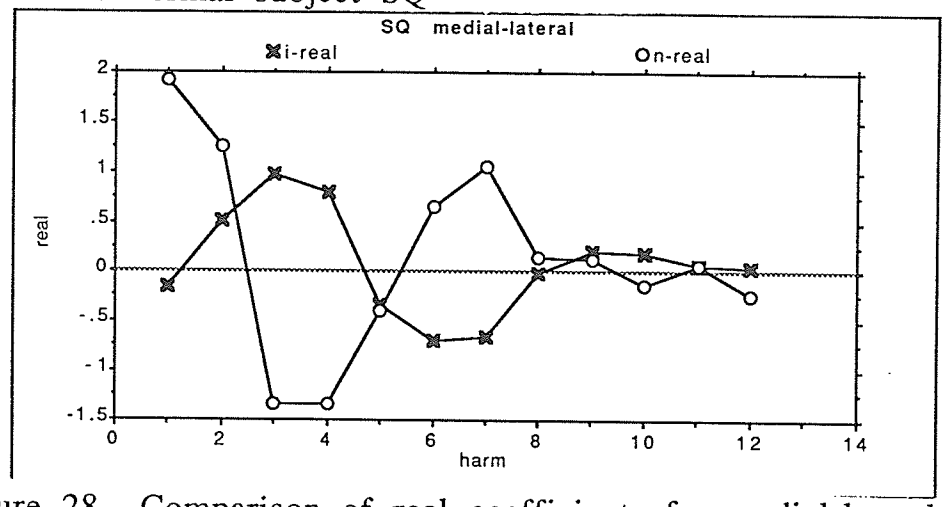

Figure 28 Comparison of real coefficients for medial-lateral forces -injured vs normal subject SQ

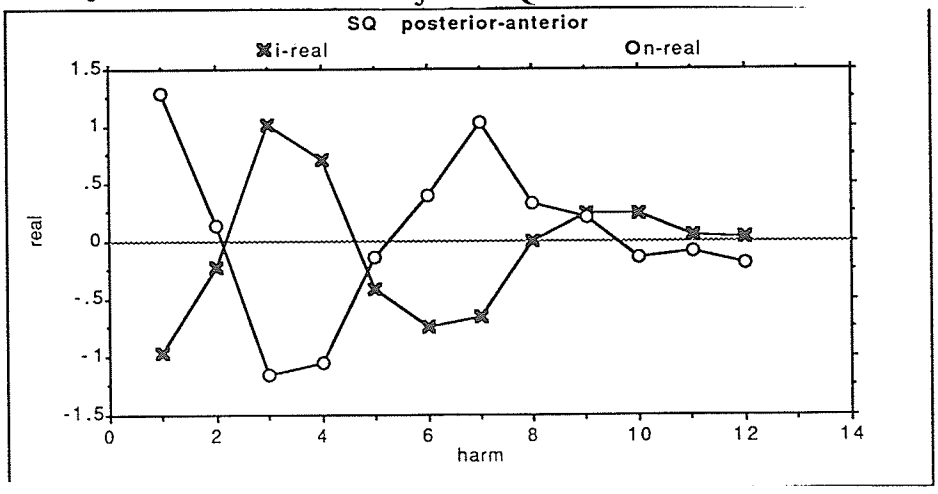

Figure 29 Comparison of real coefficients for posterior-anterior forces -injured vs normal subject SQ

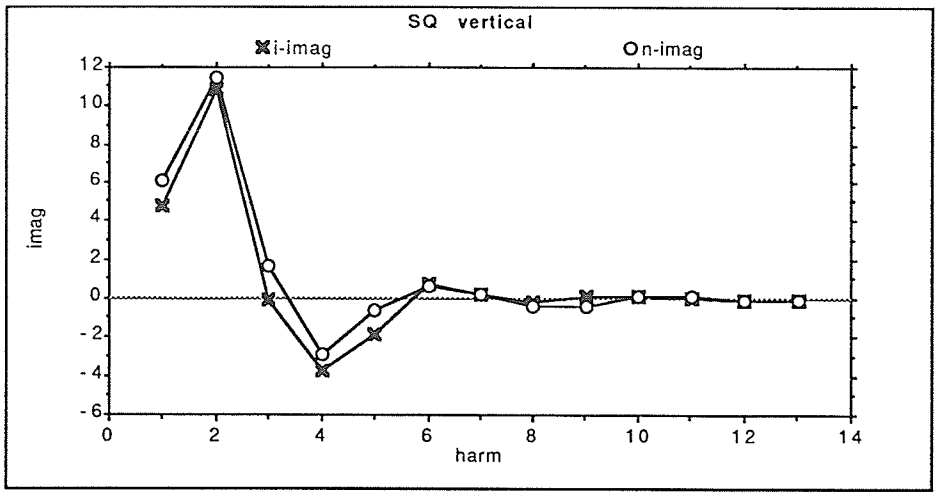

Figure 30 Comparison of imaginary coefficients for vertical forces -iniured vs normal subiect SO

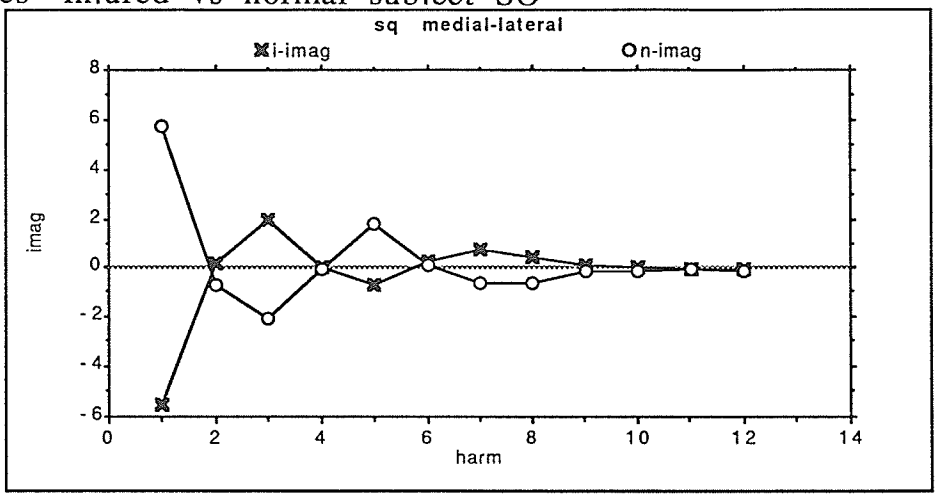

Figure 31 Comparison of imaginary coefficients for medial-lateral forces -iniured vs normal subject SQ

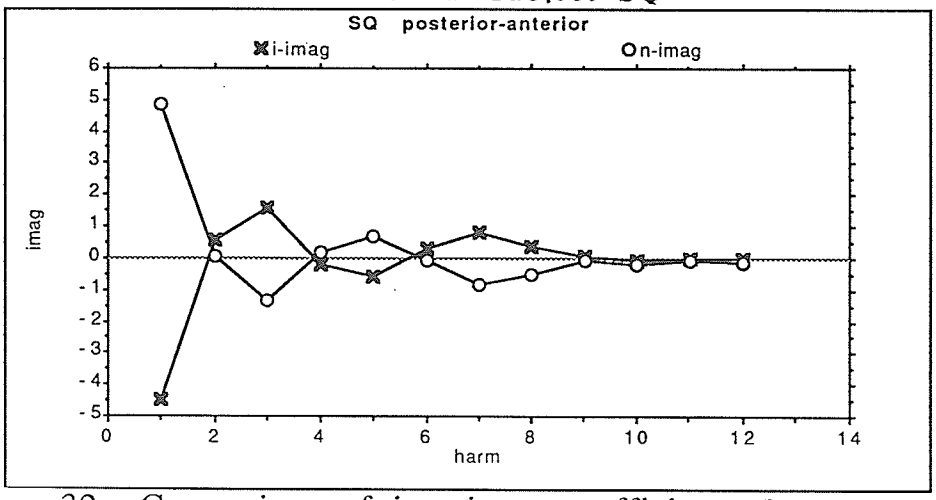

Figure 32 Comparison of imaginary coefficients for posterior-anterior forces -injured vs normal subject SQ

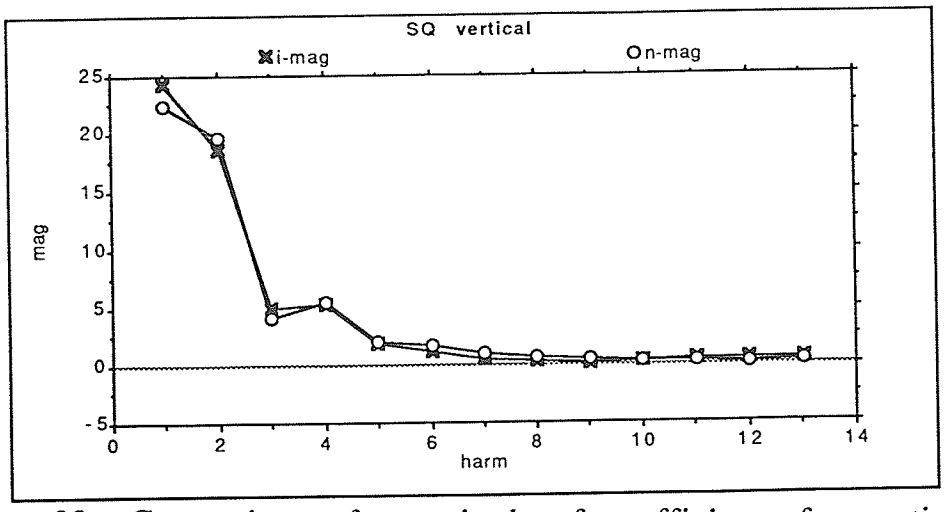

Figure 33 Comparison of magnitude of coefficients for vertical forces -injured vs normal subject SQ

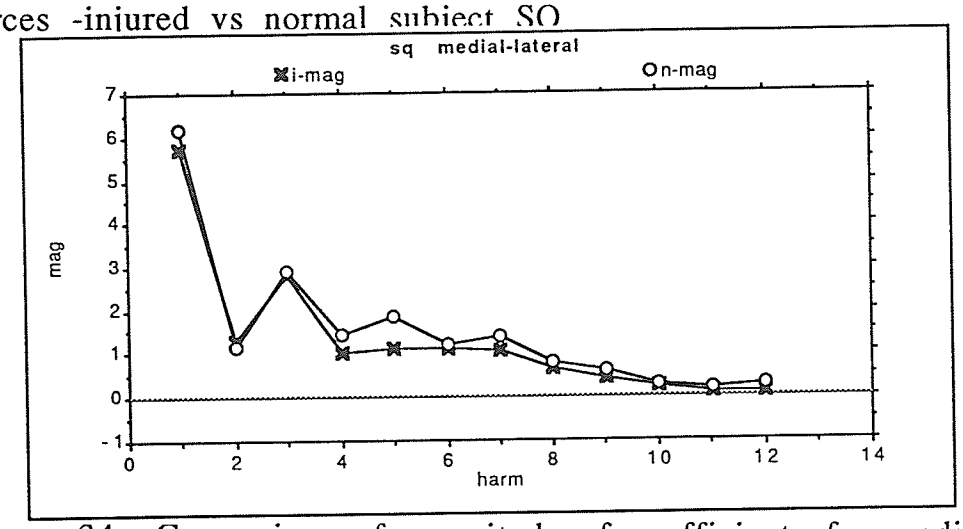

Figure 34 Comparison of magnitude of coefficients for medial-lateral forces -injured vs normal subject SQ

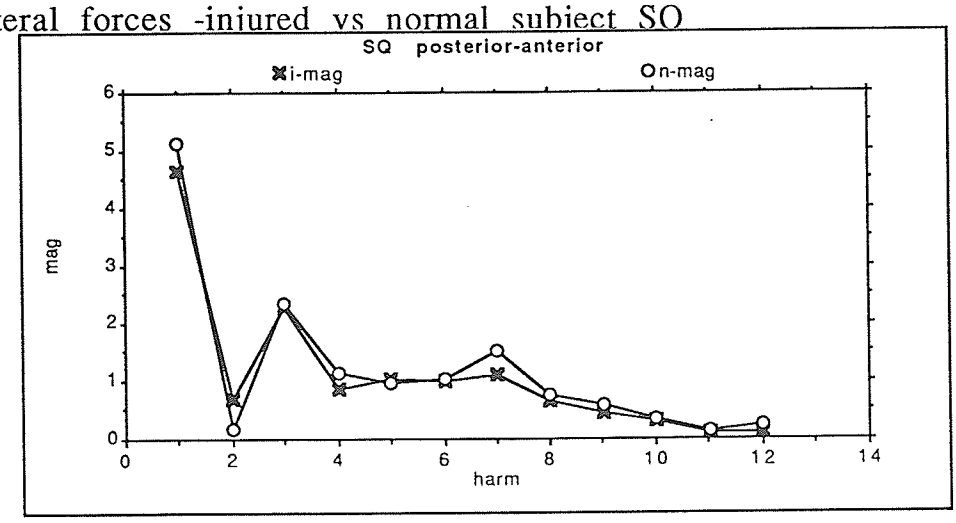

Figure 35 Comparison of magnitude of coefficients for posterior-anterior forces -injured vs normal subject SQ

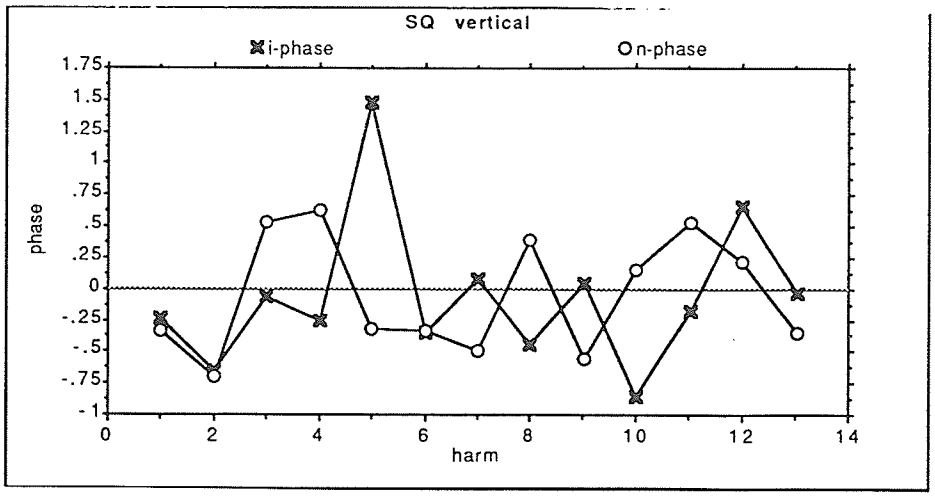

Figure 36 Comparison of phase shift for vertical forces -injured vs normal subject SO

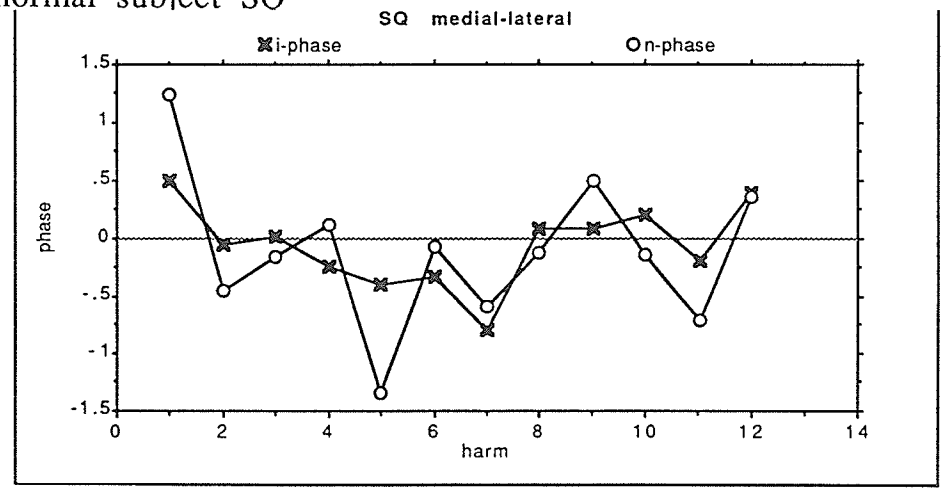

Figure 37 Comparison of phase shift for medial-lateral forces - injured vs normal subject SQ

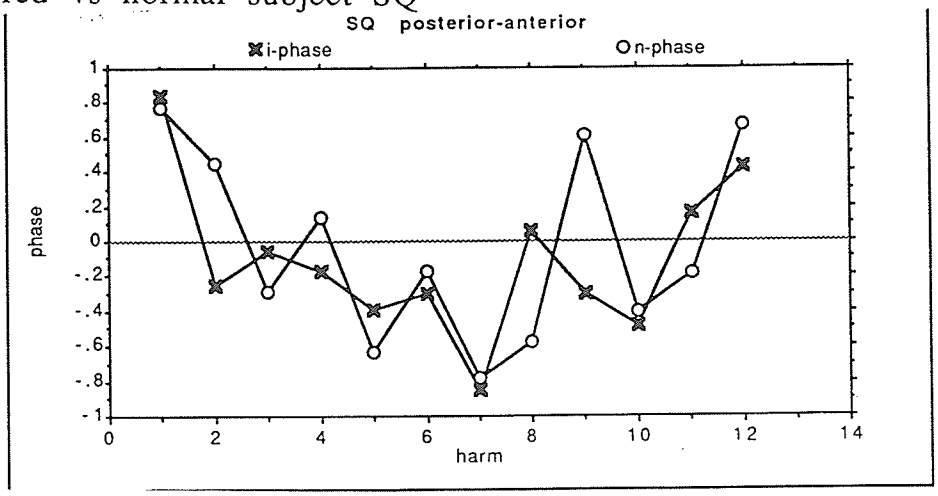

Figure 38 Comparison of phase shift for posterior-anterior forces -injured vs normal subject SQ

In order to compare differences for the group the typical curves were combined and separate tables (33) for each harmonic in each direction were generated. Tables 8, 9, and 10 contain respectively the Fourier coefficients for the first harmonic vertical forces, medial-lateral forces, and posterior-anterior forces. Each line of a table contains the typical values for both injured and normal legs of one subject. This format facilitates the administration of the paired t-tests.

Table 8 Fourier transforms for injured and normal legs vertical forces of all subjects - harmonic #1

	i-freq	n-freq	i-real	n-real	i-imag	n-imag	i-mag	n-mag	i-phase	n-phase
1	1.173	1.119	-16.525	-22.105	10.074	6.470	19.400	23.621	-.550	-.322
2	1.055	1.086	-25.155	-22.430	6.773	8.967	26.439	24.543	-2.810	-.401
3	1.166	1.166	-18.812	-20.837	7.071	6.596	20.691	22.223	-.407	-.326
4	1.161	1.197	-19.396	-20.570	9.828	5.197	21.812	21.766	-.475	-.288
5	1.081	1.129	-23.196	-18.447	5.021	8.565	23.840	20.472	-.221	-.444
6	1.175	1.192	-18.010	-15.302	7.048	7.706	19.594	17.245	-.391	-.477
7	1.093	1.020	-20.401	-22.160	8.128	8.306	22.247	23.666	-.394	-.359
8	1.190	1.151	-20.550	-21.933	8.406	8.729	22.553	23.896	-.407	-.394
9	1.092	1.159	-17.944	-22.207	17.358	7.321	25.928	24.489	-.735	-.384
10	1.216	1.122	-18.567	-23.269	10.220	9.454	22.133	25.589	-.546	-.409
11	1.223	1.151	-18.730	-20.648	4.676	9.206	19.956	22.968	-.261	-.431
12	1.151	1.152	-23.285	-20.834	4.796	6.081	24.326	22.353	-.240	-.336
13	1.100	1.020	-23.256	-27.870	7.973	4.141	24.931	28.176	-.345	-.148
14	1.067	1.096	-16.326	-20.316	2.761	10.204	18.144	23.042	-.568	-.487

Table 9 Fourier transforms for injured and normal legs medial-lateral forces of all subjects - harmonic #1

	i-freq	n-freq	i-real	n-real	i-imag	n-imag	i-mag	n-mag	i-phase	n-phase
1	1.173	1.119	3.252	-.036	3.930	-5.618	5.116	5.738	.881	-.523
2	1.055	1.073	.240	.535	-2.997	2.957	3.069	3.036	-.445	1.388
3	1.166	1.166	1.398	-.033	2.900	-4.105	4.452	4.057	.782	-.006
4	1.166	1.197	2.557	-.807	3.528	-5.254	4.944	5.502	.790	-.147
5	1.081	1.129	.140	1.449	-4.775	3.927	3.832	4.217	-.014	1.243
6	1.175	1.192	.936	-1.630	3.849	-3.336	3.991	3.751	1.330	1.118
7	1.117	.907	2.114	-.081	4.080	-4.851	4.636	4.922	1.093	.907
8	1.190	1.151	2.394	-.631	5.015	-4.003	5.626	6.056	1.123	.817
9	1.092	1.159	4.613	-.634	3.787	-5.747	6.202	6.081	6.840	.425
10	1.248	1.122	1.759	-.847	3.115	-.411	3.680	4.297	.547	.315
11	1.223	1.151	.814	-.823	3.421	-3.367	3.633	3.506	.821	.809
12	1.151	1.138	-.157	1.917	-5.543	5.782	5.722	6.179	.492	1.238
13	1.100	1.090	.618	.006	3.555	-.308	3.632	3.134	1.398	-.519
14	1.067	1.096	-.399	1.901	-4.157	3.863	4.240	4.362	-.095	1.110

Table 10 Fourier transforms for injured and normal legs posterior-anterior forces of all subjects - harmonic #1

	i-freq	n-freq	i-real	n-real	i-imag	n-imag	i-mag	n-mag	i-phase	n-phase
1	1.173	1.119	2.087	-.528	3.511	-4.195	4.093	4.306	1.036	.400
2	1.055	1.073	-.356	.425	-2.250	2.363	2.317	2.437	.893	.867
3	1.166	1.166	.881	-.380	2.229	-2.943	3.518	3.016	.816	-.135
4	1.161	1.197	1.920	-1.321	3.648	-4.412	4.307	4.742	.991	.752
5	1.081	1.129	-.881	1.729	-3.780	4.026	3.909	4.402	1.345	1.169
6	1.175	1.192	.835	-1.866	3.428	-3.487	3.356	3.973	1.332	1.082
7	1.093	1.044	.365	-.693	3.335	-3.170	3.497	3.279	1.288	.723
8	1.190	1.151	1.408	-.856	3.112	-2.130	3.453	3.522	1.147	1.180
9	1.092	1.159	2.657	-.771	2.574	-3.008	3.836	3.239	.804	.283
10	1.216	1.122	1.360	-.792	1.078	-3.005	3.599	3.166	.335	1.317
11	1.223	1.151	.730	-1.310	3.758	-3.386	3.949	3.670	.861	1.207
12	1.151	1.153	-.971	1.290	-4.475	4.840	4.659	5.144	.834	.769
13	1.100	1.090	1.018	-.396	3.958	-4.153	4.122	4.229	1.318	-.090
14	1.067	1.096	-.751	1.325	-3.463	3.071	3.584	3.391	1.356	1.159

Table 11 summarizes the results of the 135 (11 harmonics x 4 coefficients x 3 directions + 3 fundamental frequencies) paired t-tests. None of the tests of the fundamental frequencies disclosed any significant differences. There are significant differences between the two limbs throughout the frequency domain in question but harmonics four and six contain the most changes. Changes to real ($A_{n)}$ the cosine coefficient are limited to the first four harmonics, while changes to imaginary ($B_{n)}$ the sine coefficient don't commence until the fourth harmonic and continue through the eighth. Significant differences of magnitude are observed in harmonics two, four and six. The phase shift remains stable until higher the frequencies of harmonics nine and ten are reached.

TABLE 11

Differences of Fourier coefficients between injured and normal legs

harm	vertical	med-lat	post-ant
1	ns	real (t=2.39, p=.016)	real (t=1.85, p=.044)
2	real (t=1.76, p=.05) mag (t=4.02, p=.0007)	ns	ns
3	ns	real(t=1.927 p=.038)	ns
4	imag (t=2.59, p=.01) mag (t=1.87, p=.04	imag (t=2.59, p=.01)	imag(t=2.23, p=.02)
5	imag (t=2.94, p=.006)	real (t=1.94 p=.04	ns
6	imag (t=2.34 p=.02)	imag (t=2.42 p=.02)	imag (t=1.94 p=.04 mag (t=1.73 p=.05)
8	imag (t=2.18 p=.02)	imag (t=2.47 p=.01)	ns
9	phase (t=1.91 p=.04)	ns	ns
10	phase (t=2.36 p=.02)	ns	ns

If the differences are grouped by harmonics, the fourth and sixth contain the most differences. This pattern may prove significant for identification of ACL injury especially if it coincides with the strong magnitude change in harmonic #2. The majority of the differences are found in the first eight harmonics suggesting that the decision to eliminate frequencies above 14 hz was valid. The results of the paired t-tests presented in Table 11 will be discussed in terms of the direction of the forces and conceivable gait changes.

Vertical Differences

Schneider and Chao suggested, that for vertical forces the most dominant terms (the ones which governed the shape of the curve) were A_1, A_2, A_3. Differences in vertical forces were observed as follows:

a. real (A_n) harmonic #2

b. imag (B_n) harmonics #4,#5,#6,#8

c. magnitude harmonics #2, #4

d. phase shift harmonics #9, #10

The difference in one of three coefficients that govern the shape of the curve (A_n), suggests that the changes may be neither extensive nor pronounced but are nevertheless real. The differences in magnitudes in harmonics #2 and#4 might also prove important since the variable contains elements of both the sine and cosine coefficients. The change in harmonic #2

was highly significant (t=4.02, p=.0007), suggesting a consistent difference in all subjects. The differences for the sine coefficient in the middle frequencies are highly significant and tightly grouped. These are not dominant coefficients but they must exert some influence on the shape of the curve and as such are potentially valuable indicators for change. The changes in phase shift occur at higher frequencies.

Differences often encountered in the vertical curves included flattening of the curve so that it becomes more like a normal distribution than the bimodal curve typical of the normal subject, shifting of the curve either left or right, and changes in the magnitude of the forces. A flatter curve for the injured limb suggests that the subject was attempting to minimize vertical accelerations. The ACL has no capability to resist forces in the vertical direction so the changes are more likely due to the loss of normal joint motion, specifically the rolling gliding motion. The high frequency changes observed in the phase shift are possibly indicative of rapid muscle activity which might be construed as an attempt to stabilize the rolling-gliding mechanism.

Medial-Lateral Differences

Schneider and Chao (1983) reported that the dominant terms for medial-lateral forces were A_1, and A_2. Differences observed in the medial-lateral forces curves were as follows:

a. real (A_n) -- A_1, A_2, A_3

b. imag (B_n) -- B_4, B_6, B_8

The changes appear to alternate between the sine and cosine functions as though the subjects were struggling for stability possibly utilizing contractions of alternating muscle groups. ACL function in a medial-lateral direction is most likely to be directed towards resisting rotary instability and the observed changes may stem from an increase in this action due to loss of ACL function.

Posterior-Anterior Differences

The governing terms B_1, B_2, (Schneider & Chao, 1983) exhibited no significant differences for the posterior-anterior force curves. Differences were observed for the cosine coefficient (A_n) in the first harmonic, the sine coefficient in harmonics #4 and #6, and for magnitude #6. ACL function in the saggital plane is concerned with resisting anterior tibial translation and controlling the rolling gliding mechanism. Tibial translation is less a problem in a weight bearing situation due to the stabilizing action of the compressive forces in the joint so the differences are likely linked to control of the rolling gliding mechanism.

Transformation of the data from a time domain to the frequency domain requires a substantial conceptual adjustment. Relationships between the changes in the Fourier

coefficients and the changes in the shape of the curves has not been documented. Consequently the preceding discussion must be regarded as conjecture until further investigation in this area is completed.

CHAPTER 5

Summary & Conclusions

The purpose of this study was to evaluate the measurement of ground reaction forces using a force plate as a functional test for ACL insufficiency. Apparent differences were observed when ground reaction forces measured by the force plate were plotted as a function of time. The differences could not be measured using conventional scales included in the plots or software developed for this purpose because forces generated by vibration in the force platform were included in the plots. Fourier analysis, which involves transforming the data from the force-time domain to the frequency domain was utilized to overcome this problem.

The Fourier coefficients, once calculated, proved to be an effective solution to the problem of comparing curves for injured and normal legs. Not only could the vibrational noise be removed but the discrete values for each harmonic provided direct means of comparison. The only drawback appears to be a disassociation between the Fourier coefficients and the mechanics of walking.

The original intent of this study was to evaluate the force plate with the intent of utilizing the instrument in a diagnostic capacity. Although the test is able to detect

differences in the ACL deficient limb, it represents at best circumstantial evidence for ACL insufficiency. This is not at present, substantial enough to justify the time and expense involved when direct evidence can be obtained through the use of the arthroscope. No tests have been conducted to determine if different injuries produce the same results, consequently the specificity of the test is unknown. Other factors such as expense and and aquisition of equipement might also restrict the use of the force plate for this application.

This does not mean that there is not a place in sports medicine for the force plate. The differences reported were significant, The objective results allow comparison over time, and the protocol used was simple, noninvasive, and easily tolerated. Tibone et al. (1988) reported that the differences observed in a prior investigation had disappeared two years later when the recovery was apparently complete. An appropriate application of the test lies in the treatment and rehabilitation phase of the injury. As recovery progresses and function returns to normal the differences between contralateral limbs theoretically should diminish. At some point the test would indicate a complete or terminal stage of recovery which would allow return to activity.

Conclusions

1. Individuals suffering ACL insufficiency demonstrate different gait patterns in the affected leg than in the normal leg.

2. The force plate is an appropriate instrument for measuring these differences.

3. Fourier analysis of the force plate data is the appropriate means to dectect changes.

4. The protocol employed in this investigation has potential as a tool to monitor rehabilitation and recovery following injury.

Recommendations

To this end recommendations for further study include reformulation of the force curves from the sanitized Fourier coefficients and comparison to the original curves, investigation of the relationship between the Fourier coefficients and the force curves, identification of force curves for different injuries, and longitudinal studies involving recovery from injury.

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APPENDIX A

Force curves for all subjects

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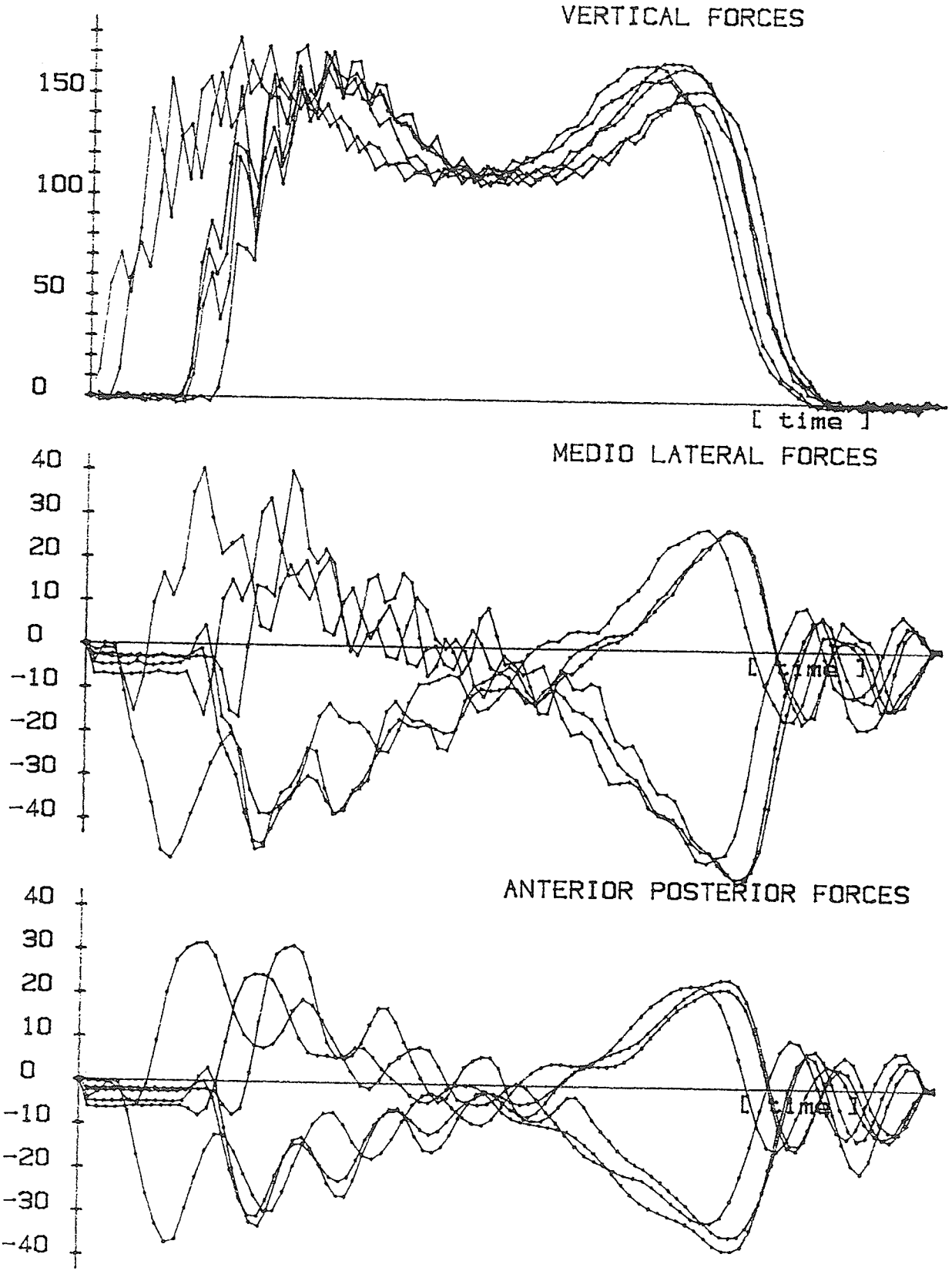

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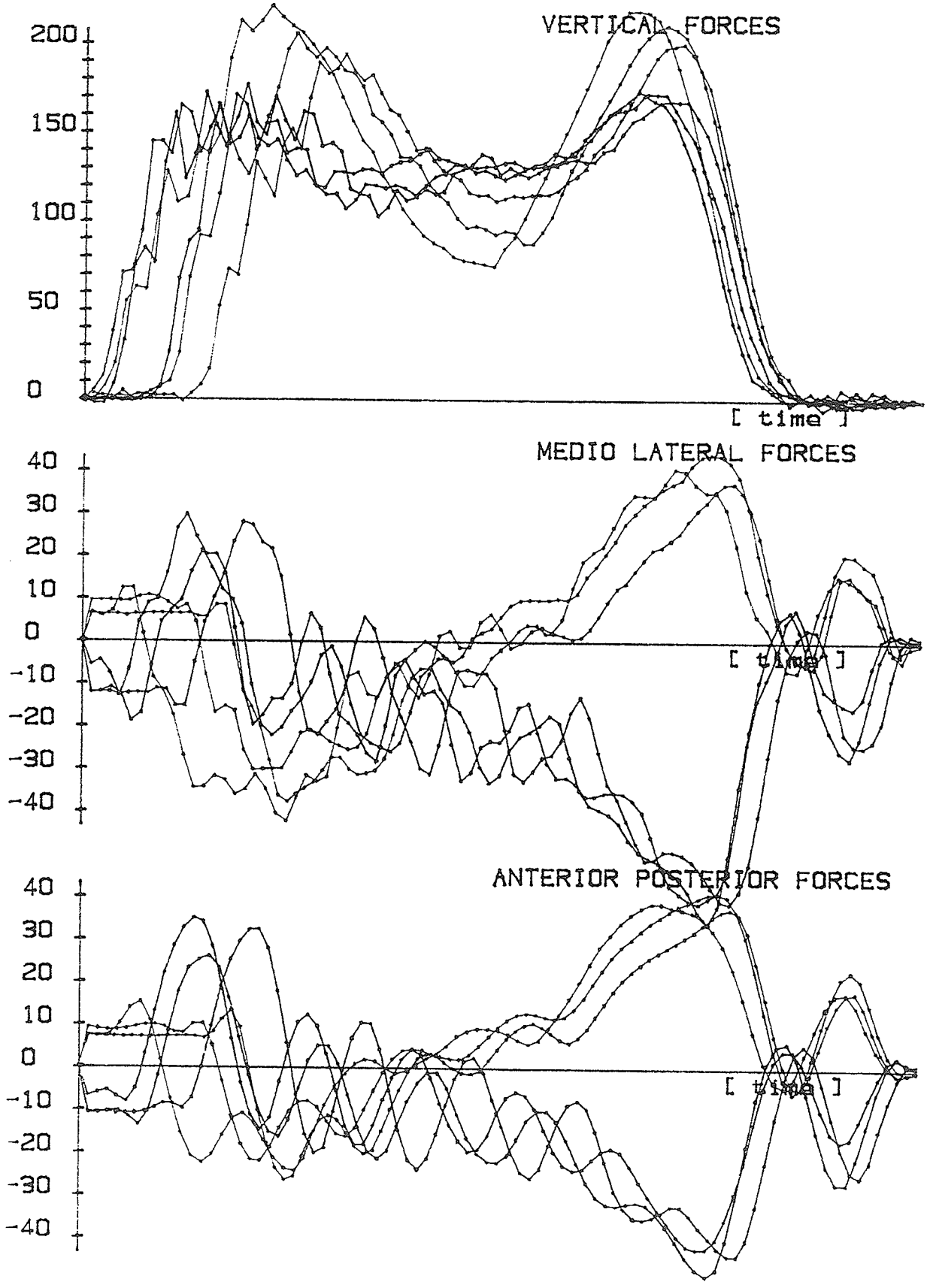

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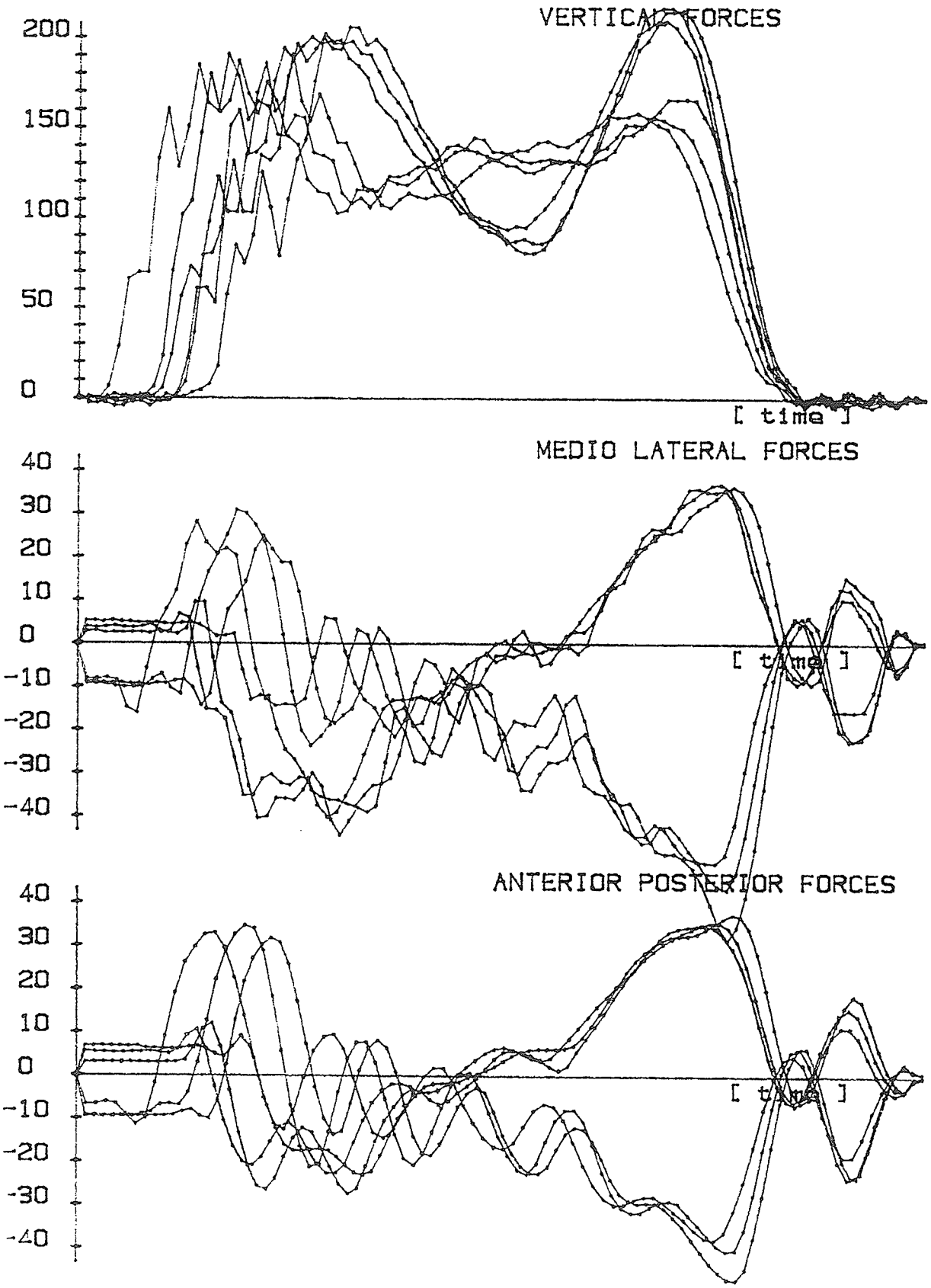

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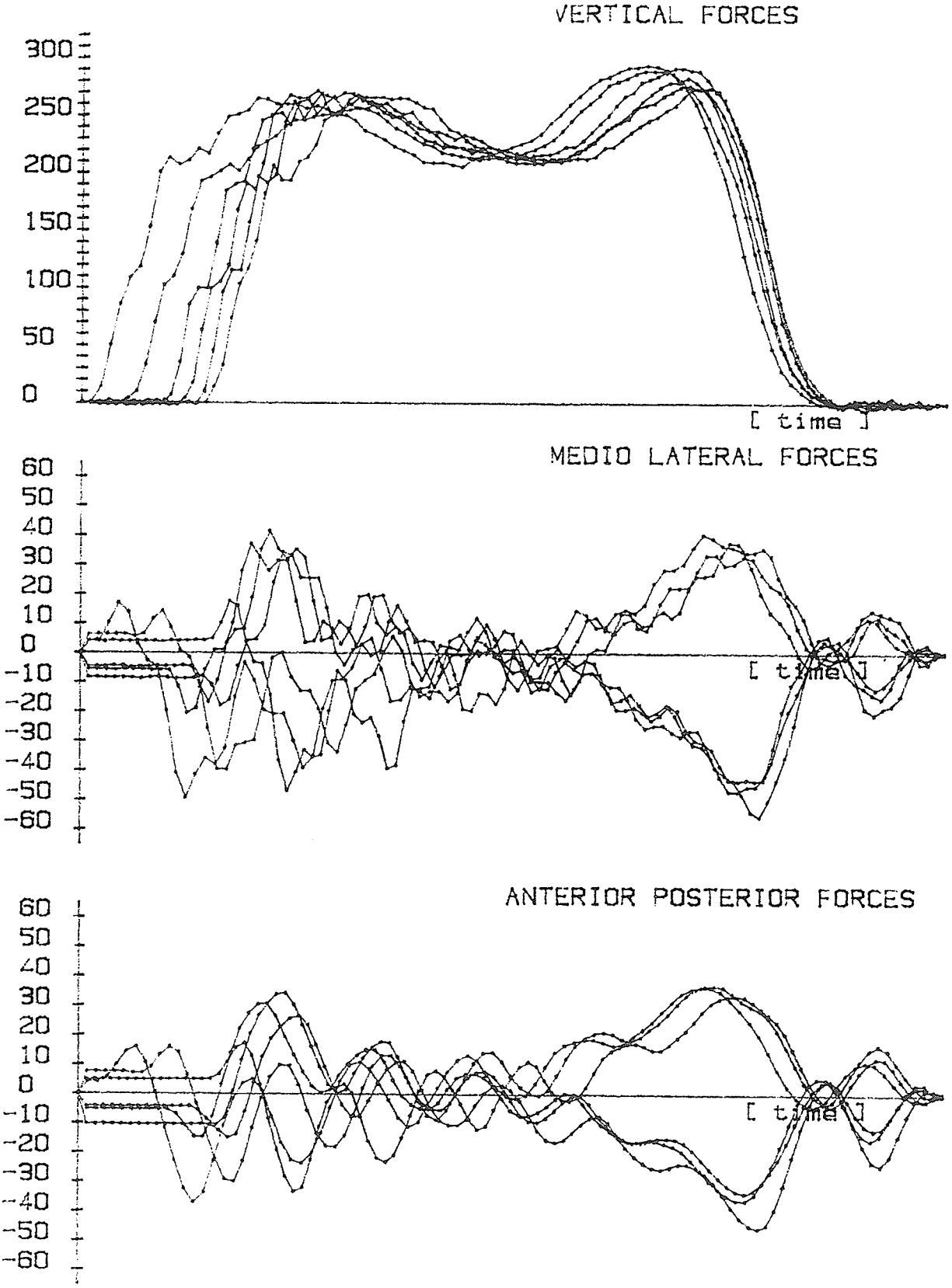

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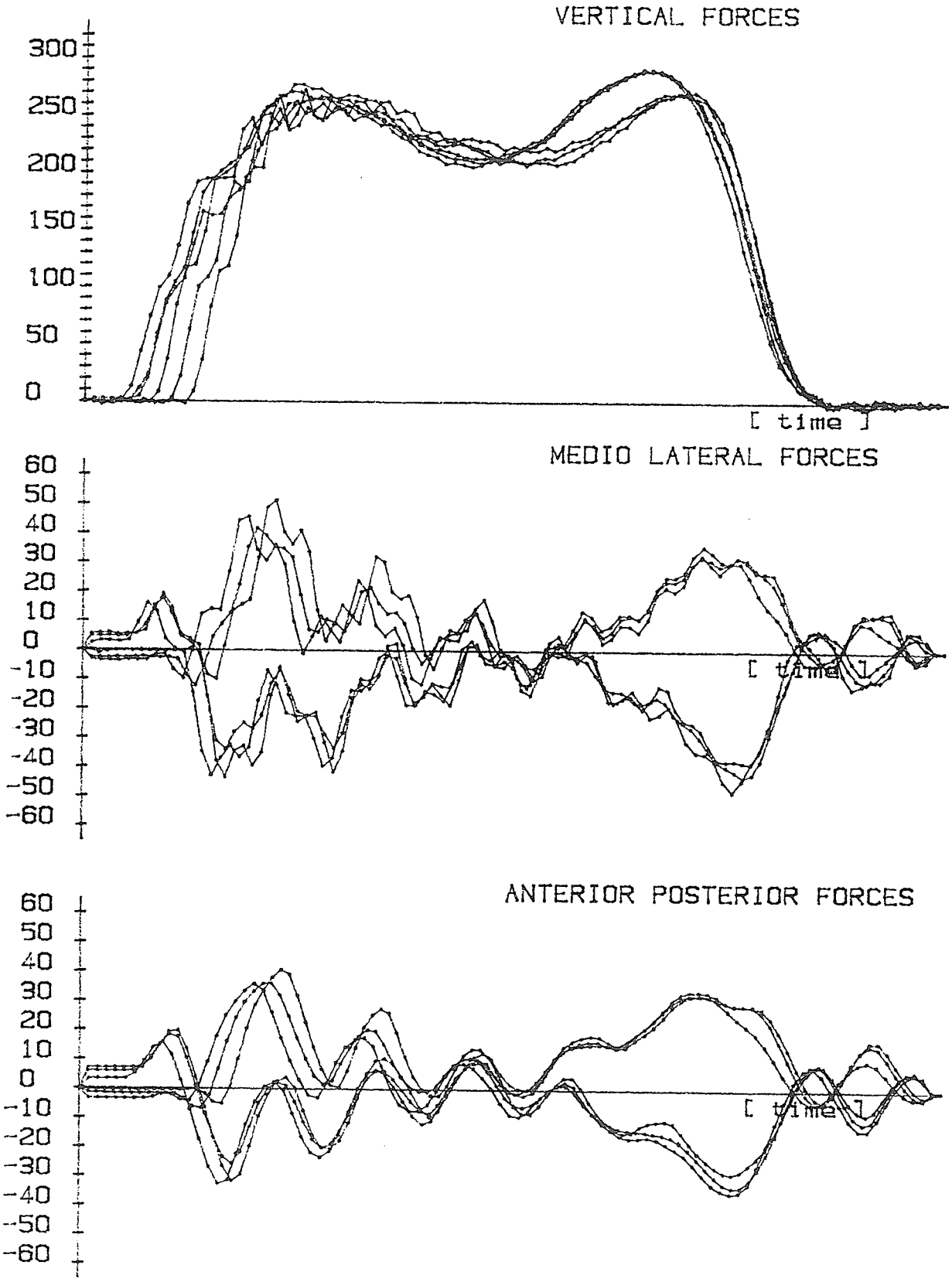

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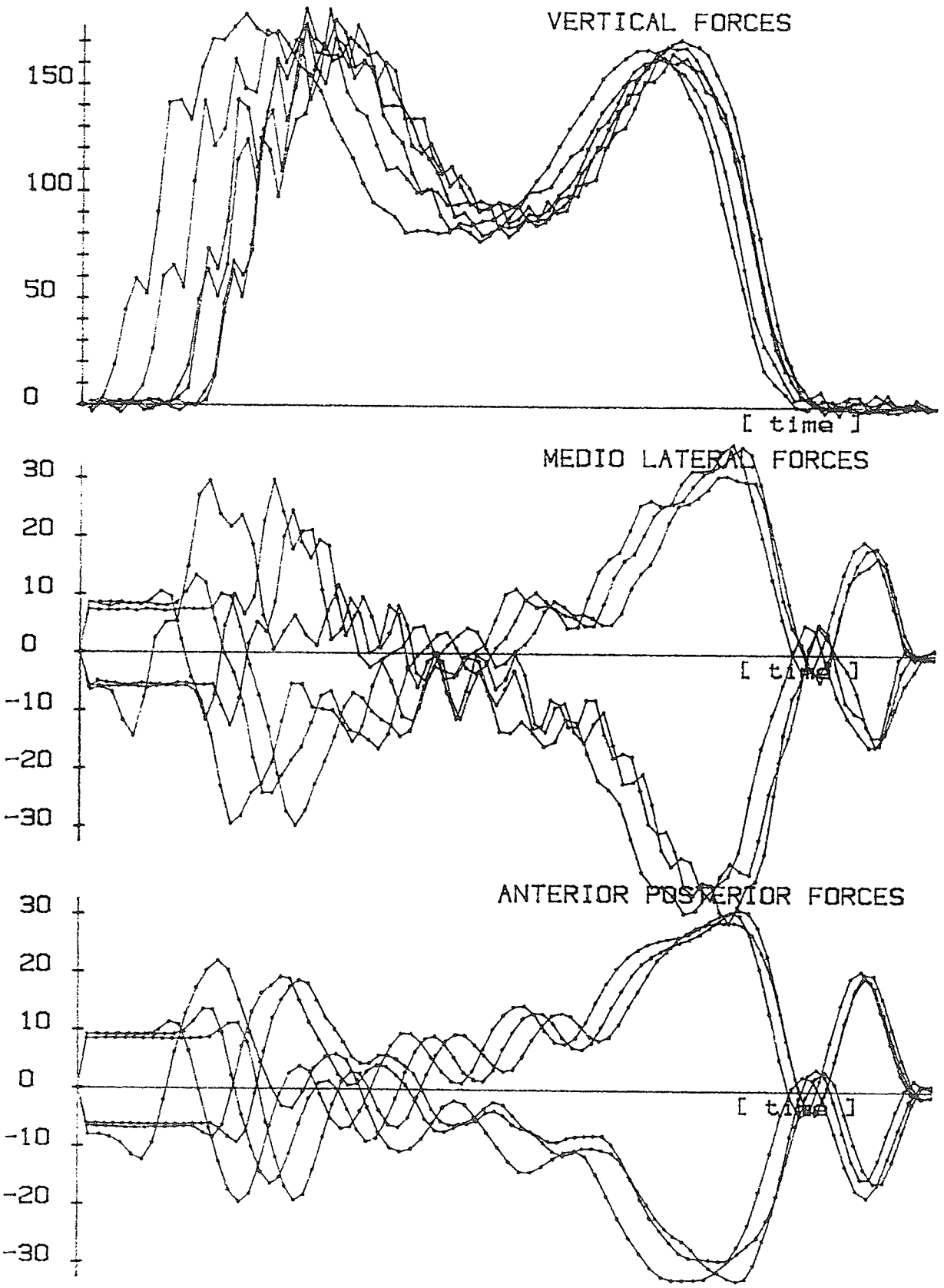

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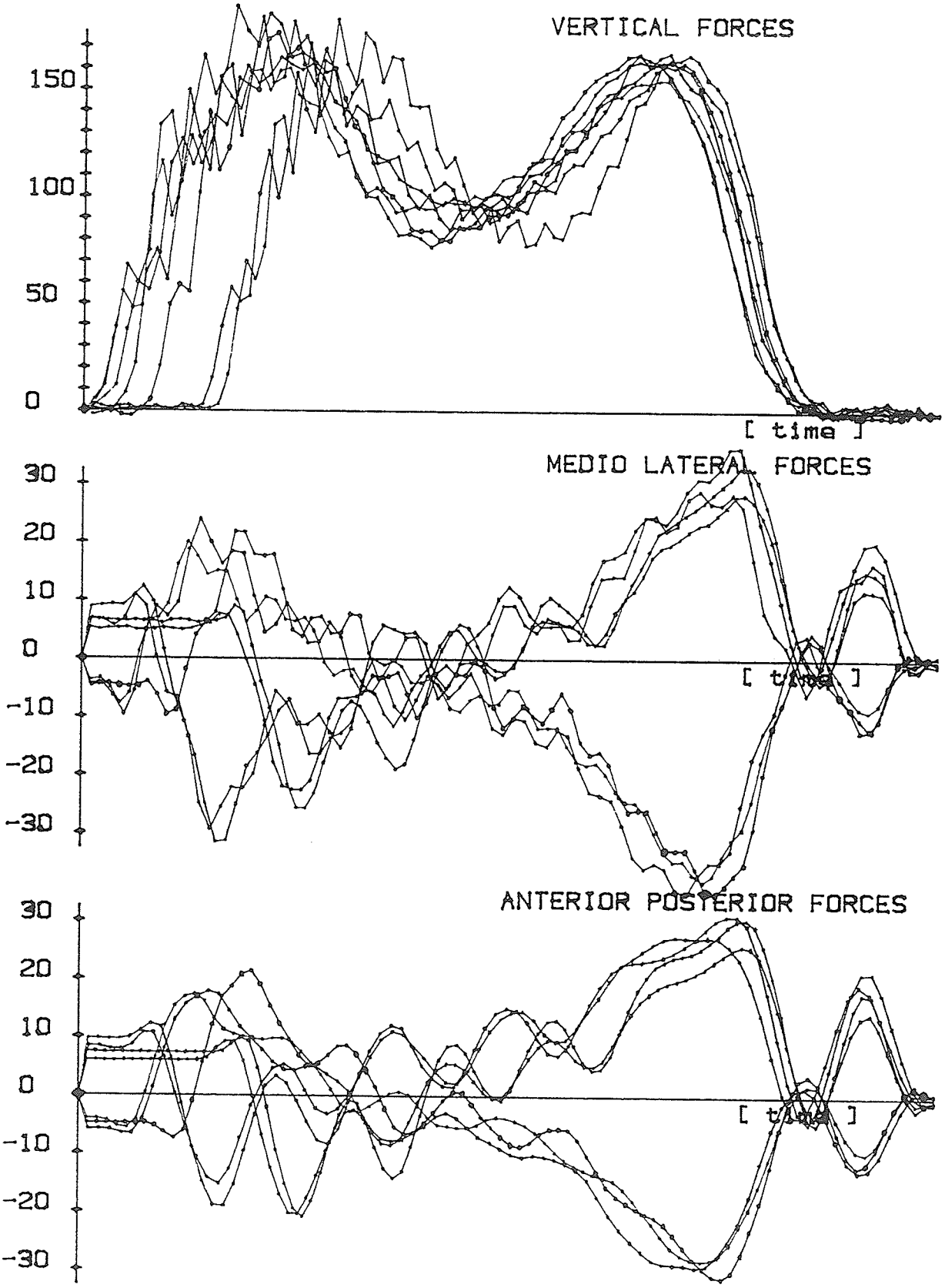

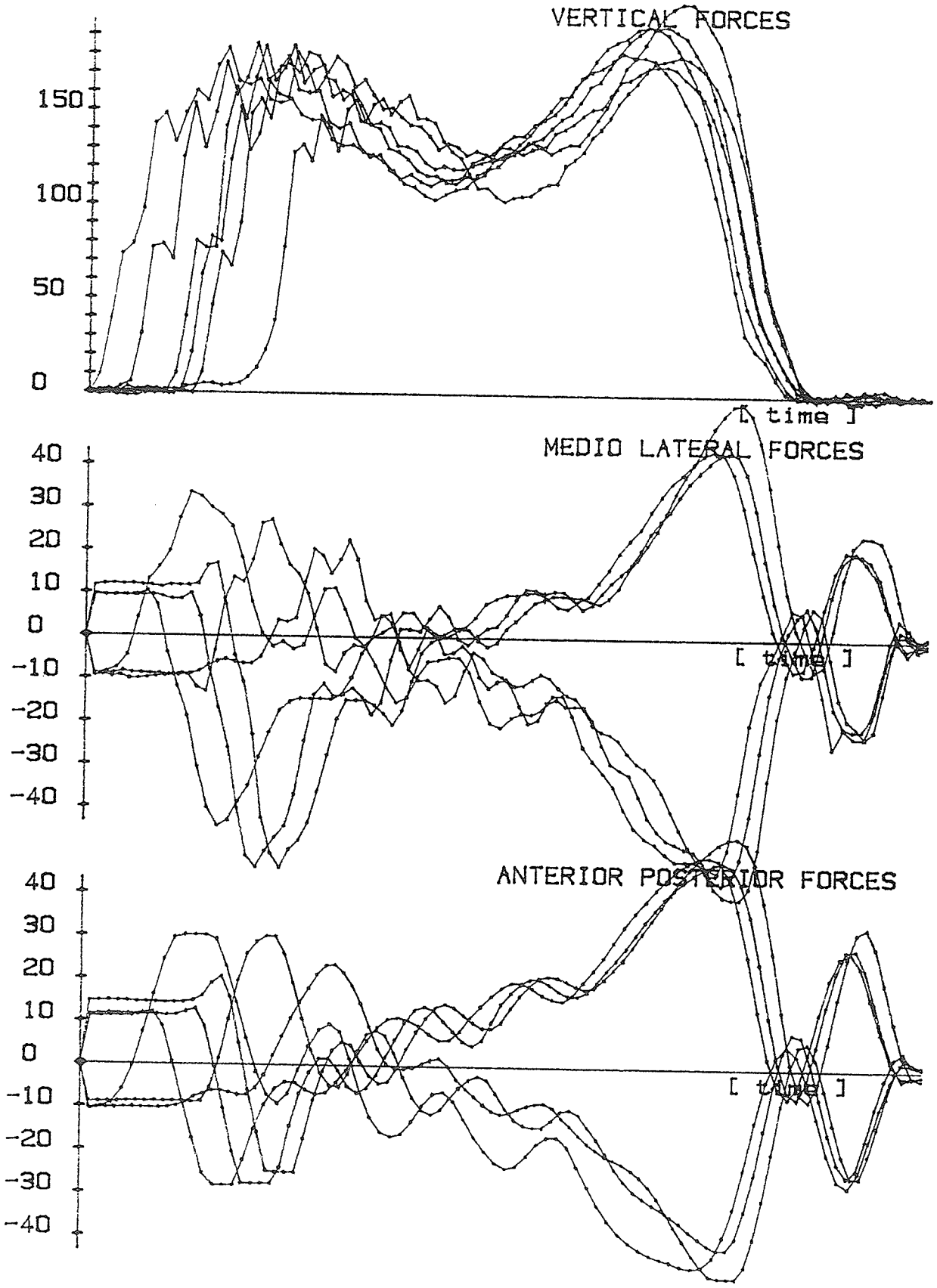
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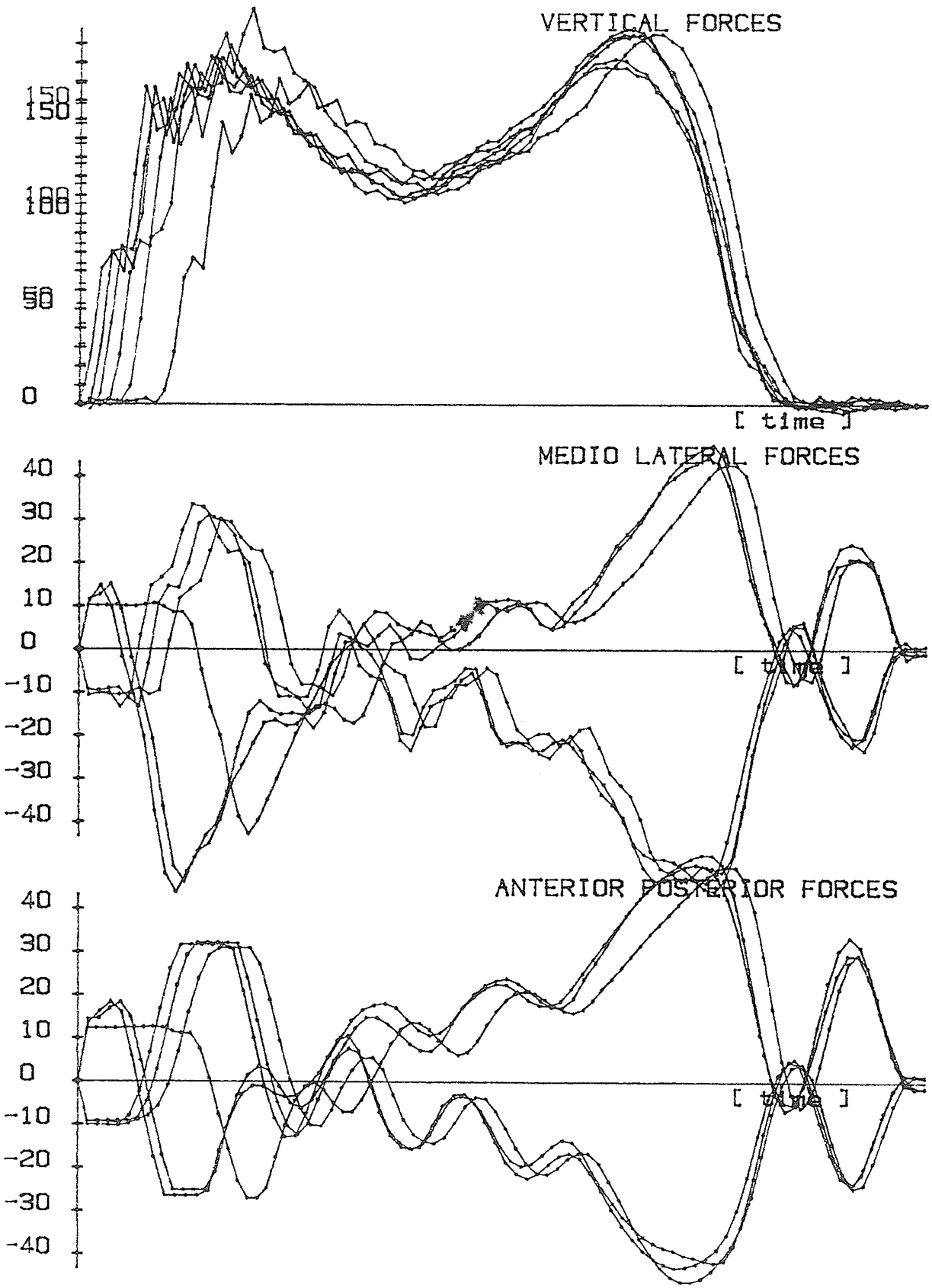

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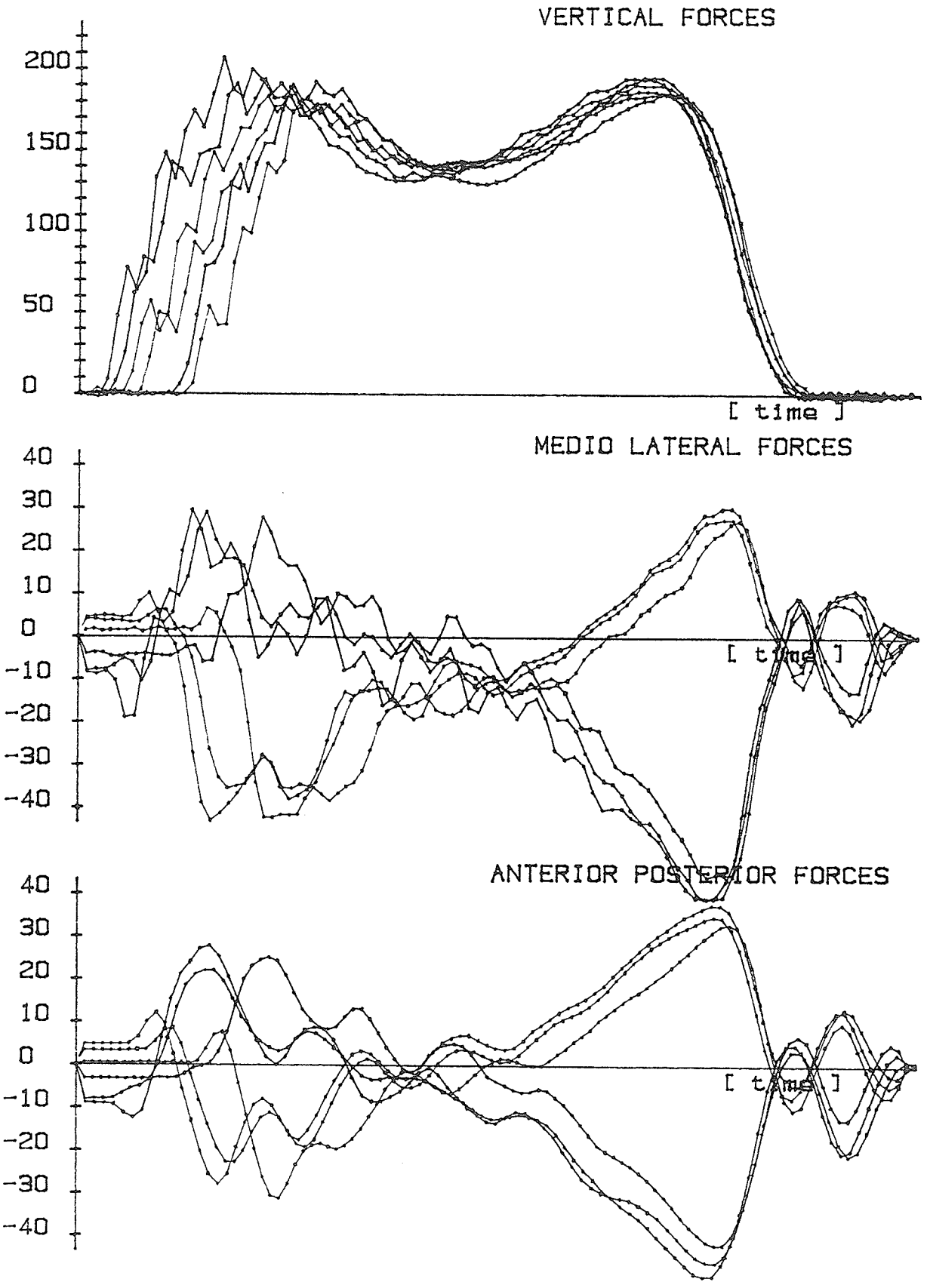

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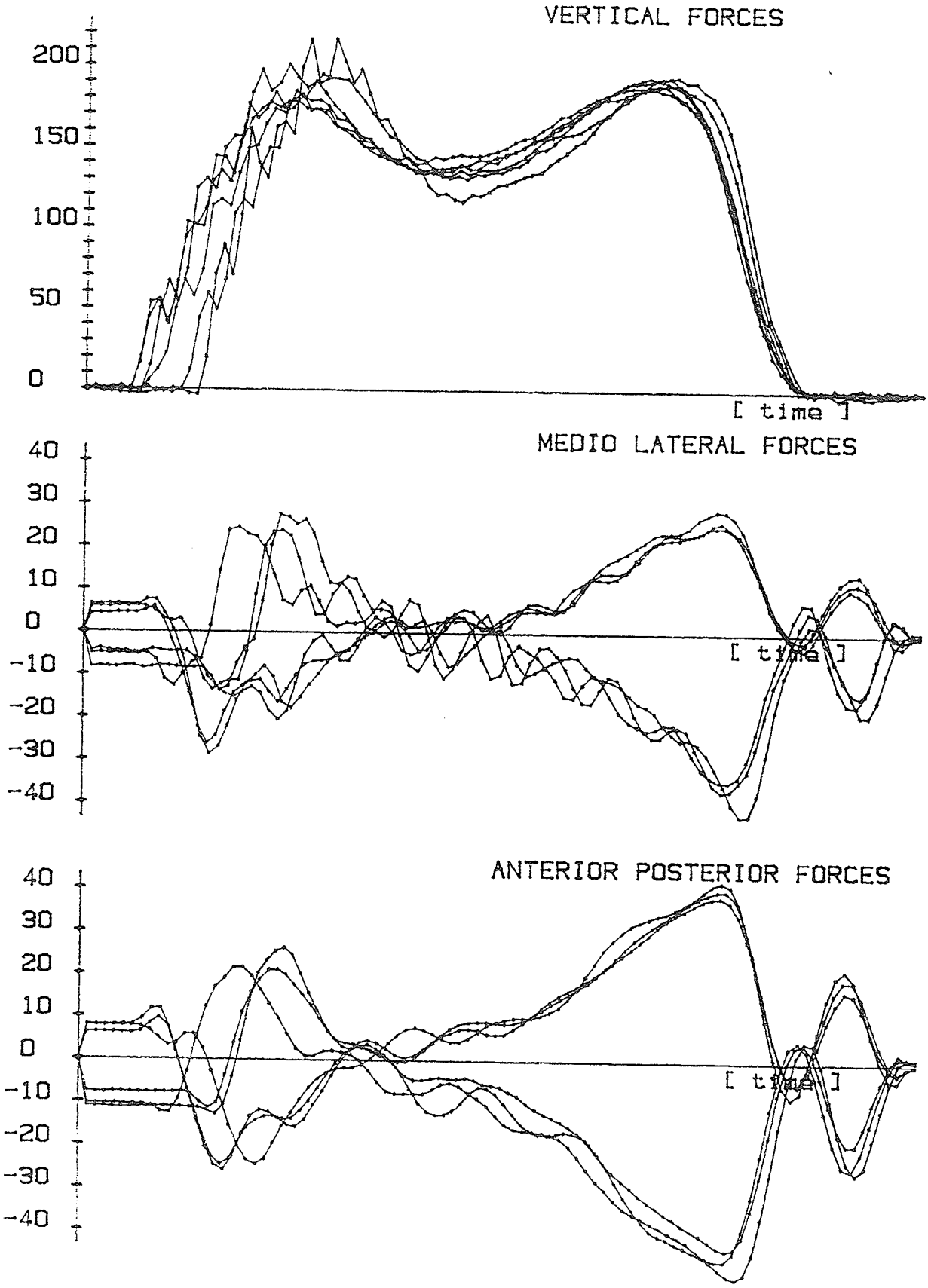

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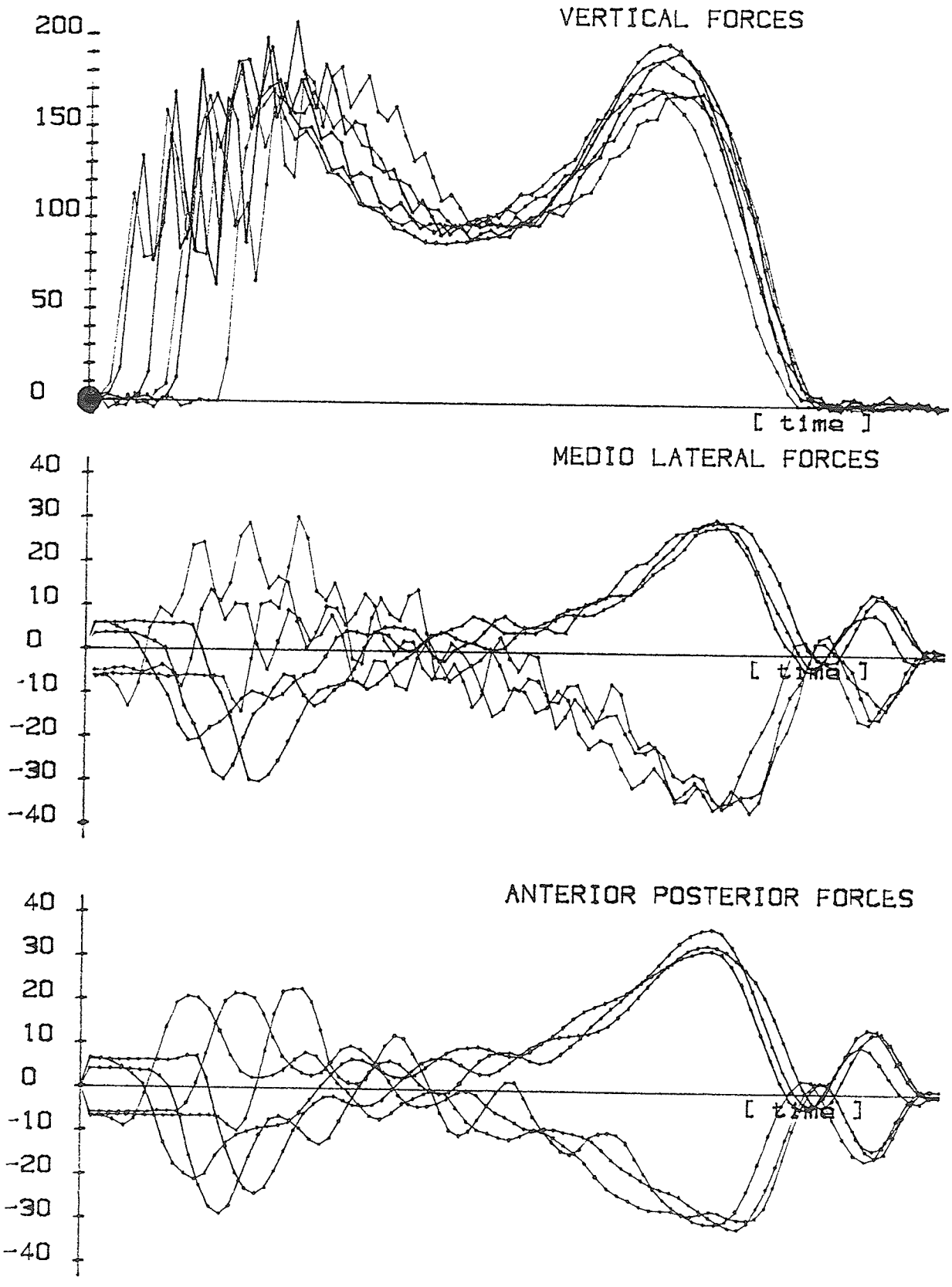

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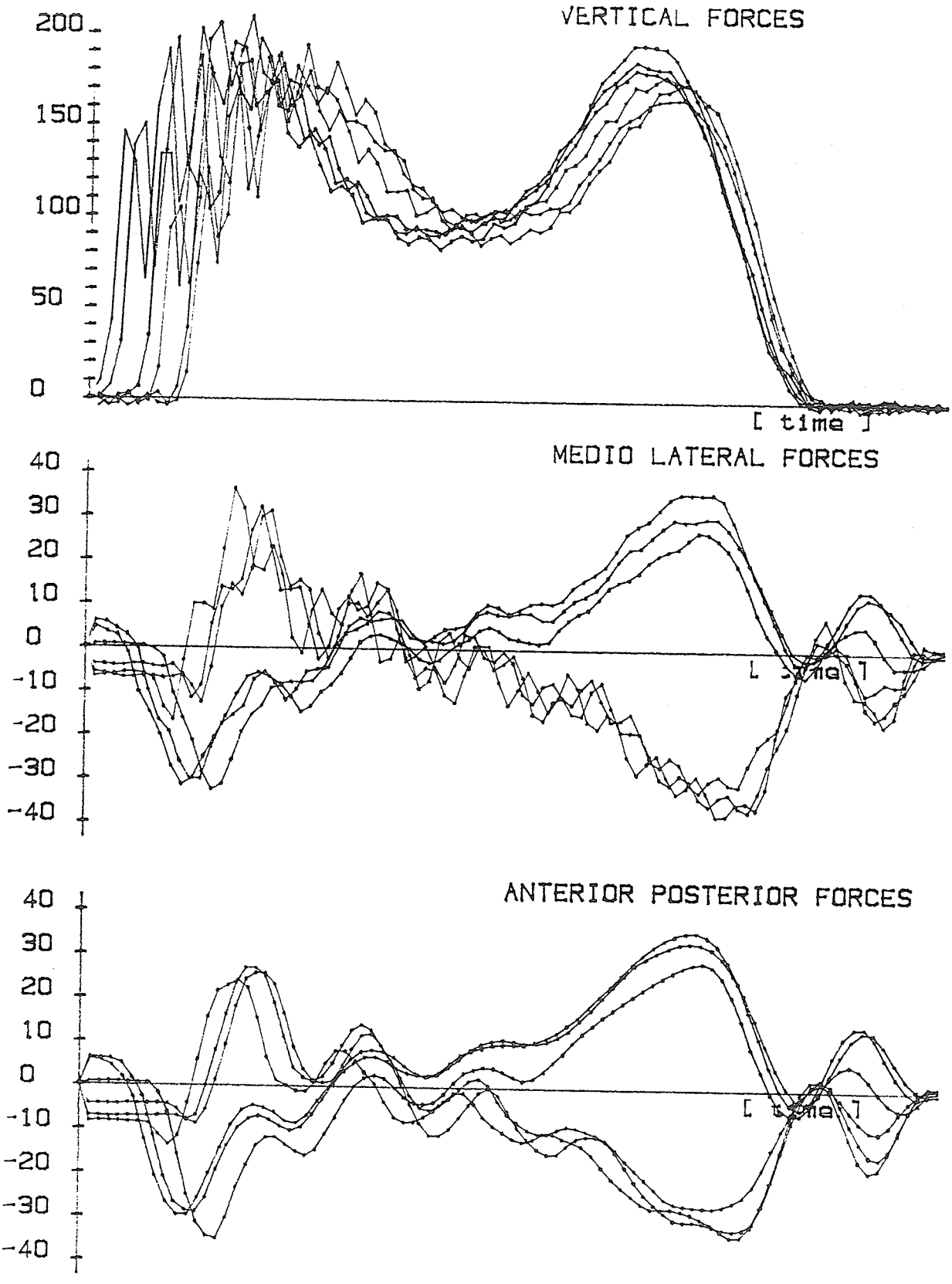

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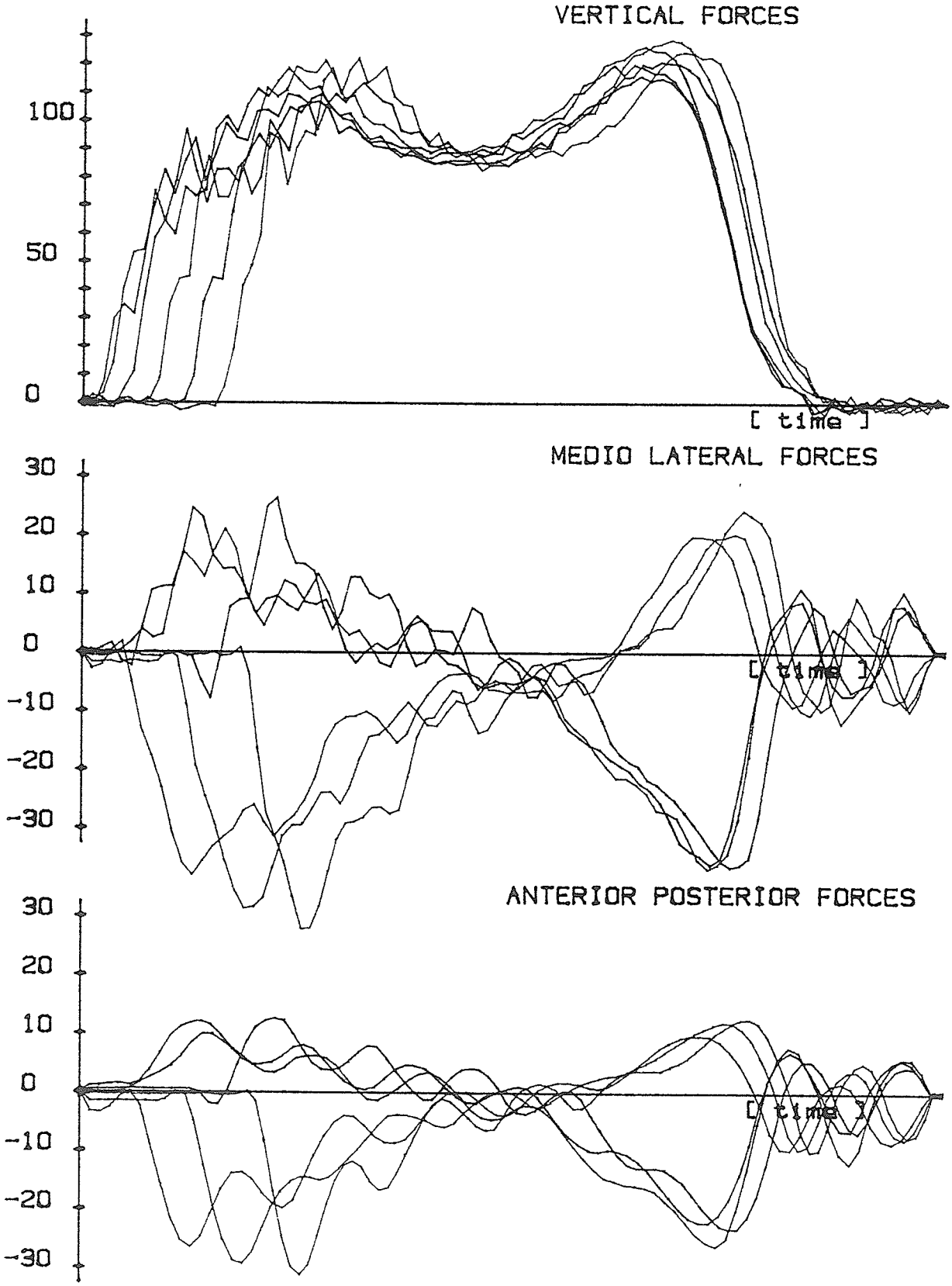

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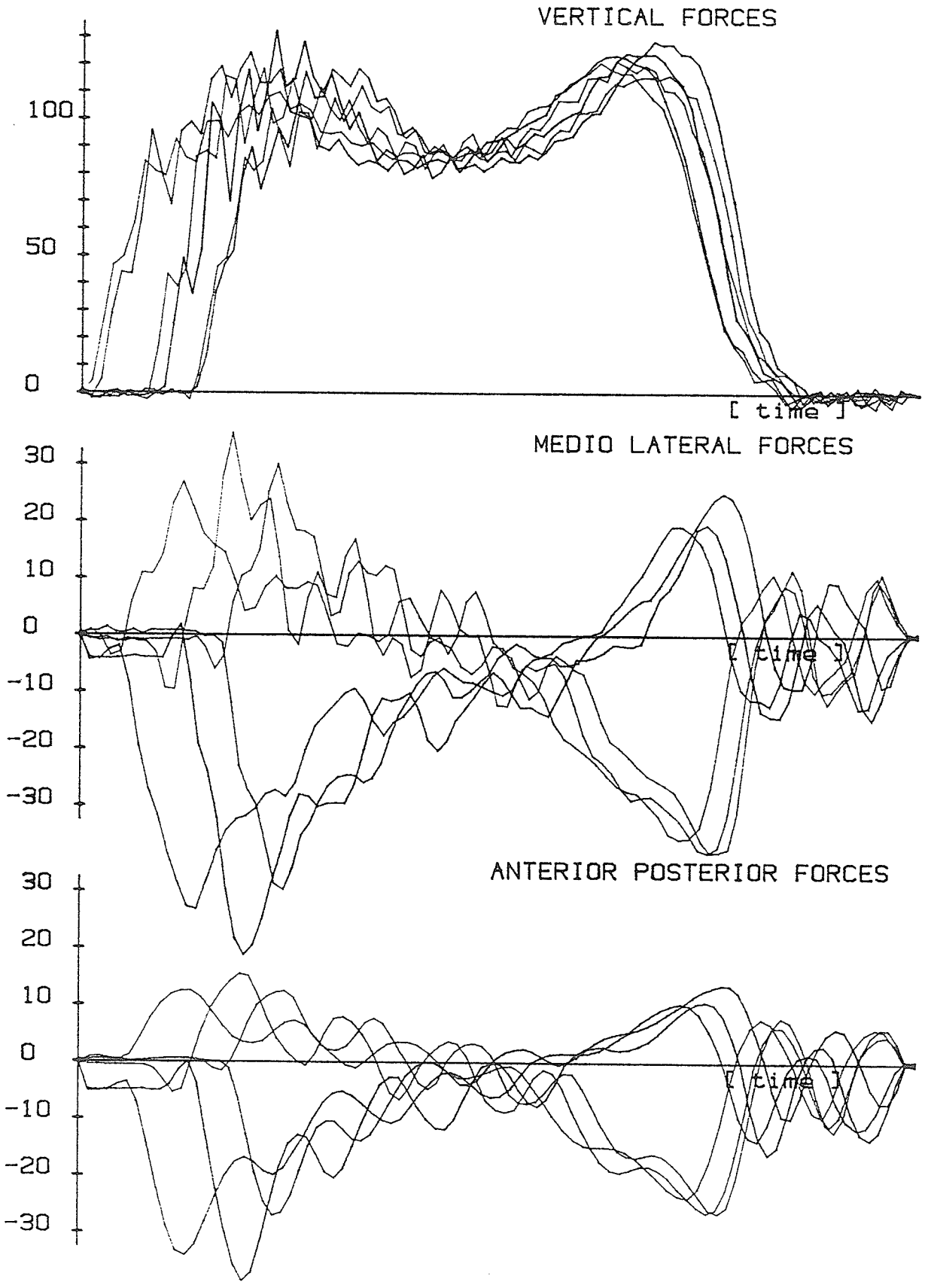

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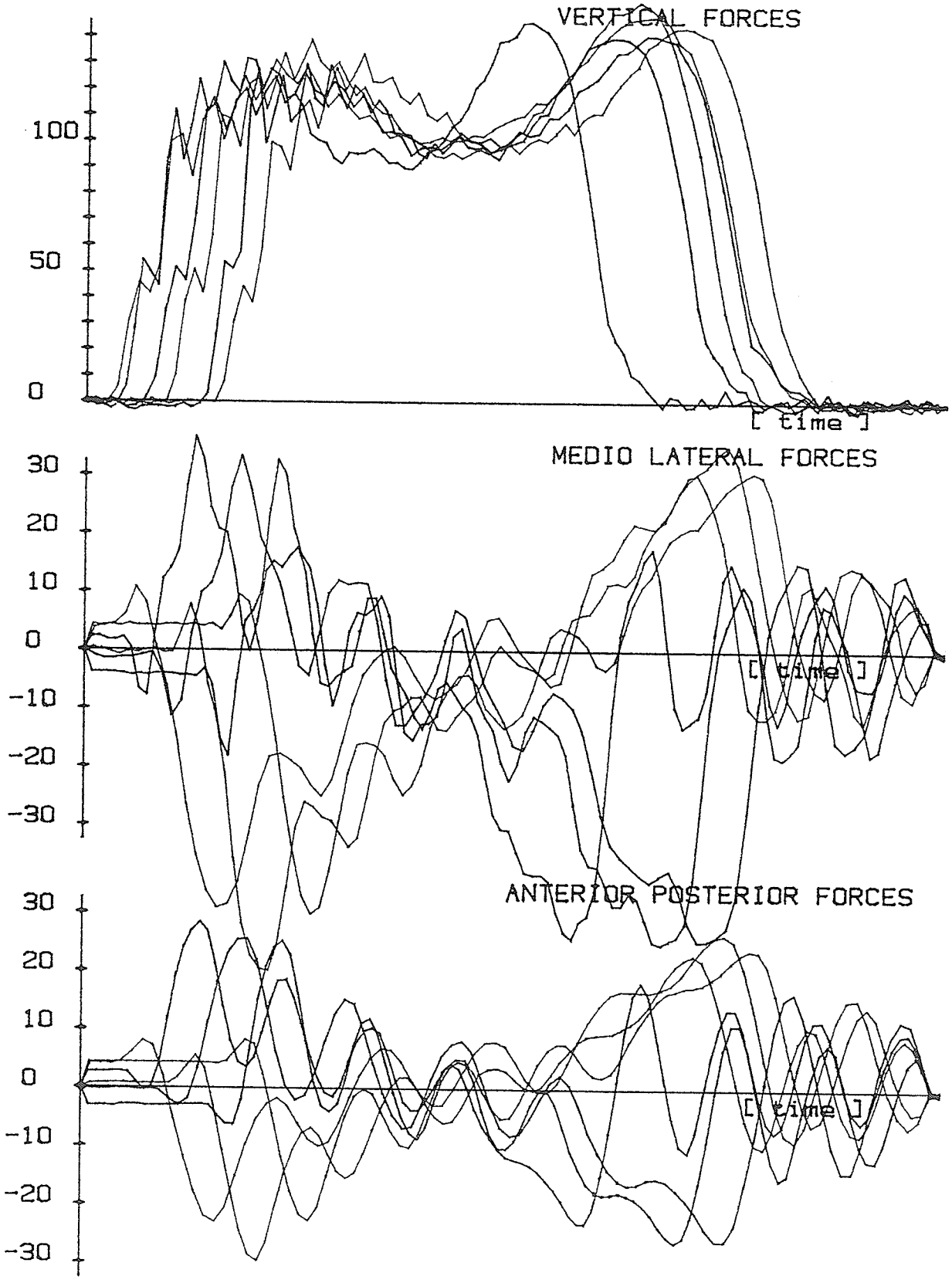

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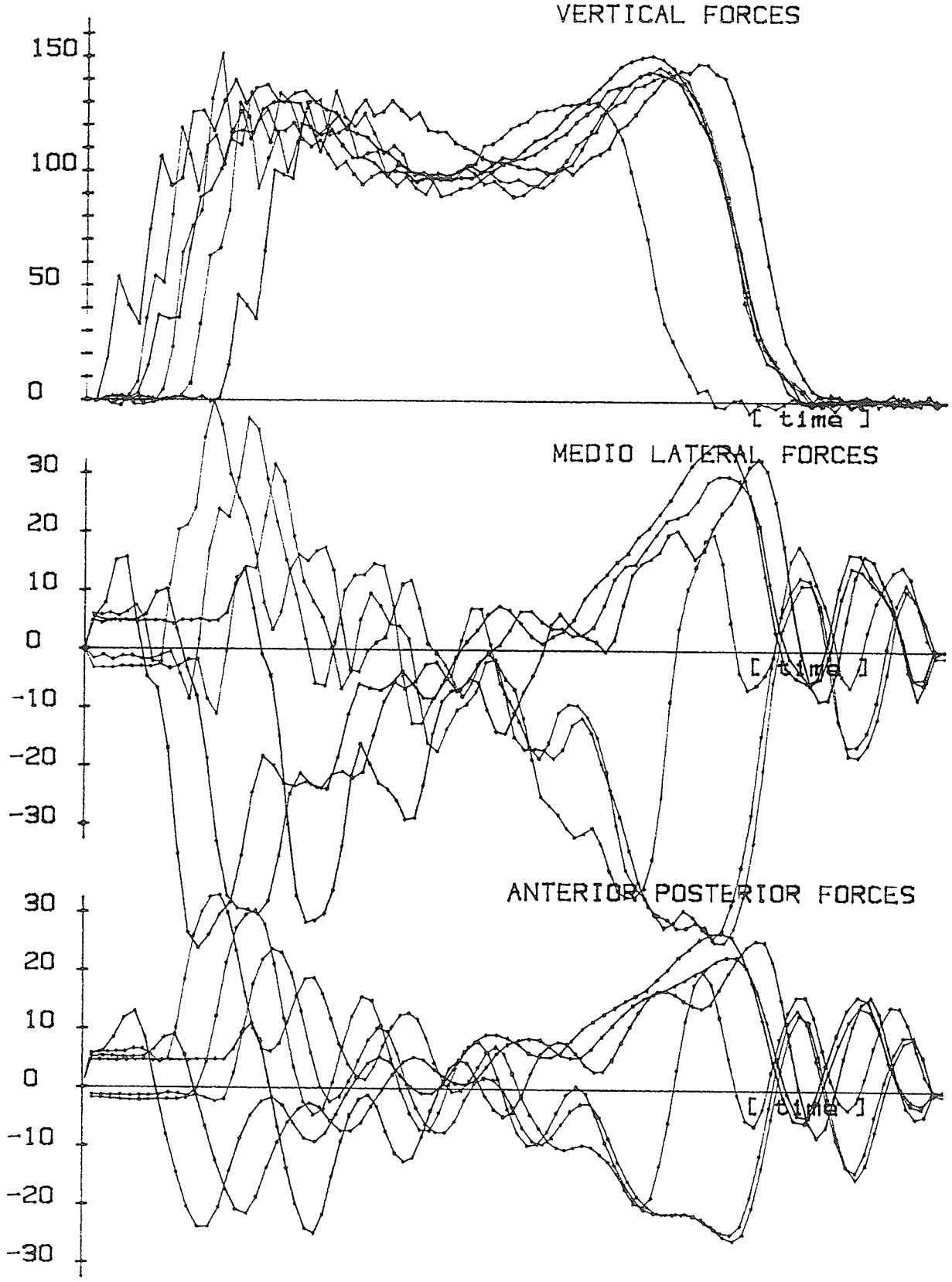

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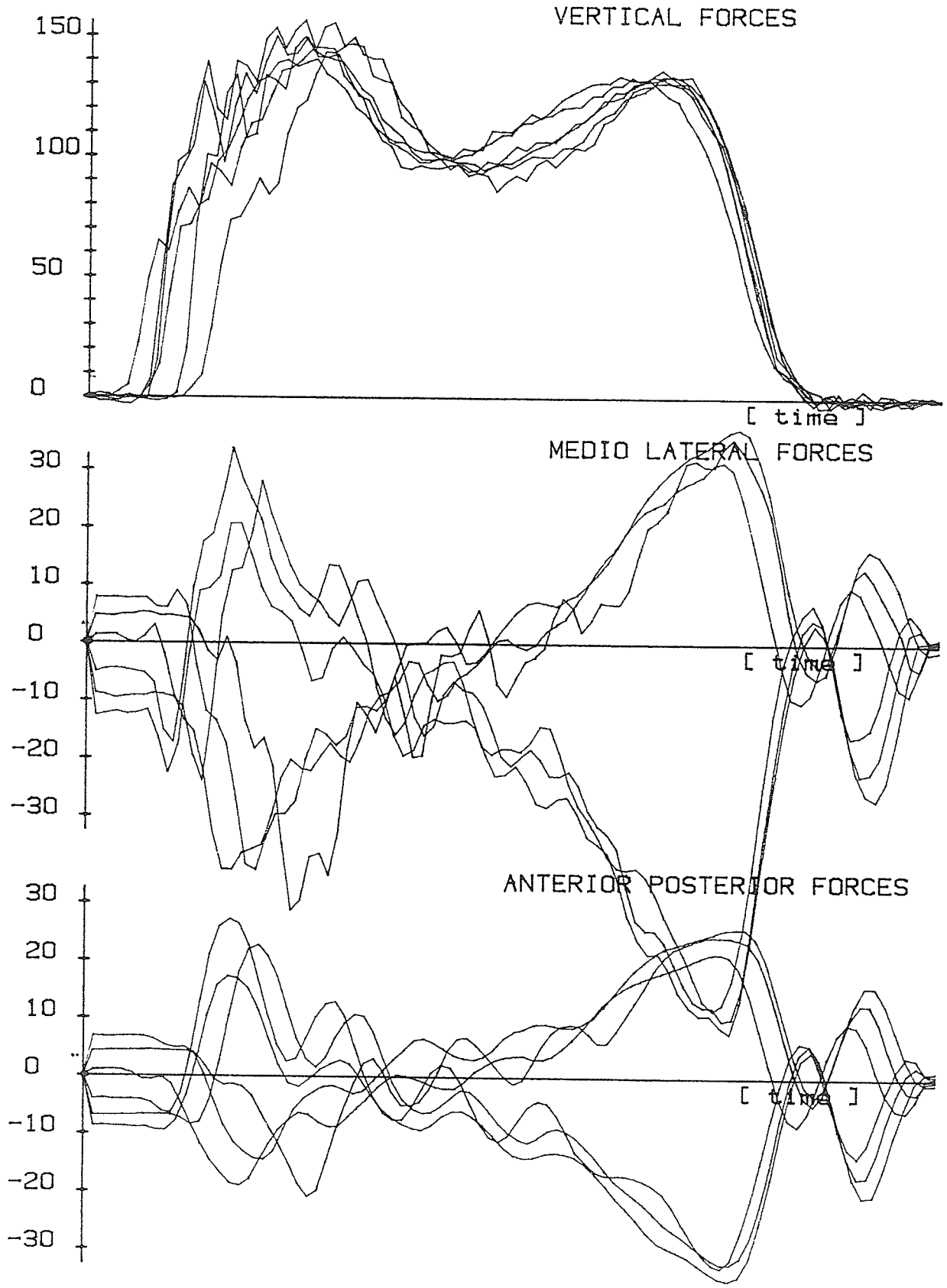

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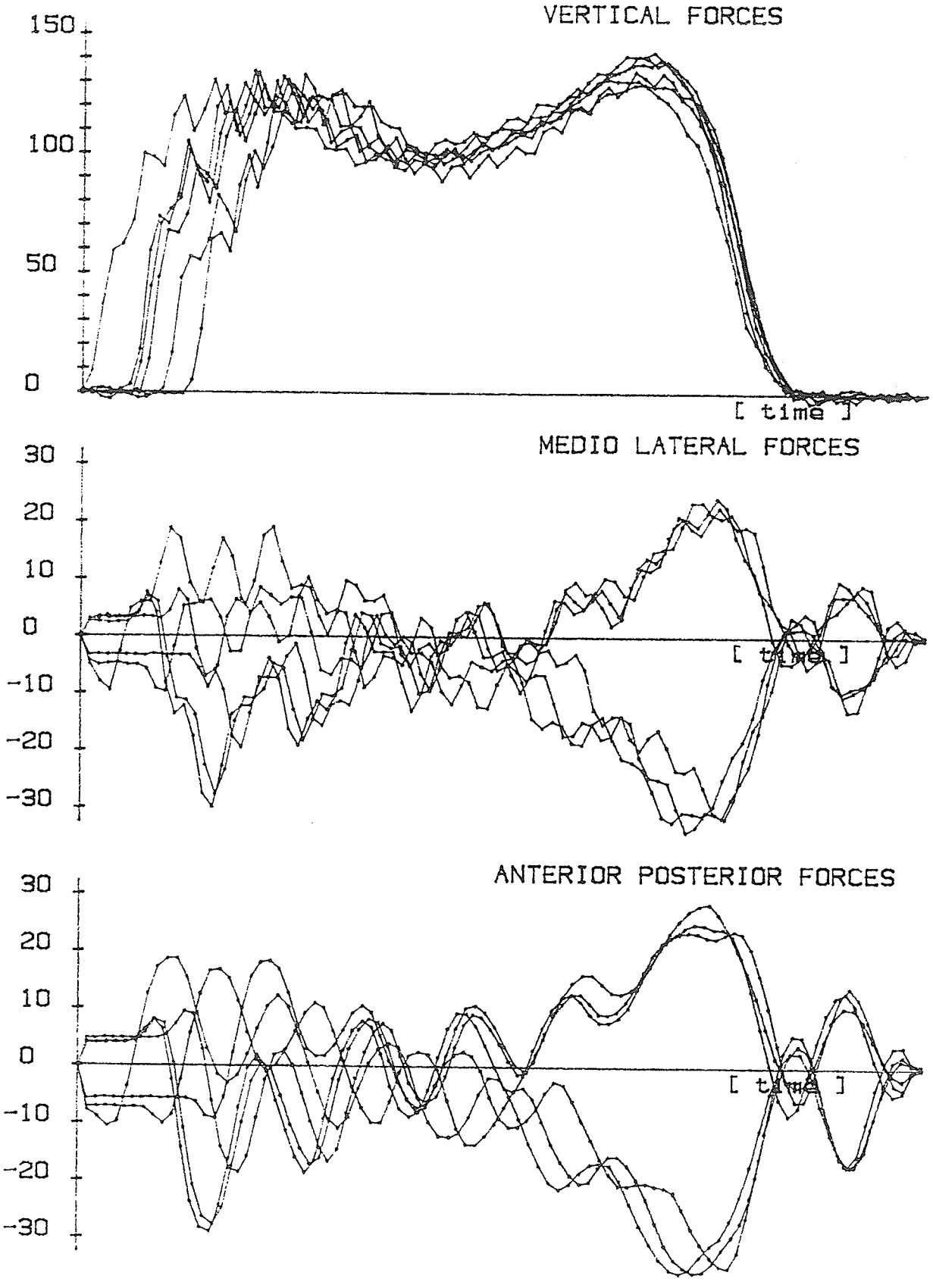

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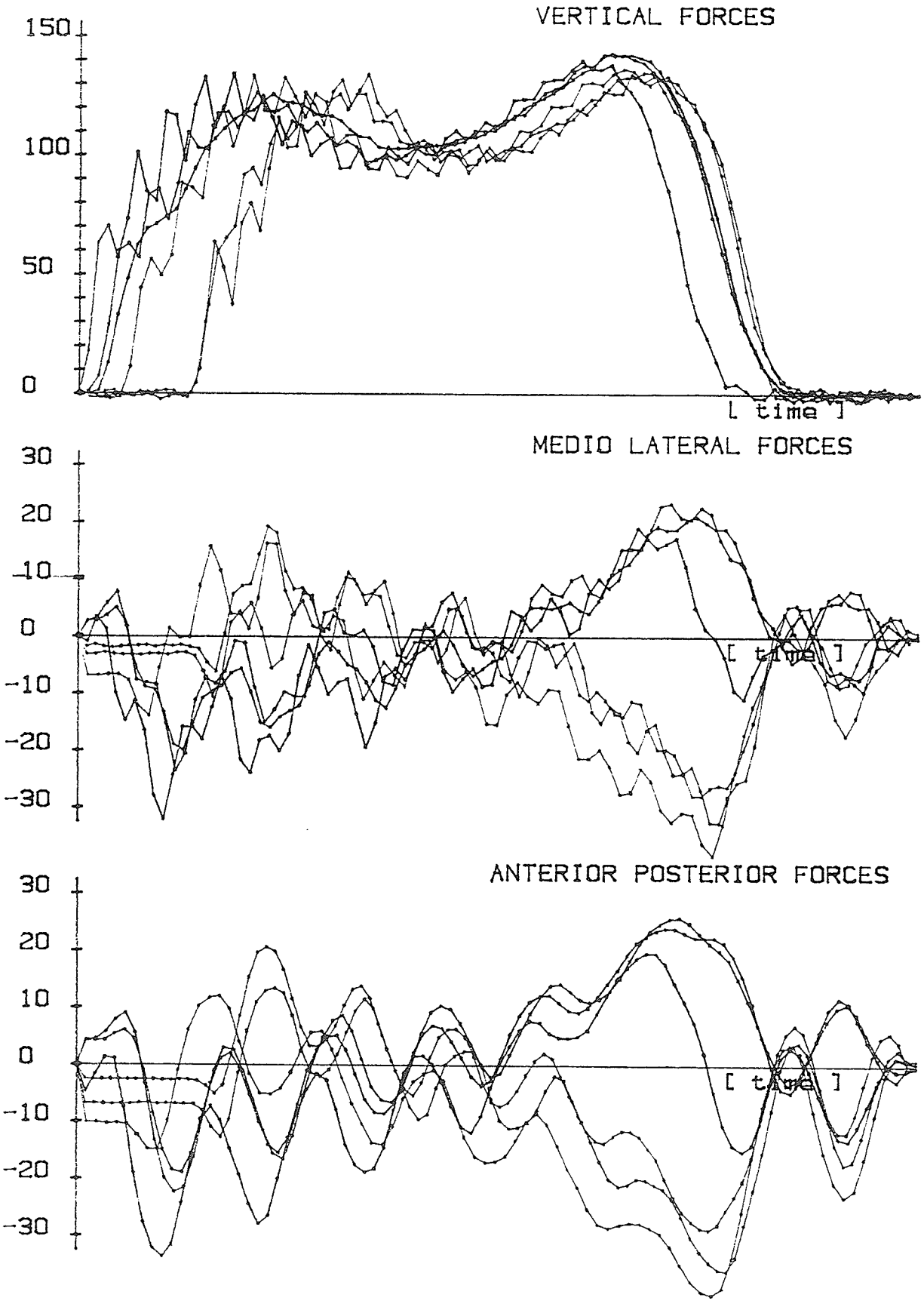

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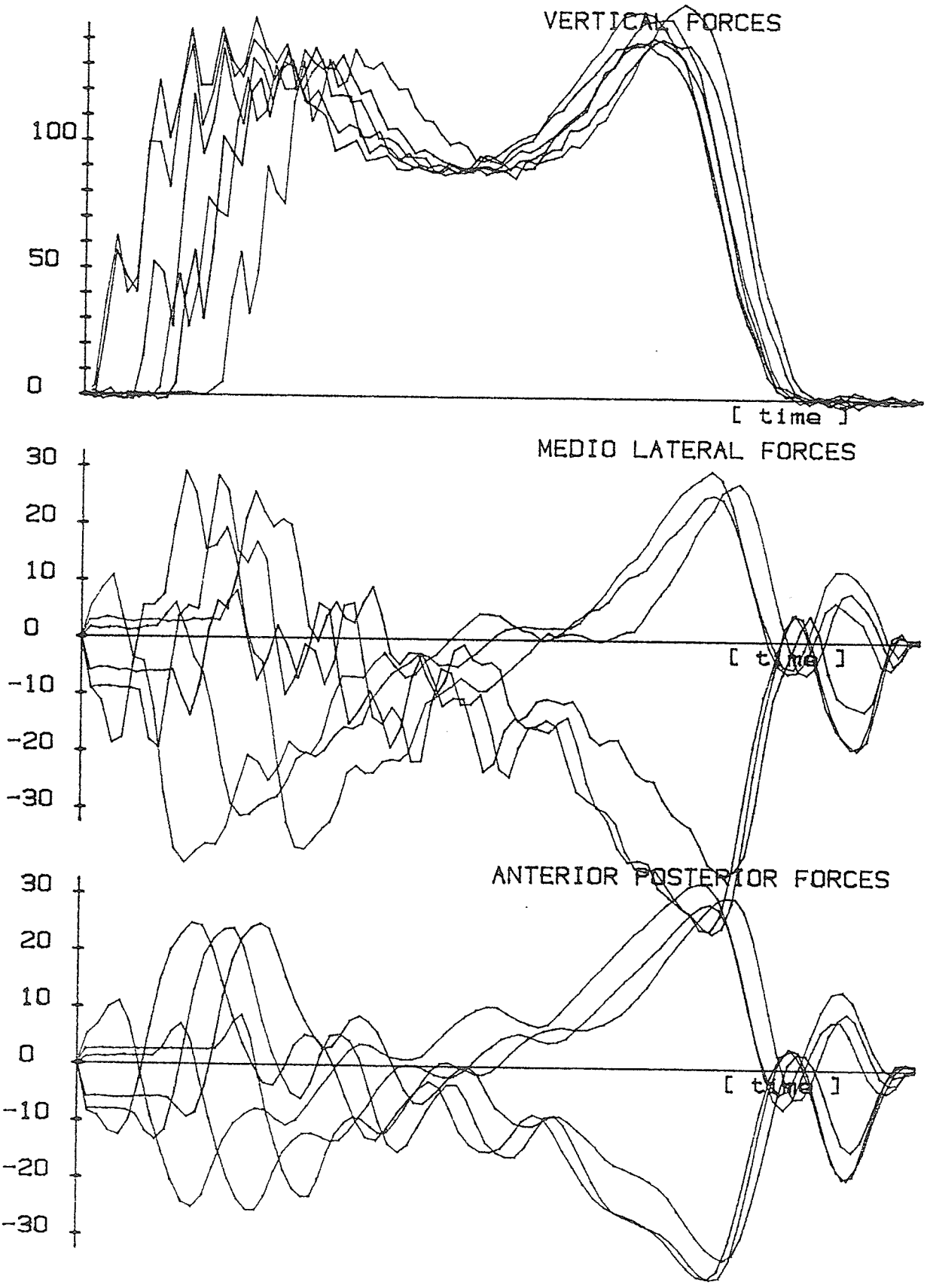

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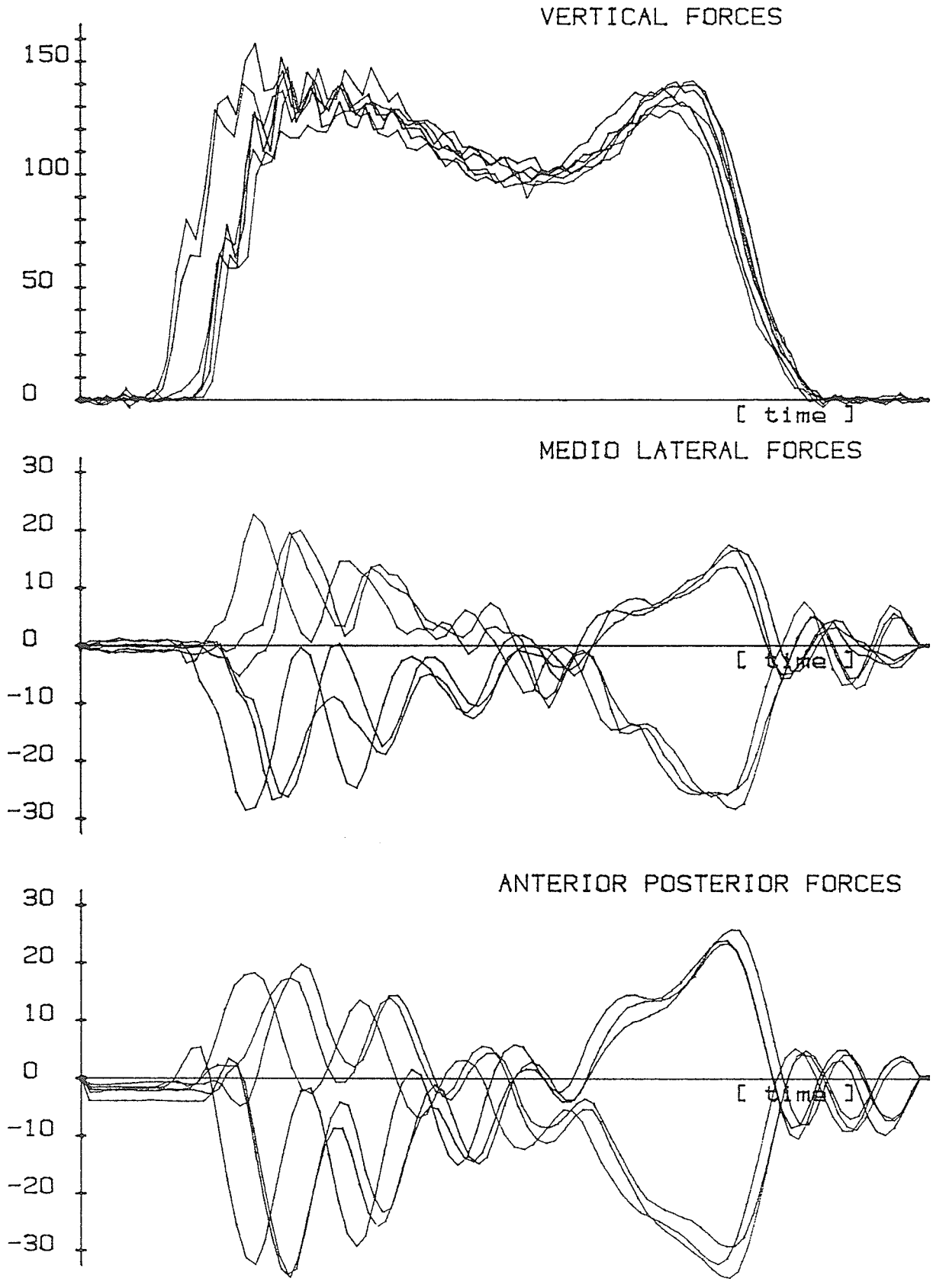

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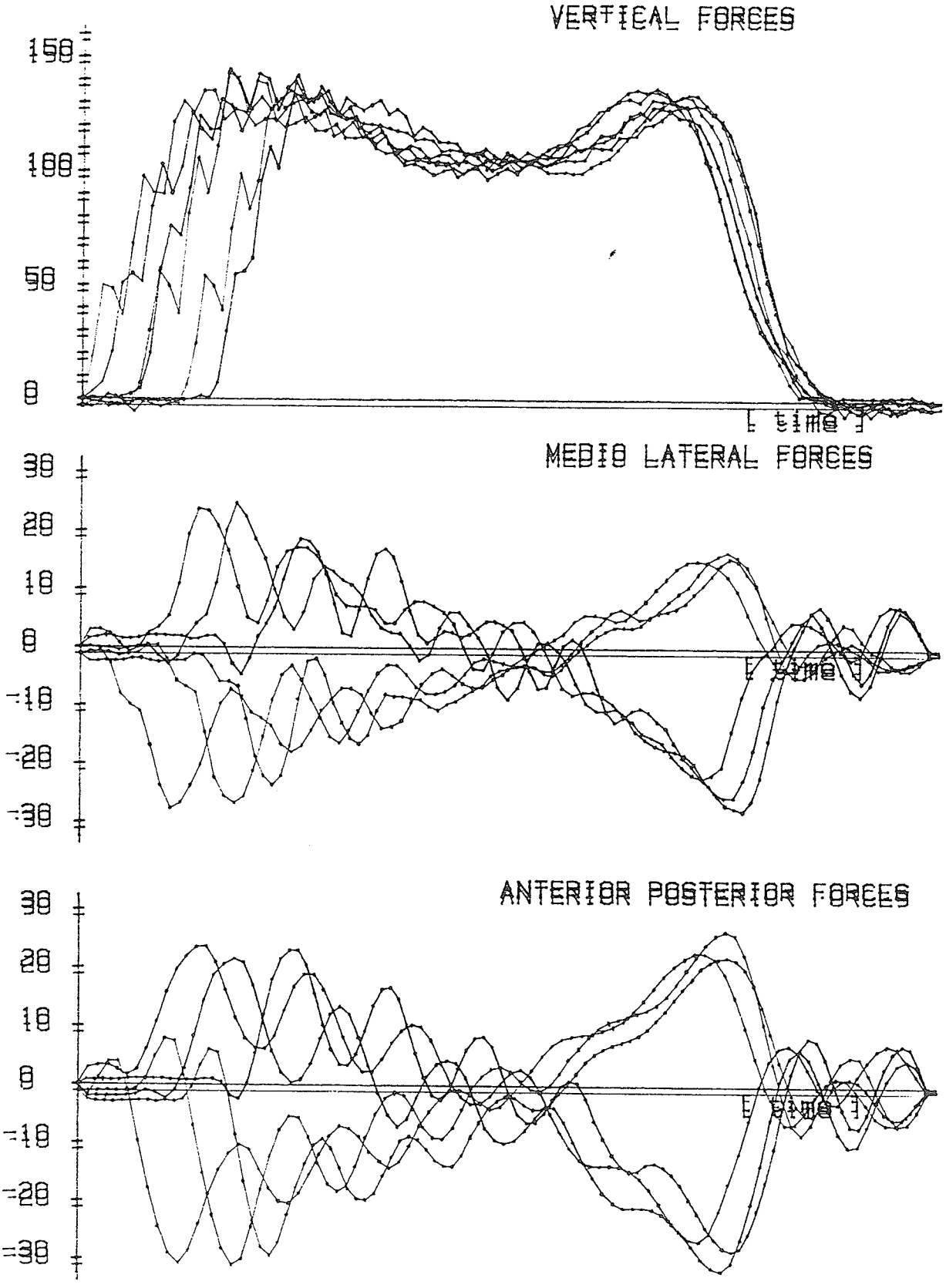

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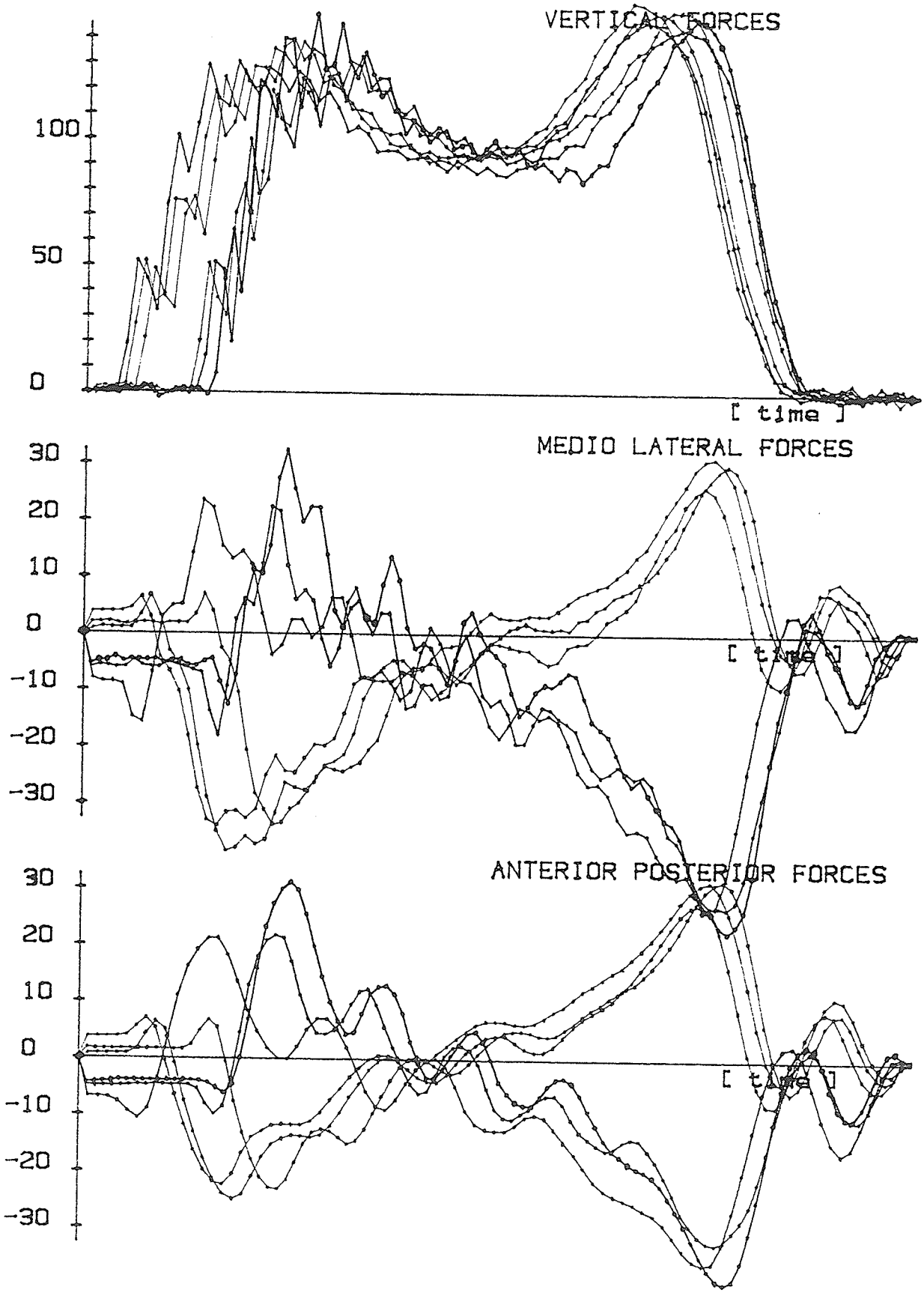

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