ASSESSMENT OF MOVEMENT IN THE ELDERLY DURING SLEEP WITH REFERENCE TO PRESSURE SORE PREVENTION

BY

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ABSTRACT

This thesis describes the assessment of movement in the elderly with reference to pressure prevention.

Chapter 1 provides an introduction to the problem of pressure sores, and outlines the importance of age and immobility in their causation.

Chapter 2 contains a review of the literature pertaining to the aetiology and epidemiology of pressure sores. A study conducted by the author at McGill University, into the pathophysiology of pressure sores - using the pig as an experimental model, is described.

Chapter 3 discusses the previous published work on movement during sleep. Typical patterns of movement during sleep, and principles and techniques used to monitor overnight mobility are described.

Chapter 4 describes an investigation into the relationship between body - support surface interface pressure and overnight mobility. Previously established monitoring systems were utilised. A high degree of correlation between the two variables was established.

Chapter 5 deals with the design and development of a mobility monitoring system for routine use in the clinical environment. The system consists of three elements, force transducers, processing instrumentation - the output of which is representative of the size of move by the patient, and an F.M. analogue tape recorder - used to transfer the mobility data between the hospital and the laboratory.

Chapter 6 contains a review of present methods of assessment of patients with reference to their risk of developing sores, selection of elderly subjects, an outline of the experimental protocol utilised and techniques of analysing the data.

Chapter 7 contains the results of the experimental investigation into overnight mobility of elderly patients in hospital, with associated discussion. These results indicate the feasibility of developing simple, reliable parameters to characterise body movements during sleep in a clinical environment and that there are clinical factors associated with consistently low mobility.

Chapter 8 provides a summary of this thesis.

Relevant appendices and bibliography are included at the end of the thesis.

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CONTENTS

CHAPTER 1

INTRODUCTION

When the weight of the body is supported, the forces required are transmitted through the soft tissues to the supporting surface. Weight bearing bony prominences of the skeleton, such as the sacrum, ischial tuberosities, trochanters, scapula, occiput, malleoli and heels, are regions where much of the body weight is concentrated when an individual is supported by a firm surface. The tissues over the bony prominences are compressed and blood flow to the soft tissue may be arrested. If the tissue is deprived of blood for a prolonged period, the tissue may die. Such lesions have been termed "pressure sores", "ischaemic ulcers", "decubitus ulcers" and "bedsores". Their appearance is characteristically a zone of damage extending from the deep tissues to the skin. Initially these "deep" pressure sores are observed as a persistent patch of red skin, which eventually breaks down to form a small opening in the skin's surface. With loss of the superficial layers, a cavity is revealed in the deeper tissues, which includes extensive damage to the muscle. Often these sores may involve damage to the bony prominence itself (periostitis).

Additionally, the term "pressure sore" has been attributed to superficial lesions which result from the effects of friction on tissue softened by moisture. Superficial sores are initially confined to the skin, although if they are untreated and become infected they can progress to the deeper tissues.

The aetiology of deep pressure sores, suggests that two primary factors are associated with their causation.

- compression of tissues, causing ischaemia
- reduced mobility, impairing the regular relief of ischaemia.

The clinical condition of the patient and the environment in which he is nursed, can influence a range of parameters which may directly or indirectly affect these two primary factors (figure 1.1). Patients who are debilitated or who are nursed on support surfaces with inadequate pressure relieving properties, are subject to significant compression of the tissues over their bony prominences. Patients who are unconscious or suffering from neurological deficit may be unable to move spontaneously and relieve compression in vulnerable tissue, due to a loss of motor function. Additionally, as a result of neurological

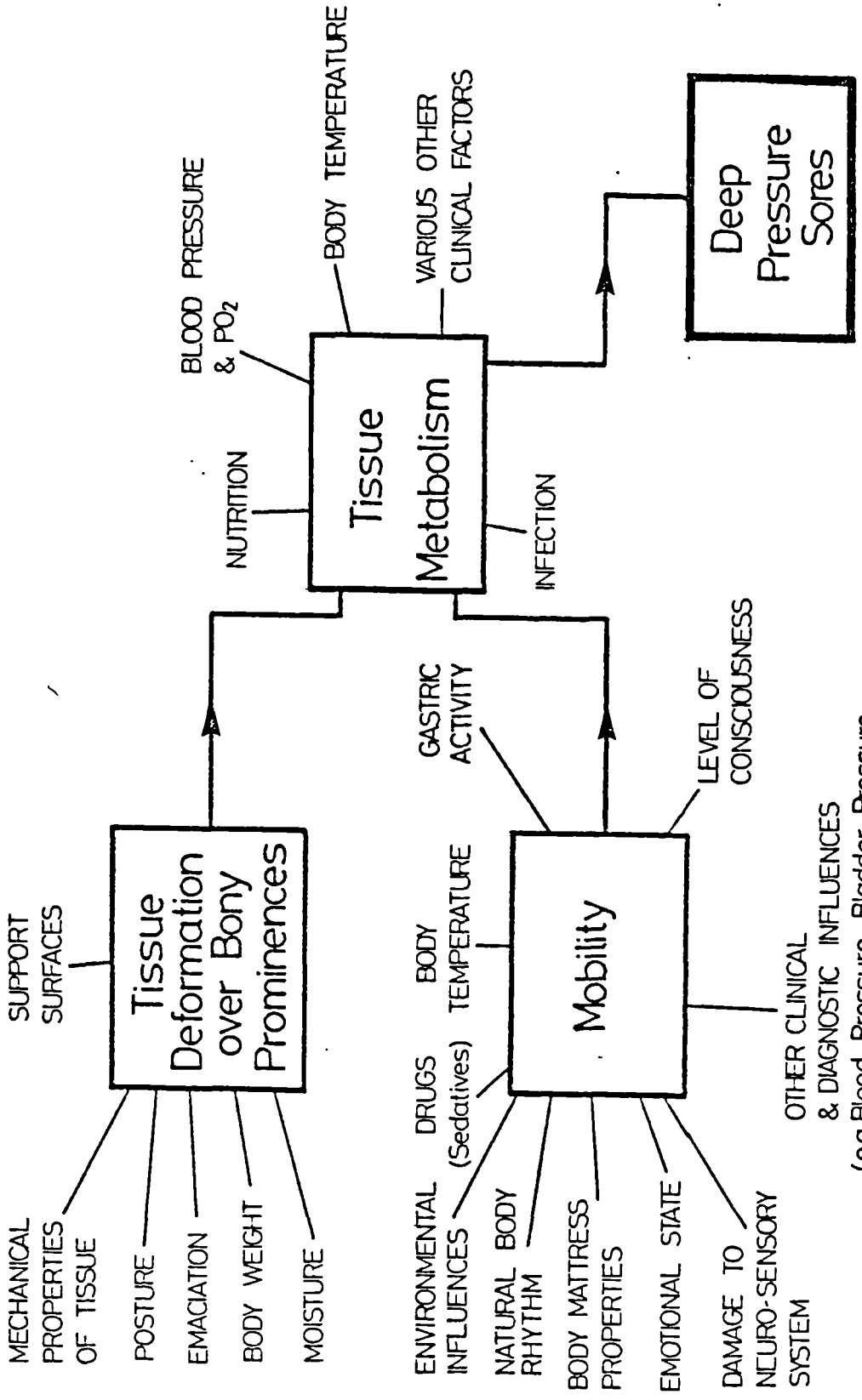


Figure 1.1 Parameters associated with the causation of deep pressure sores.

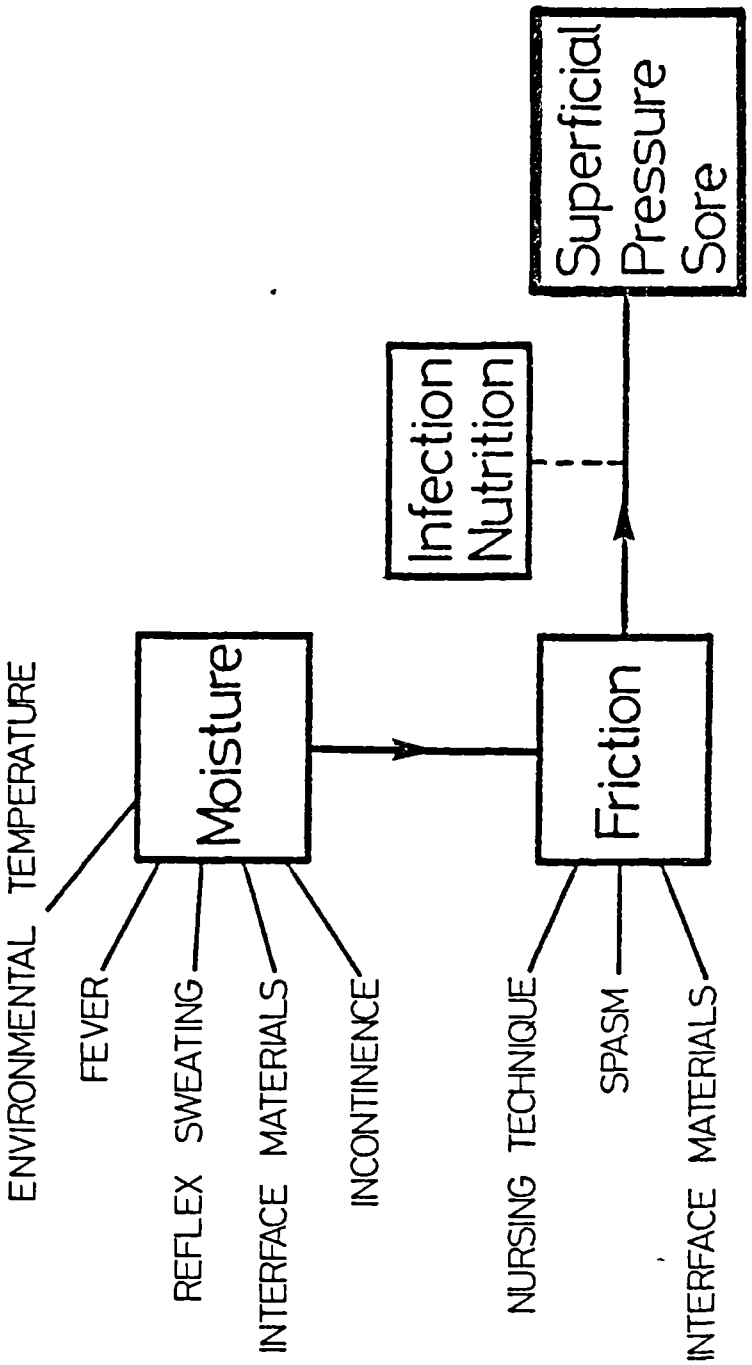


Figure 1.2 Parameters associated with the causation of superficial pressure sores

deficit, the movements that a patient would normally make in response to sensation of discomfort may be reduced. Examples of indirect parameters, such as the tightness of the bed linen, may modify the effectiveness of support surfaces in reducing tissue compression and the use of hypnotics, sedating the patient, may reduce his motility. Other parameters, such as diabetes, blood pO_2 etc. can act to predispose to deep pressure sores by altering the tissue metabolism and therefore the ability of the tissue to withstand ischaemic insults.

The aetiology of superficial pressure sores indicates that two primary factors are associated with with their causation.

- friction, between the skin and the support surface.
- moisture, the presence of which promotes maceration of the skin.

In a similar manner to the aetiology of deep pressure sores, a number of clinical and environmental parameters can influence these two primary variables (figure 1.2). Patients suffering from spasm, are subject to the continuous rubbing of their skin against an abrasive surface increasing the risk of developing superficial sores. The skin of patients who are incontinent or who suffer from uncontrolled sweating, may be wet, rendering it vulnerable to damage in association with friction. Examples of environmental parameters include interface materials (bedsheets, drawsheets, patient clothing etc.) which may modify the friction developed.

Variables such as systemic infection and nutrition can affect both deep and superficial sores. Bacteria, resulting from a systemic infection, congregate in tissue during mechanically induced ischaemia predisposing it to damage. The presence of bacteria is also commonly associated with established superficial sores - delaying healing and complicating treatment. Poor nutrition can affect all of the soft tissues, but it is most often linked to pressure sores in relation to an impairment of the skin's metabolism.

A pressure sore often has major social impact for the patient. To an already disabled person the occurrence of a sore imposes further difficulties, both for himself and for those who care for him. To those patients otherwise

recovering from illness, a pressure sore may retard their recovery and prolong hospitalisation. As a result of this the patient, removed from his home environment, is without the personal attention of family and friends and may be unable to provide a stable role in the family unit.

Pressure sores have traditionally been considered to represent a failure in the care provided by medical and nursing staff, responsible for the patient. They are "care-intensive", and inadequate management or availability of resources for their prevention, have often justified the thesis that they could be used as an index of the quality of basic care being delivered by a ward or hospital. The guilt that has become associated with a patient developing a pressure sore, is indicative of their being seen as a measure of nursing proficiency. However, this view has become moderated by recent studies which indicate that, of those patients in hospital who have pressure sores, many already had a sore or had suffered irreversible damage to their tissues that would result in a sore, before being admitted. In addition, it has been suggested that many of those sores that do arise in hospital are attributable not to nursing care directly, but to the use of trolleys, transit wheelchairs (Crewe, 1981), and operating tables with hard surfaces.

Economically a pressure sore can have considerable importance. Not only is a patient prevented from returning to work, but some of the difficulties inherent in a pressure sore, involving the possibility of further spells in hospital without any warning, or guarantee against recurrences, can act to make it difficult for them to get a job. This may be illustrated by the example of a sheltered workshop known to the author, which employs disabled people, but is concerned about employing patients particularly vulnerable to pressure sores. The limited number of positions available means that, while a patient with a pressure sore is in hospital and his job is kept open, other disabled persons on the waiting list for sheltered employment who are able to provide consistent attendance, would be denied an opportunity to fill the vacant space. Pressure sores may have an economic influence on the families of the patient. Not only may the patient be unemployed but the cost of time and travel involved in coming to visit him in hospital may prove to be important, particularly if he is

hospitalised for a long duration.

Pressure sores make a significant impact on health care budgets, both in privately funded and state funded systems. Extensive resources of manpower and equipment are required in their prevention and treatment. There have been a number of attempts to estimate the cost of pressure sores to the Health Service (eg. Fernie, 1973). However, these were based upon the premise that patients with pressure sores were occupying a hospital bed solely for the purpose of treatment of the sore. In reality such patients are usually in hospital for treatment of a "primary" condition, the sore being an additional factor. For this reason these estimates may be misleading. However, extensive resources are required for the treatment of pressure sores. Typically a patient with a sore requires a regime of care including assessment of the severity and causes, extensive nursing to treat it topically and in many cases, plastic surgery. Special support surfaces may be used, eg. ripple mattress, rotating bedframe, pillows, sheepskins, etc. To aid in the treatment of clinical factors (eg. systemic infection, poor nutrition etc.) drugs might be utilised. In an effort to avoid any recurrence of the sore, suitable prevention regimes may be initiated. Thus, pressure sores require considerable effort, resources and in many cases ingenuity, in their care.

It has been shown that in the Greater Glasgow Health Area 66% of those hospital patients with a pressure sore are elderly (Barbenel et al, 1977). Other epidemiological studies have shown similar trends. Thus, pressure sores are increasingly prevalent with age. The movement towards increasing life span, allied with that of increasing population (it is estimated that by 1996 the number of people aged 75 or over, will have increased by 42% (Norton, 1979)), suggests that the number of pressure sores will rise dramatically. Thus, if the same level of care is to be maintained, the resources required for treatment of pressure sores will have to increase correspondingly. The increasing percentage of elderly patients in the overall population suggests that the Health Service will need to spend proportionately more on the problem of pressure sores in the future.

Care of pressure sores has been a subject of interest for much of man's

history. The oldest known medical record, the Smith papyrus (2600 - 2200 B.C.) describes a method of treating "gaping wounds in the flesh" (Majno, 1975). Since that time, innumerable different types and forms of topical therapy, regimes of care, special support surfaces and mechanical aids have been advocated and nearly as many have been discarded.

The criteria for selection of a particular regime for the prevention or treatment of pressure sores, is based primarily on the aetiological factors responsible for their causation. Examples of such regimes include regular turning of a patient, whose own mobility is impaired; modification of support surfaces to improve the distribution of pressure around the bony prominences; topical applications to the sore and to the skin surrounding it, to counter infection and to encourage normal tissue metabolism; correction of posture, encouraging the patient to lie in such a way as to reduce in-plane forces (particularly in the area of the sacrum); drug therapy correcting systemic factors such as cardiac insufficiency, or infection; implementation of a suitable diet to improve nutrition and tissue metabolism. This diversity of the forms of treatment of pressure sores, emphasises the associated complexity of their aetiology.

However, even after successful treatment resulting in healing, a pressure sore remains a cause for concern. The initial damage, reducing the coverage of tissue over the bony prominence, results in the skin being subjected to a higher proportion of load, and the limited mechanical properties of healed skin (reduction in extensibility, inferior blood supply), may mean that the site of a previous sore is particularly susceptible to further tissue breakdown. A recurrent sore is often more severe and correspondingly difficult to heal and sometimes requires plastic surgery. In an effort to avoid future recurrences, it is usual for patients to make a regular assessment of their skin, particularly at sites of previous sores, checking for any persistent red patches which might indicate damage. Any such cases can then be treated before the skin breaks down.

The limitations and extra demands presented to a patient with a pressure sore may result in depression during a period of hospital care with the

knowledge that there is the possibility of a recurrence, and the social and economic difficulties that would involve.

The epidemiology of pressure sores has been established in a number of studies, including those of Petersen and Bittmann (1971) and Barbenel et al (1977). The prevalence of sores was established in relation to variables describing the characteristics of the population studied (age, sex, site on the body, etc.) and clinical variables commonly associated with their aetiology (mobility, diagnosis, incontinence, level of consciousness etc.). Information obtained by these epidemiological surveys, served to establish the relative importance of some of the different aetiological factors. All of the surveys emphasised the susceptibility of the elderly to pressure sores. Both bedfast and chairfast patients who exhibited an impaired mobility also displayed a marked susceptibility to pressure sores.

The importance of pressure in the aetiology of pressure sores, has prompted a number of studies to determine the magnitude of pressure and duration required to produce a sore. Animal studies involving the replication of sores by the application of a range of different pressures for varying durations have been conducted by Groth (1942), Husain (1953), Kosiak (1959), Dinsdale (1970) and Daniel et al (1981). Each of these has demonstrated an inverse relationship between the applied pressure and the duration necessary to initiate a sore, although they are in disagreement as to the magnitude required. The relationship established in these different studies varies considerably in terms of the pressure - time magnitude required to produce a sore. However, because of the different soft tissue coverings of animals, compared with those of man, these results although demonstrating an important physiological relationship should not be extrapolated quantitatively to man. In addition, these studies considered only the effect of a single episode of pressure, without the influence of any predisposing factors (other than paraplegia), further removing their results from the human situation.

Research conducted by Exton-Smith and Sherwin (1961) demonstrated that mobility was a significant factor in the aetiology of pressure sores. They monitored the spontaneous mobility of 50 elderly patients during sleep and

discovered that those who moved infrequently developed pressure sores. The significance of infrequent pressure relief as an aetiological factor in the production of pressure sores has been confirmed by epidemiological studies. Bardsley (1977) studied the spontaneous mobility of young adult volunteers during sleep. He designed a monitoring system capable of accurately recording changes in position of the subject in the bed, and developed associated analysis techniques. This study provided baseline data describing the overnight mobility of normal subjects sleeping in a controlled environment. Additionally, Bardsley demonstrated that the use of different mattresses can affect a subject's overnight mobility. This study was limited to:

i Normal young volunteers

ii Monitoring in a sleep laboratory rather than the normal or clinical environment.

In association with these studies, a research programme at the University of Strathclyde was undertaken to investigate the effect of different support surfaces upon conditions at the patient/support interface. This involved a number of individual projects. Using a range of support surfaces, studies of the interface pressure, temperature and humidity and overnight mobility were conducted (Bardsley, 1977; Ferguson-Pell, 1977; Adamson, 1978). The mechanical properties of different mattresses were also assessed (Small, 1977). The research reported in this thesis has extended the work of Bardsley, from the normal subject and the controlled environment to the elderly patient in the clinical environment. To evaluate the significance of the body movements recorded in relation to the pressure relief obtained, studies with normal subjects were undertaken. In order to obtain measurements of mobility in the clinical environment suitable apparatus was developed. Data produced by this apparatus were used to establish the characteristics of a patient's mobility with time, throughout the night and over a period of several days after admission. Clinical factors were recorded, including the nurses' assessment of the patient's vulnerability to pressure sores and the use of sedatives.

CHAPTER 2

OVERVIEW OF THE AETIOLOGY AND EPIDEMIOLOGY OF PRESSURE SORES

The extensive literature on the causes and possible methods of prevention and treatment of pressure sores, emphasises the extent of the problem and its complexity. Interpretations of the aetiology of pressure sores have been based on experimental studies, clinical experience and in some cases, rather simplistic theorising. In this chapter an overview of this literature will be given, with particular emphasis placed on studies which have provided sound experimental or clinical evidence to illuminate the problem. Epidemiology is a powerful method for identifying the causes of disease. Not only is the extent of the problem established, but careful use of suitable statistical, analytical methods in carefully documented surveys, can provide a valuable insight into the clinical factors associated with its causation.

2.1 CURRENT CONCEPTS OF THE AETIOLOGY OF PRESSURE SORES

Daniel et al (1979) refer to an observation by Thompson Rowling (1961) describing pressure sores on the body of Egyptian mummies, thereby demonstrating that the aetiology and treatment of pressure sores probably extends to the same distant past. The complexity of the problem that pressure sores present is indicated, in part at least, by the plethora of ideas, opinions and conclusions relating to this subject, that have been proposed in the literature over the past two centuries.

Reviews of the topic by Fernie (1973), Berecek (1975), Guttmann (1976), Small (1977) and Torrance (1981) have suggested that the aetiological factors may be grouped into two categories,

- extrinsic, to do with the patient's environment and,
- intrinsic, to do with the patient's general and clinical condition.

Alternatively it is possible to consider the factors as "Primary", which serve as the principle variables, and "Secondary" which perform a supplementary role and may be considered as predisposing factors.

2.1.1 Primary factors

Pressure sores may be divided into two groups with reference to the mechanism of their pathology:- those that start in the deeper tissues (deep sores) and those that start superficially (superficial sores).

Deep sores: Primary factors responsible for this type of sore are deformation of the tissue and immobility, (vide chapter 1).

Usually the forces responsible for deformation of the tissues, are generated as a result of body weight being transmitted through the skeletal frame and soft tissues to the support surface (Ferguson-Pell, 1980). Areas of particular concern in relation to pressure sores are those where the localised forces within the tissue are highest - usually occurring over bony prominences (ischial tuberosities, sacrum, trochanters, heels, etc.). These forces may act in any direction. However, to simplify analysis, they are usually considered in component form and are termed either "normal" or "in-plane" (shear). As discussed in much of the literature, these components are

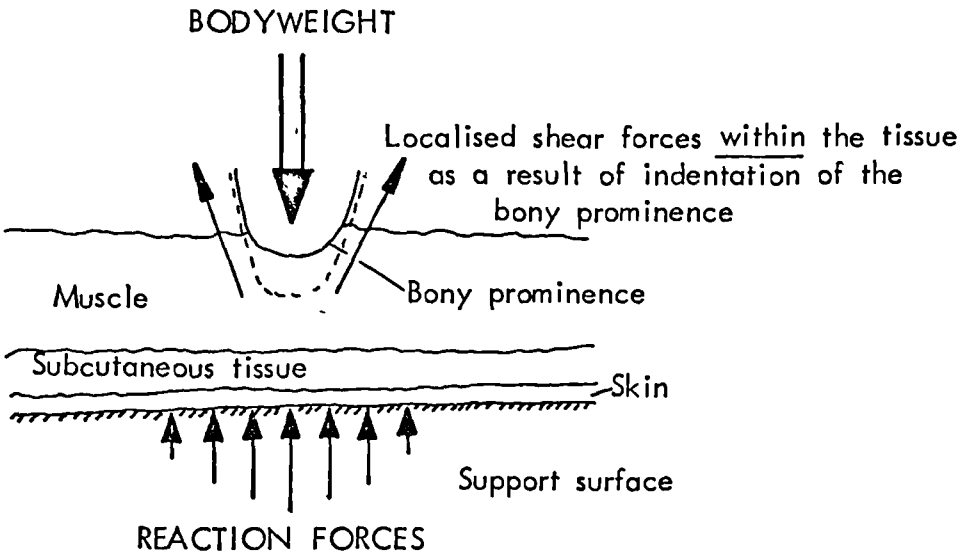


Figure 2.1 Schematic representation of the affect of a weight bearing bony prominence, generating normal reaction forces at the body/support interface and shear forces within the tissue itself.

considered as reaction forces that are developed between the surface of the skin and the support surface. For this reason the pathology of pressure sores described in terms of deformation of the tissue, has usually been related to the action of interface forces only. However, studies by Reddy et al (1980) have demonstrated that there may be significant shear forces within the tissue, resulting from indentation of the bony prominence, although at the interface only a simple normal force is evident (figure 2.1): this combination of the normal and shear forces, resulting in a complex distribution of stress within the tissue. The production of in-plane forces at the skin-support surface interface, perhaps as a result of the body sliding down the bed, serves to augment the effects of shear force already present within the tissue (figure 2.1), resulting in the potential for increased damage. There are principally two explanations as to how deformation can cause damage to the tissue, resulting in a pressure sore.

Firstly, deformation causing damage to the microvasculature (Brand 1976). The accumulative effects of repeated loading, with associated impairment of the healing mechanism, leads to irreversible damage to the microvasculature, producing a gradual reduction in the blood supply.

Brand (1976) applied repetitive "stress" to the footpads of rats. He was able to modify the force applied and frequency of application. Pressures of 15 kPa (115 mmHg) with 5,000 - 15,000 repetitions, produced damage to the tissue characterised by a rise in temperature, associated with an increase in the time taken for it to return to normal. Additionally, when the pressure was reapplied, approximately a week later, the tissues were found to have an increased sensitivity, requiring a smaller number of repetitions to produce an equivalent increase in temperature. Histologically, inflammation was evident, associated with oedema and necrosis.

This study has served to introduce the concept of repeated tissue deformation damaging the microvasculature, leading to pressure sores. However, its effect and exact mechanism in relation to man, is still unclear in quantitative terms.

Secondly, the more widely accepted theory, suggests that the

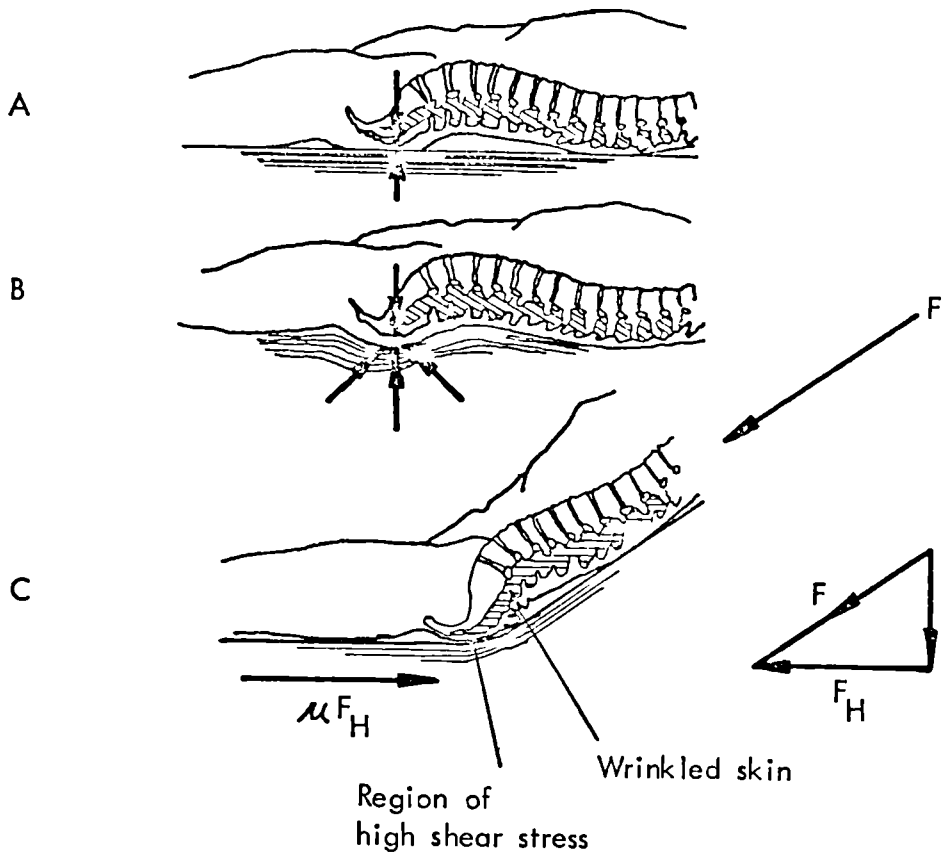


Figure 2.2 Tissue damage at the sacrum, resulting from normal and shear stresses. (after Reichel, 1958)

deformation and occlusion of the blood vessels, resulting from the application of normal or in-plane forces to the tissue, if allowed to persist for long periods of time, leads to tissue death due to a lack of nutrients and an excess of metabolites. Kosiak (1959), Bailey (1967), Bell et al (1974), Guttmann (1976) and others, have repeatedly emphasised the importance of relieving localised pressure to prevent pressure sores.

Action of in-plane forces has been reported by Reichel (1958) (figure 2.2). When a patient is lying supine in bed, the tissues overlying the sacrum are compressed (figure 2.2 (A)) a softer support surface providing an improved distribution of the interface forces (figure 2.2(B)). If the head of the bed is raised then the body will tend to slide down it. The induced horizontal component of force (F_H), opposed by a frictional force μF_H, produces in-plane (shear) forces in the soft tissue overlying the sacrum (figure 2.2(C)). Because of the anatomy of the vasculature in this region, these in-plane forces render large areas of the tissue ischaemic. If this ischaemia is unrelieved then the tissues will die. It is evident therefore, that the mobility of the patient, in association with the deformation of his tissues, is of crucial importance in determining his vulnerability for developing pressure sores.

In normal subjects, spontaneous body movements are continually occurring (Kleitman, 1939, 1963). These reposition the body, providing relief of pressure, allowing nutrition of those formerly ischaemic tissues previously in contact with the support surface. However, if a patient is not able to perform these spontaneous movements (eg. he may be paralysed or suffering from a debilitating illness) then they become particularly susceptible to pressure sores.

The inverse relationship between normal force and duration of application necessary to initiate a pressure sore has been experimentally determined (vide 2.3). Exton-Smith and Sherwin (1961) provided experimental evidence of the importance of body movements in the aetiology of pressure sores. In a study involving a group of elderly patients, it was shown that those with low overnight mobility were particularly likely to develop pressure sores.

Details of this study and of other investigations into the measurement, assessment and analysis of mobility are presented in chapter 3. Additionally, experimental studies of the effects of pressure on the soft tissues are reviewed in later sections (vide 2.3 and 2.4).

Prevention of pressure sores arising from the action of normal and in-plane forces demands careful and frequent observation of the patient. The use of a regular turning regime, or of mechanical aids, such as ripple mattresses, (Bliss et al, 1967 ; Redfern et al, 1973), or rotating and tilting bedframes (Conway and Griffith, 1956; Young and Ogden, 1973) provide a regular relief of pressure. Additionally, support surfaces capable of redistributing the interface forces, such as water beds (Young and Ogden, 1973), and air beds (Scales and Hopkins, 1971), are routinely used in both prevention and treatment of sores.

To help eliminate in-plane interface forces, the patient, if possible, should lie horizontally. However, if it is necessary to lie in a semi-recumbent position, then care should be taken to prevent the patient from slipping down the bed. Schell and Wolcott (1966) suggested a maximum elevation of 30°. Reichel (1958) advocated the use of a padded footboard, fitted at the end of the bed, although this technique has led to difficulties, as it relies upon the patient's legs remaining straight. Usually, weak and paralysed patients are unable to transmit the in-plane forces because their legs bend and their body tends to slide down the bed. The need for vigilance and care by the health care team was given by Agate (1977), who claimed that a single badly executed nursing lift could give rise to a shearing action capable of producing a pressure sore.

Superficial sores: Initially superficial sores (vide chapter 1) are confined to the skin. The principal aetiological mechanism for their formation is related to the frictional force developed between the skin of the patient and the bedclothes, particularly if the skin is moist.

Friction initiates the superficial pressure sore by abrading the epidermis of the skin. The extent of the damage is related to the frictional properties of the support surface, distance over which the skin moves as a result

of the action of frictional forces and factors which might soften the skin, such as the presence of moisture. However, the friction reaction force can also give rise to shear effects in lower layers of the tissue, which may compromise its vasculature, even when there is no movement. Thus, in situations where forces are being produced sufficient to induce frictional damage, there are also likely to be associated shear forces that may be a further cause of damage. Experimental evidence of the effect of friction on skin, has been provided by Cruickshank (1976), who showed that the junction between the epidermis and the dermis is relatively resistant to friction and as a result its effect is initially confined to the epidermis. Dinsdale (1970) employed an experimental animal model in a study investigating the mechanism of pressure sore formation, resulting from the application of friction to the skin surface. By means of a mechanical pressure application system capable of simultaneously applying frictional force, pressure was applied to the soft tissue overlying the transverse and posterior spinous processes of the lumbar and thoracic vertebrae of a pig, with and without friction. An isotope clearance technique (^{86}RbCl) was used to assess the blood flow in the skin and subcutaneous tissues of the loaded areas. He concluded from his results that friction does not lead to the production of pressure sores by rendering the tissue ischaemic. Earlier studies by Dinsdale had determined that the combination of friction and pressure did reduce the magnitude of pressure necessary to form a sore. As a result, he was left to conclude that friction is a factor in the production of pressure sores and acts by applying mechanical forces to the epidermis.

However, Dinsdale's terminology fails to distinguish between damage resulting from shear, as opposed to that from friction. Damage caused to the epidermis alone is due to the effects of abrasion whereas any effects in the lower layers of the tissues, such as ischaemia in the skin, results from shear produced by the frictional force.

The effect of moisture on the skin resulting from incontinence or perspiration, is to reduce the resistance of the skin to friction while increasing the coefficient of friction between the skin and the bed surface (Bors and Comarr 1948; Schell and Wolcott, 1966; Lowthian, 1970; Woodbine, 1979). In

addition, moisture, by increasing the frictional coefficient, acts to augment the potential shear force developed in the tissues before slipping, further compromising the tissue. If the urine of an incontinent patient is infected, then there is the risk of an infected pressure sore (Rudd, 1962). An illustration of the importance of incontinence has been provided by Norton et al (1962), who found that within their test group (elderly patients), 39% of those suffering from incontinence developed pressure sores, as opposed to 7% of the fully continent patients.

To prevent damage to the skin by friction, Agate (1977) recommends the use of simple bandages, which are applied to areas of the body most at risk of damage due to friction, eg. the heels, knees and elbows. He also suggests mild sedation, warning that in other circumstances, sedation may affect the mobility of the patient, increasing the risk of pressure sores (vide 2.1.2.). Dyson (1978) recommends the use of air and foam rings, placed under the body at those sites which are particularly prone to damage by friction.

2.1.2. Secondary factors

Neurotrophic: The existence of a neurotrophic factor responsible for the development of pressure sores has long been a matter of controversy. Charcot (1879) was one of the earliest proponents of this theory. He suggested that a neurotrophic factor was responsible for regulating nutrition of the skin. If this system was damaged and pressure was applied to the skin, then the tissue would break down to form a "neurotic ulcer" (sic.). Munro (1940) also believed that injury to the nervous system was of importance in the aetiology of pressure sores. He believed that nutrition of the skin was related to the integrity of two reflex arcs, associated with the spinal cord. Thus, pressure sores in a patient with a concomitant injury involving the spinal cord were larger and developed quicker as a result of spinal shock affecting the local cutaneous vascular reflexes. However, corroborative evidence of a neurotrophic factor in the aetiology of pressure sores is limited and there has been no reported anatomical evidence of the trophic nerves suggested by Charcot (1879).

Whimster (1976) conducted a series of experiments on geckos. He amputated the tails of two geckos and removed the spinal cord from the amputation stump in one animal, and left the other intact. Within a few months the intact stump grew another tail, whereas the other animal did not. In a further set of experiments he describes removing a length of spinal cord from the tail of a gecko. After two months the skin in the denervated area had ulcerated and did not heal until nervous tissue had been re-established. Whimster concluded from these experiments that denervation of the skin predisposes to ulceration and inhibits healing. Brand (1976) commenting on the results of Whimster (1976) expressed doubts about the results in relation to humans. Brand claimed that, in fact, an ulcer in denervated skin of humans heals at approximately the same rate as they would if the tissue was normal, and that the principal effect of denervation is to lower the threshold to damage, preventing relief from applied pressures.

Evidence which is contrary to the existence of a neurotrophic factor has been provided by Brown-Séquard (1852), Husain (1953), Barton and Barton (1968).

Brown-Séquard (1852) severed the nervous supply to the hind limbs of a series of rabbits and placed half in a cage with a padded floor and the other half in a cage with a hard floor. After a period of weeks, only those animals in the cage without padding had developed pressure sores. Brown-Séquard concluded that the pressure sores had developed as a result of continued pressure in the presence of urine and faeces, and that there was no evidence of a neurotrophic factor. Thus, 27 years before Charcot's study, experimental evidence existed which indicated that pressure sores were not attributable to any neurotrophic affect. Husain (1953) demonstrated that division of the sciatic nerve of a rat, gave rise to no changes in the muscles of its leg (after three days). He applied pressure to the leg of a rat by means of a cuff which fitted around the leg. After application of 13 kPa (100mmHg) for one hour, the muscle was removed. Microscopic examination revealed the presence of cellular infiltration, oedema and haemorrhage. This was a similar result to that obtained with a normal rat at the same level of pressure

and duration. Thus, there was no evidence to substantiate the claim for a neurotrophic factor.

Barton and Barton (1968a) studied plantar ulcers occurring after neurectomy in mice. Severing the tendo-calcaneus or dividing the sciatic nerve, caused pressure sores to form on their heels within six hours of the operation. The sores were a result of thrombosis of capillaries in the sub-cutaneous tissues. However, division of sensory nerves did not lead to a tissue breakdown. In a similar manner, division of the sciatic nerve, failed to alter pressure sores, which had formed as a result of a previous tenotomy. Barton and Barton (1968a) concluded that the trophic effect of nerves supplying nutrients to the skin was unlikely.

Bors and Comarr (1948) and Constantian (1980) comment that although nerve injury may predispose tissue to pressure sores by loss of sensation and impairment of mobility, there is no objective evidence of a neurotrophic factor.

At a clinical level the secondary effect of nerve loss - muscle wasting, leads to a loss of muscle bulk and tone, over the bony prominence. This reduction in the volume of soft tissue available for dispersing any applied forces, helps to explain the increased vulnerability of spinal-cord injured patients to pressure sores.

Support surface: As described in section 2.3.1, one of the primary factors in the aetiology of pressure sores is related to the problem of supporting body weight, which leads to deformation of the surface tissues of the body, causing them to become ischaemic. If the pressure is unrelieved then irreversible changes occur in the tissue, leading to necrosis.

The support surface, that is the surface upon which the body rests, is important in determining the magnitude of the interface pressure. This has been illustrated by Kosiak (1961) who measured the magnitude of the interface pressure between the skin and the support surface, in normal human volunteers. He monitored the pressure using a number of flat rubber butterfly type valves placed on the skin overlying the ischial tuberosities. When the subject was sitting on a plain wooden office chair, the pressure under the ischial tuberosities was measured to be in excess of 40 kPa (300 mmHg). However, by the

use of a two inch thick foam cushion, altering the mechanical characteristics of the support surface, the interface pressure was reduced to approximately 21kPa (160 mmHg). Other studies by Lindan (1961), Newell et al (1970), Redfern et al (1973) and Jeneid (1976) have all produced results which indicate that the body/support interface pressure is influenced by the nature of the support surface. Thus, by the simple expedient of modifying the support surface, it is possible to reduce and modify the magnitude of the pressure applied to the weight bearing prominences of the body.

A support surface may act to provide prophylaxis of pressure sores by one, or the combination, of two methods (Bell et al, 1974).

Redistributing the pressure, such that the body is not subject to concentrated, localised pressures, particularly in the area of the bony prominences, but rather reduced pressures distributed over a large area. Examples of support surfaces providing a redistribution of pressure include, "floatation" devices such as the water bed (Young and Ogden, 1973), support pads of wool, Acrilan or synthetic gel (Merlino, 1969; Moolten, 1972), total contact beds such as the fluidised bed (Artz et al, 1970), or even a careful arrangement of pillows (Redfern et al, 1973).

Regular relief of pressure, reducing the duration of ischaemia which is the result of an external pressure compressing the tissues. Support surfaces in this category can, while not promoting natural movement, provide a regular readjustment of the body position. Devices employing this principle include, alternating pressure mattresses (Bliss et al, 1967; Redfern et al, 1973), and constantly rotating bedframes (Conway and Griffith, 1956; Young and Ogden, 1973).

A comprehensive review of the use of the support surface in the prophylaxis and treatment of pressure sores is provided by Small (1977).

Nutrition: The effects of malnutrition have long been regarded as one of the major factors in the aetiology of pressure sores (Wada, 1922). Poor nutrition may lead to one or a combination of illnesses, such as hypoproteinaemia and avitaminosis, which predispose to the formation of pressure sores.

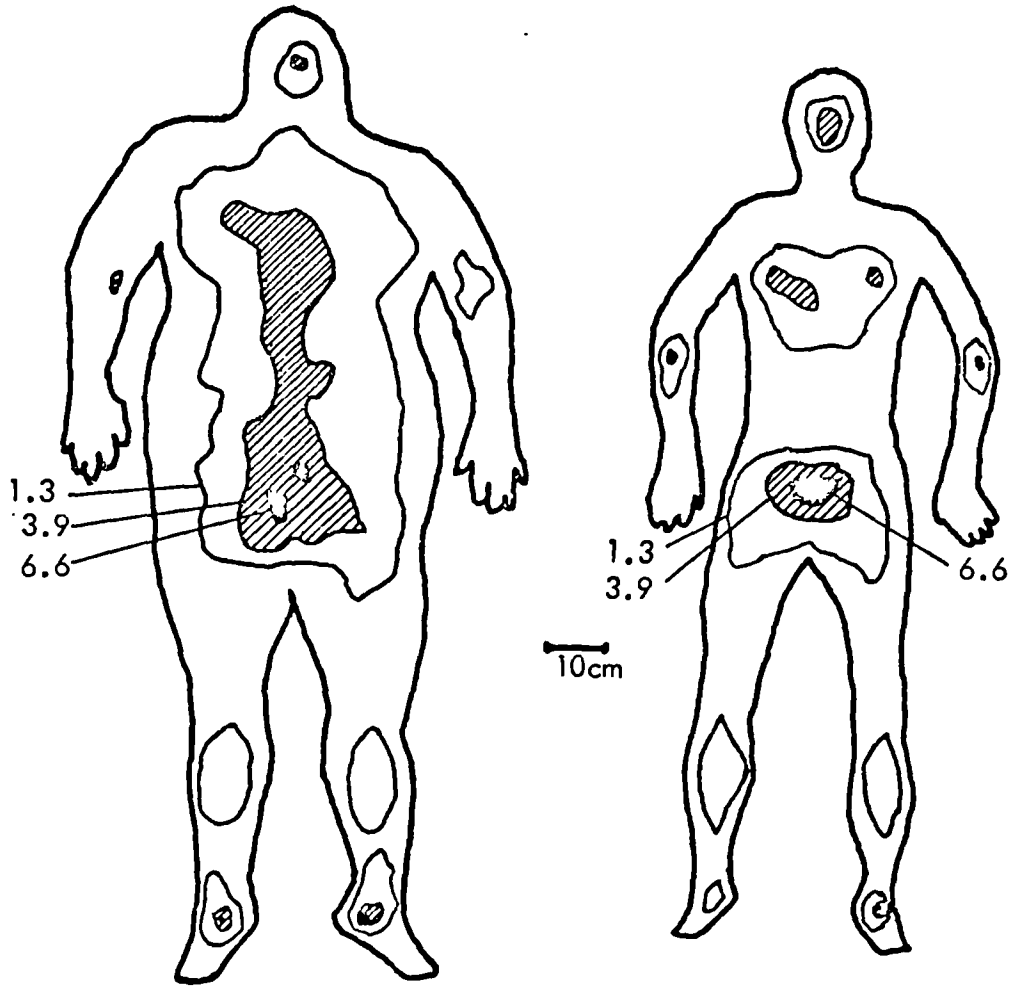


Figure 2.3 Pressure distribution over the body surface for underweight and overweight subjects lying in a supine position (kPa) (after Lindan, 1965).

Typically this should be a high carbohydrate, high protein, moderately low-fat diet (Schell and Wolcott, 1966; Moolten, 1972). Additionally, if the sore is large there may be a significant loss of metabolites. To replace these, and to improve the rate of wound healing, the diet may be supplemented with extra proteins and vitamins. Moolten (1972) also advocates the supplementary use of an anabolic steroid.

Drugs: The use of some drugs may predispose the patient to the formation of pressure sores (Torrance, 1981). Perhaps the most commonly referred to drugs in this context are those affecting the neurological faculties of the patient. These may be "hypnotic" drugs, used to sedate the patient, or pain killing drugs, affecting the neuro-sensory system. However, whatever the nature of the drug it will act to predispose the patient to a pressure sore in a secondary manner, influencing mobility or depriving the body of an effective stimulus, such as pain, to warn of impending damage by ischaemia. A study by Exton-Smith (1967) provided evidence that the majority of sedatives lead to an impairment of the natural mobility of a patient.

A rather less obvious hazard posed by the use of drugs was outlined by Barton and Barton (1968b). They established that a single dose of ACTH provided protection against traumatic pressure sores. For maximum efficacy, the drug must be administered four hours before the pressure damage is expected to occur (typically an operation). However, if the drug is applied in the peri-operative period, that is immediately before, during or after the operation, it will afford no protection and may even prove to be deleterious. Thus, due to the often complex interaction between drugs and the body, their ability to predispose towards pressure sores is not always clear.

When prescribing drugs, their possible side effects, with regard to pressure sores, should be considered, and this is particularly true if the patient is elderly or paralysed.

Weight: Lindan et al (1965) compared the distribution of pressure on the body surface of an obese male (overweight) and a thin female (underweight). Figure 2.3 indicates that the peak pressures were slightly larger in the under-weight subject, although regions of lower pressure extended over larger areas

in the overweight subject. This result may be explained in relation to soft tissue covering. An overweight subject has substantial subcutaneous fat and this acts to distribute the interface pressures. However, underweight subjects have little subcutaneous fat and this provides for a poor distribution of interface pressure, indicative of high peak pressures.

Williams (1972) compared the occurrence of pressure sores with some of the more commonly accepted aetiological variables (eg. age, body weight, incontinence etc.) in a survey of 26 non-ambulatory patients of varying age and sex, suffering from different illnesses. She determined bodyweight to have the highest correlation with pressure sore incidence, suggesting that those subjects who were underweight were more liable to develop pressure sores.

Anaemia: A patient suffering from a deficiency in either quality or quantity of red blood cells is said to be anaemic.

Moolten (1972) observed fifty patients with pressure sores, 31 of these had "deep" pressure sores, that is involving both the skin and underlying tissue, a significant number, 8 (26%) had levels of haemoglobin less than 10g/100ml. Kosiak (1959) considered anaemia to be an important factor in the aetiology of pressure sores, arguing that it is possible for ischaemic tissue to survive for longer periods, if the haemoglobin content and the supply of oxygen, within the blood are normal.

In an effort to avoid anaemia, Matheson and Lipshitz (1956) and Moolten (1972) advocate the transfusion of whole blood, to maintain a haemoglobin level of 10 - 12.5g/100ml.

Infection: It is commonly accepted that established pressure sores are liable to infection, (Constantian, 1980). However, the role of infection in the causation of pressure sores is less clear. Severe systemic infections such as typhoid, puerperal sepsis, osteomyelitis, nephritis and chronic blood diseases cause anaemia and affect nutrition, which in their turn are known to predispose a patient to pressure sores.

Husain (1953) conducted a series of experiments, whereby he injected a bolus of Streptococcus haemolyticus into the jugular vein of rats, and applied a pressure cuff to their legs. The rats were divided into three groups, which

were subjected to 13kPa (100mmHg), three days, one day and immediately after the injection of the bacteria. The animals were sacrificed three days after pressure application. Results indicated that, when present in the blood, bacteria became localised at the site of the pressure application. Large abcesses of the muscle were present in the animals to whom the pressure had been applied immediately after administration of the bacteria. Smaller abcesses in the muscle formed when pressure was applied 24 hours after injection of the bacteria. In the group of animals subjected to pressure three days after administration of the bacteria, when the blood was "sterile", no abcesses or bacteria were located at the site of pressure application. The variability in the magnitude of the abcesses was presumably related to the level of bacteria in the blood, although no attempt was made to either measure or to estimate it. Husain concluded from these results that a systemic infection may predispose towards pressure sores, by the formation of abcesses at the site of pressure application.

Torrance (1981) suggests that another possible mechanism by which infection might act in the formation of pressure sores, is by causing pyrexia, leading to moistening of the skin with an increased risk of the development of a superficial sore (vide 2.1.1).

Heat: Schell and Wolcott (1966) described heat as a factor in the production of pressure sores. A rise in body temperature increases the metabolic rate and therefore the demand for oxygen, in a region already deficient because of the ischaemia caused by compression of the tissue. Williams (1972), in a survey of 26 non-ambulatory patients, provided experimental evidence of this, when she determined a correlation between body temperature and skin breakdown, concluding that those who have an elevated body temperature are at an increased risk of pressure sores. However, the low number of subjects in her survey precluded any rigorous statistical analysis. Dyson (1978) proposed that the action of an increased ambient temperature led to additional perspiration with possible maceration of the skin and a concurrent increase in the growth of bacteria, all of which are factors predisposing to pressure sores.

2.2 EPIDEMIOLOGY OF PRESSURE SORES

The epidemiology of pressure sores has been the subject of a number of studies (Petersen and Bittman, 1971; Barbenel et al, 1977; Jordan and Nicol, 1977; Lowthian, 1979). Their results have indicated an overall prevalence of pressure sores in the hospital, ranging from 3% (Petersen and Bittmann, 1971) to 9.4% (Jordan and Nicol, 1977). David (1981), in a comparison of some of these surveys, considered their results to be of use in the numerical estimation of those patients with pressure sores and as an indicator to the particular groups of patients most prone to sores.

These epidemiological studies, also indicated the relative prevalence of superficial sores to deep sores. Barbenel et al (1977) employed a grading system in the assessment of the severity of the sores, ie. 1 = skin discolouration, 2 = superficial sore, 3 = destruction of the skin-no cavity and 4 = destruction of the skin-cavity. However, because of the limited time available for a nurse to make an assessment of the skin's condition, it was not possible to reliably differentiate between persistent red skin, a precursor to a pressure sore, and that resulting from simple reactive hyperaemia, a normal healthy response. For this reason, Barbenel et al discarded the grade 1 classification. Additionally, on the basis of the description of deep and superficial pressure sores, provided in section 2.1.1, both grades 2 and 3 are representative of superficial sores, while grade 4 describes a deep sore. Thus, combining grade 2 and grade 3 sores as superficial, 84% of the pressure sores observed were superficial, 16% being deep, indicating the predominantly higher prevalence of superficial sores. However, these studies are unable to determine the relative prevalence of those that start superficially, as against those that start in the deeper tissues. Thus it is possible that some sores assessed to be deep, might have started superficially, or alternatively, that deep sores in an advanced stage of healing could have been assessed to be superficial.

2.2.1 Site

The epidemiological surveys suggested that pressure sores can occur at a wide range of sites on the body. However, those identified as being the

Data related to sores occuring in a population of 7725

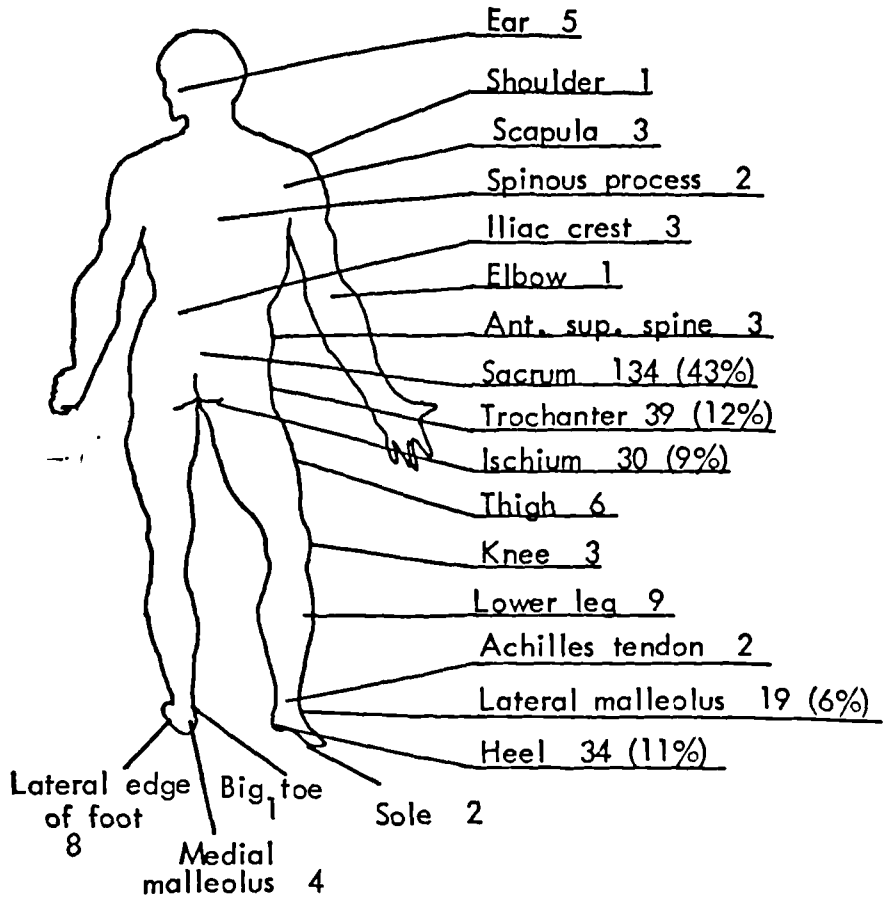


Figure 2.4 Common sites on the body of pressure sores.
(after Petersen and Bittmann, 1971)

most prevalent were the sacrum, ischial tuberosities, greater trochanters and heels, the largest number of sores occurring at the sacrum. The results of (Petersen and Bittmann, 1971) in relation to the site of pressure sores are described by figure 2.4.

Bardsley (1977) attributed the distribution of the number of sores in the pelvic area to the posture of the patient. The majority of patients with ischial pressure sores being chairfast, and the majority with trochanteric ulcers being bedfast. However, both chairfast and bedfast patients suffer from sacral sores suggesting a possible explanation for the increased prevalence of sores in this area.

2.2.2 Population at risk

The results from all of the surveys emphasised the importance of age, consistently showing a steady rise in the prevalence of pressure sores with increasing age (figure 2.5).

Patients suffering from a certain disease, or group of diseases, were identified in these surveys as having an increased prevalence of pressure sores. Barbenel et al (1977) discovered that those patients suffering from multiple sclerosis, paralysis agitans and cerebral paralysis demonstrated a particular vulnerability to sores. Petersen and Bittmann (1971), Jordan and Nicol (1977) were able to link particular diseases with pressure sores. In general, these diseases were those commonly associated with old age such as arteriosclerosis, cerebral haemorrhage, cardiac insufficiency and peripheral vascular disease.

2.2.3 Mobility

The mobility of patients was assessed in terms of their ability to walk. Additionally, some of the studies distinguished between chairfast and bedfast patients.

Results from the pressure sore surveys all indicated that decreasing mobility was linked to an increasing susceptibility to pressure sores (Petersen and Bittman, 1971; Barbenel, 1977; Jordan and Nicol, 1977; Lowthian, 1979). The bedfast and chairfast patients exhibited a significantly higher proportion of pressure sores than the totally ambulant subjects (Barbenel et al,

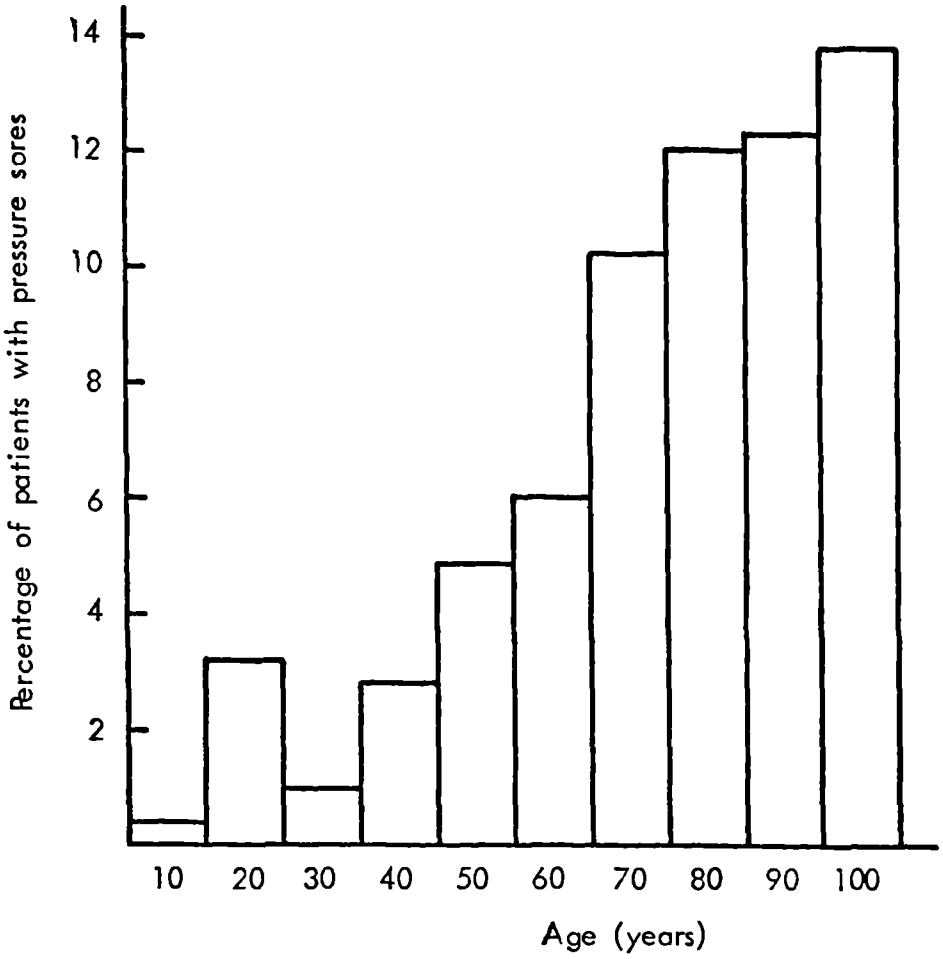


Figure 2.5 Frequency of pressure sores with increasing age. (after David, 1981)

1977). Further analysis of this data showed that the chairfast patients exhibited a marginally higher prevalence than did the bedfast patients. This is in contrast to Petersen and Bittmann (1971) who determined an increase in the incidence of sores in the bedfast relative to the chairfast. Jordan (1979) studied the pressure sore incidence in the elderly, using data gathered in an earlier survey (Barbenel et al, 1977). Of these elderly patients, 50% who were classified as "bedfast", developed pressure sores as compared with 3.2% of the totally ambulant, suggesting the vulnerability of the elderly to reduced mobility.

Experimental evidence, relating mobility to pressure sores, was established by Exton-Smith and Sherwin(1961). They constructed a movement monitoring apparatus (Sherwin et al, 1961) (vide 3.4.1) and applied it to the bed. Fifty elderly patients were monitored and their movement assessed. The data was characterised by the number of movements made by each patient during the night. Comparing the mobility of those patients that developed pressure sores to those who did not, it was apparent that overnight mobility was closely linked to the development of pressure sores. A threshold of movement score was developed, below which the patient usually developed a pressure sore. Additionally, one patient with a score initially greater than the threshold, developed a sore two days after their score fell below the threshold level.

The results of the clinical study and of the surveys, serve to illuminate the importance of mobility in the aetiology of pressure sores.

2.3 EXPERIMENTAL STUDIES OF EXTERNALLY APPLIED PRESSURES

The importance of tissue deformation and immobility in the formation of pressure sores was outlined in section 2.1.1, and it has been the subject of a number of experimental studies. The author was a participant in one of these and that study is the subject of section 2.4.

2.3.1 Animal models

Groth (1942) applied pressure to the ischii of rabbits by means of a simple balance beam, one end of which held two 15 mm diameter spheres. By placing weights on the opposite end, it was possible to apply a pressure to the skin of the animal. The influence of infection and spinal cord transection were also investigated. His principal conclusions were;

- there is an inverse relationship between the pressure and duration necessary to develop a pressure sore.
- deep tissues are more susceptible than superficial tissues to the effects of pressure. In addition, due to the absence of gross pathological changes in the large blood vessels, he proposed that direct mechanical damage, rather than ischaemia, was the principal factor in the damage of the muscle.
- in infected animals, bacteria localise at the pressure site.
- the susceptibility of paralysed animals to develop pressure sores was the same as that of the normals.

Husain (1953) studied the effect of external pressure, by means of a pressure cuff, attached to the legs of rats and guinea pigs. He applied pressures of varying magnitude for varying durations. Tissue from the pressure site was removed, and prepared for histological analysis. As with Groth (1942), further studies were carried out into the effect of infection and spinal cord transection. The findings of Husain (1953) were in agreement with those of Groth (1942). Additionally, he established that a load equally apportioned over large areas is preferable to a localised or point load.

Kosiak (1959) applied pressure to the tissue overlying the femoral trochanter and ischial tuberosity of mongrel dogs. The pressure was applied by inverted 20 ml hypodermic syringes connected, in a closed system, to the

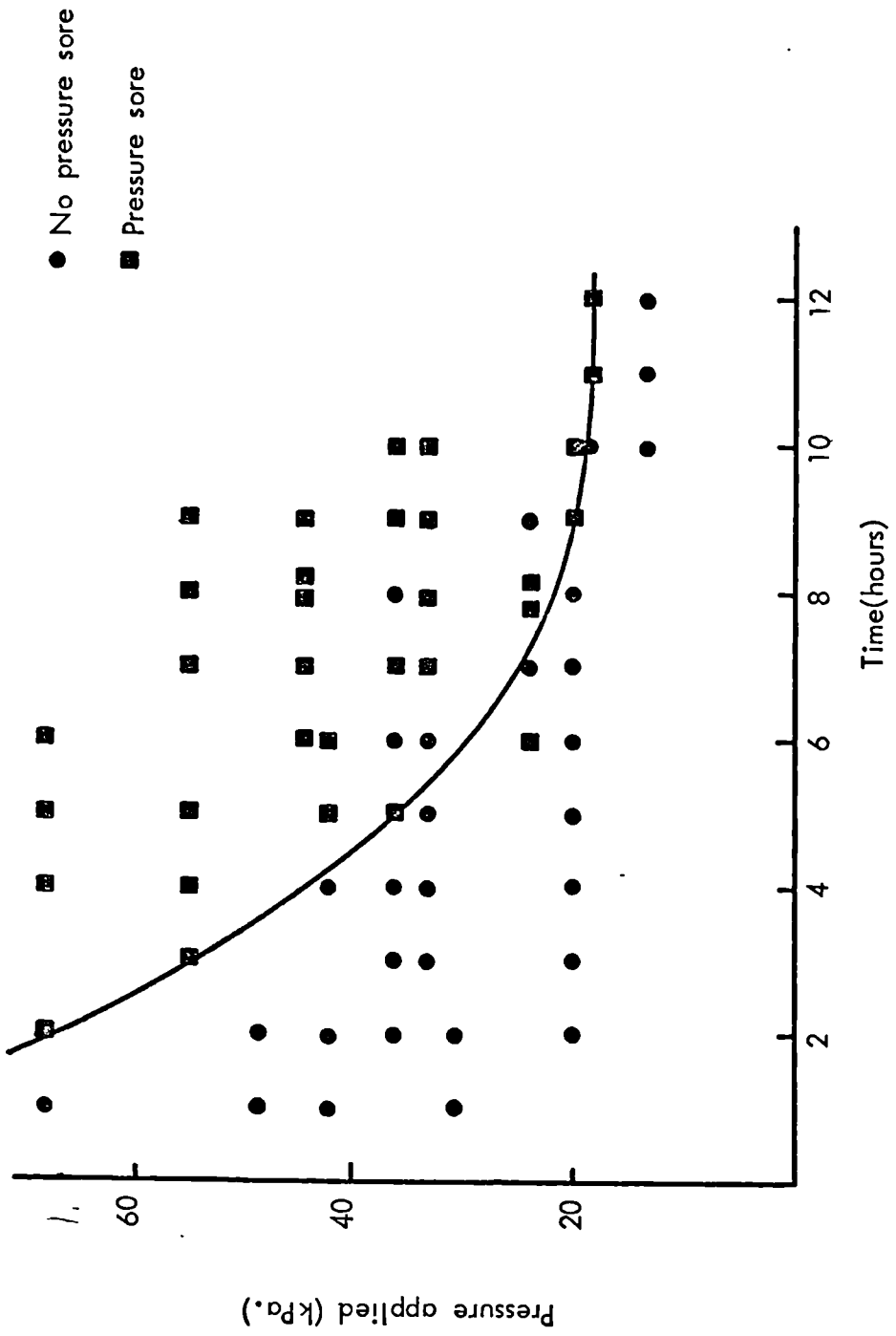


Figure 2.6 Values of pressure and duration necessary to produce pressure sores in the skin of dogs. (after Kosiak, 1959)

pressure reduction valve of a compressed air cylinder. The plunger of the syringe acted as an indentor, pushing against the skin surface. Calibration showed a 10% difference between "systemic" pressure, that is at the reduction valve, and the pressure measured under the indentor. After removal of the pressure, the animals were put into a pen and the pressure sites examined daily for 14 days, after which time the dogs were sacrificed. 62 experiments were performed on 16 dogs, showing an inverse relationship between pressure and duration, in agreement with Groth (1942) and Husain (1953) (figure 2.6). The average time to ulceration was four and a half days after removal of the pressure. The stress field in the tissue, resulting from the applied pressure, leads to the tissues of the contralateral side being subject to mechanical stress. This invalidates the use of the site contralateral to that being mechanically tested. In view of this, the results of Kosiak (1959) who performed 62 experiments on just 16 dogs, require careful interpretation.

In a later study, Kosiak (1961) applied pressure to the legs of rats. The animals were divided into two groups, the first of which received constant (that is unrelieved) pressures, ranging from 4.6 to 32 kPa (35 to 240 mmHg) for periods of one to four hours. The second group were subject to an alternating pressure for equal periods of time, with the pressure being relieved every five minutes for a period of five minutes. A further set of animals was rendered paraplegic and were subjected to a similar range of pressures. Twenty-four hours after removal of the pressure, the animals were sacrificed, the hamstring muscle removed and prepared for histological analysis. Results indicated a marked susceptibility of the muscle to continuous pressure, with 9.3 kPa (70 mmHg) for two hours producing changes in the muscle fibres. This was comparable to Husain (1953). The paraplegic animals showed no increase in susceptibility to the effects of externally applied pressure. Tissue subjected to alternating pressure, showed consistently less change, or no change, when compared with that subjected to an equivalent magnitude of unrelieved pressure. This result is contrary to the findings of Brand (1976) (vide 2.1.1) who demonstrated that repeated loading may impair the microvasculature sufficiently to produce a pressure sore. The disparity between the two repeated

loading studies is probably attributable to differences in the number of repetitions and in the experimental site on the animal.

Lindan (1961) investigated the vascular response to pressure in animals. Pressure was applied to the distal end of the pinna of rabbits, by means of a clip consisting of two plastic discs, each with a surface area of 360 mm^2. Springs connecting the disks were adjustable allowing the application of a range of pressures. The method was said to be accurate to within 10%. A single pressure of 13.3kPa (100mmHg) was applied for varying durations. Three hours after removal of the pressure, the compressed site had become hyperaemic and oedematous. However, the tissue recovered, and after a period of two days no significant damage was evident. This result contrasts to those of Groth (1942), Husain (1953) and Kosiak (1959), who observed changes at a pressure of only 8kPa (60mmHg) applied for one hour. However, these studies involved compressing tissue overlying a bone which acts to prevent an even distribution of the externally applied pressure throughout the tissue, and leads to areas of high stress. Further application of pressure by Lindan (1961) for 7 - 9 hours produced increasing damage. However, not until a duration of 13 - 15 hours of pressure application, was tissue necrosis apparent, with the development of a concomitant pressure sore. Lindan then reversed the protocol, applying varying magnitudes of pressure for a duration of 13 hours. No pressure sore was evident until 8kPa (60mmHg) had been applied. It was proposed that this technique, applying 13.3kPa (100mmHg) for 13 - 15 hours, was capable of reliably producing a pressure sore on the ear of rabbits. Lindan (1961) concludes that the use of a "pressure clip" in this manner may be extended to investigate the effect of other factors in the aetiology of pressure sores. However, the vasculature, soft tissue and lack of bony prominences of the pinna are unrepesentative of the usual site of a pressure sore.

All of the studies described in this section have involved the use of experimental animals, whose soft tissue covering is significantly different to that of man. Thus, as noted by Fernie (1973), their results should be treated in a qualitative manner and not extrapolated to the human situation.

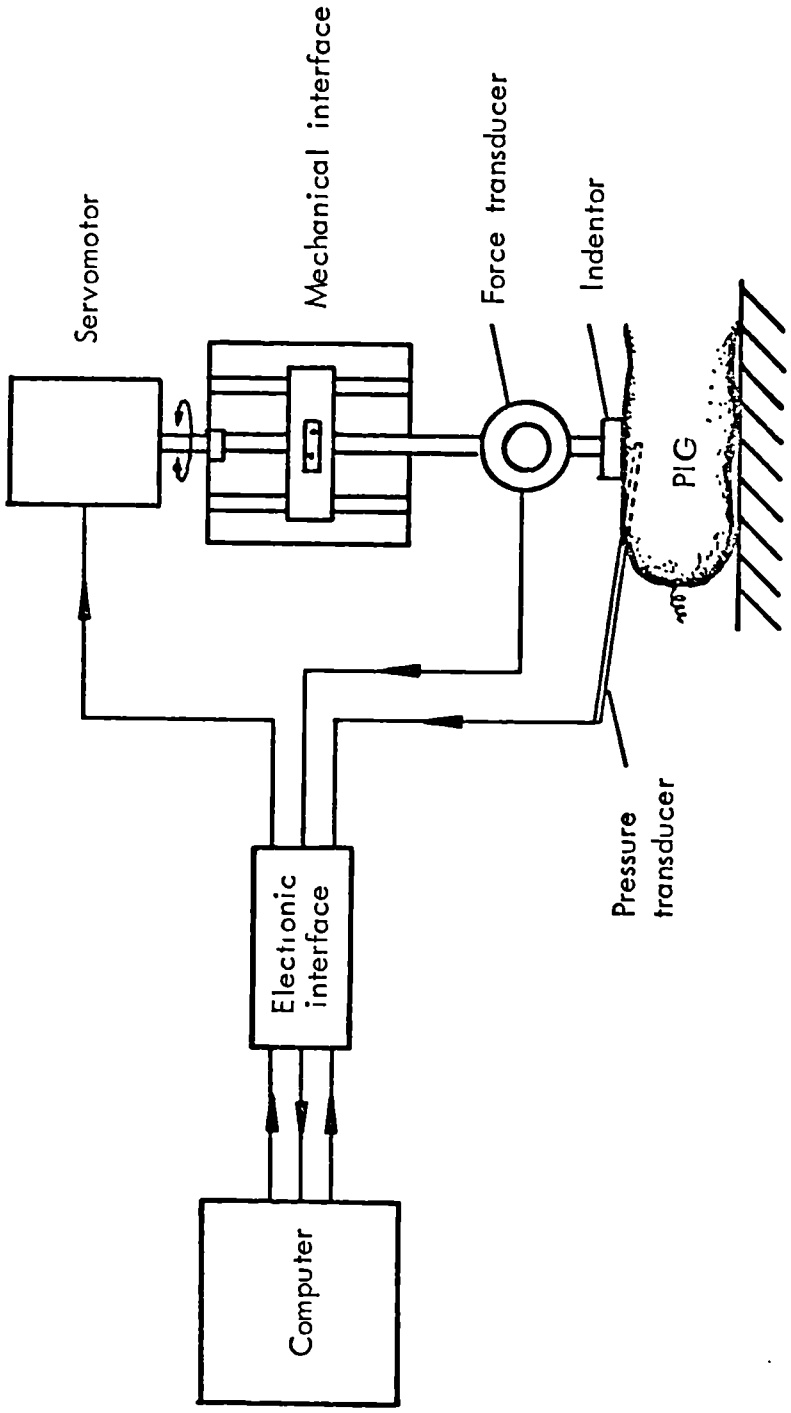


Figure 2.7 Schematic representation of the computer controlled pressure applicator.

2.4 A STUDY RELATING TO THE PATHOPHYSIOLOGY OF PRESSURE SORES

2.4.1 Introduction

An investigation into the effect of pressure on external tissues was conducted at the Royal Victoria Hospital, Montreal, Canada. The study was conducted by a team comprising a plastic surgeon, operating theatre technician, computer programmer, pathologist, and the author.

The primary aim of the study was to establish a relationship between the magnitude of external pressure, the duration of its application and any resulting tissue damage. All the experiments were performed on animals. The results of tests using "normals" were considered to represent a control group. By introducing different factors, thought to be significant in the aetiology of pressure sores, and comparing the results with those of the "normals", it was hoped to assess the relative importance of each.

Selection of an experimental animal for the study was based upon structural and functional similarities of its external tissues to those of man. The majority of the previous investigations into the pathophysiology of pressure sores, utilised loose-skin mammals, such as the rat, rabbit, and dog (Groth, 1942; Husain, 1953; Kosiak, 1959, 1961; Lindan, 1961; Nola and Vistnes, 1980). The skin of such animals is characterised by a thin epidermis and a dermis rich in hair follicles. The blood supply to the skin being closely associated with the nutrient requirement of the hair follicles. Alternatively, in the pig, the thickness of the epidermis, rate of mitosis and vasculature, are all similar to that of man and for this reason the pig was chosen as the experimental animal.

2.4.2 Apparatus

A computer controlled pressure applicator was used to compress the external tissues overlying the greater trochanter of a pig (figure 2.7). The apparatus consisted of a stepping motor, connected to an indentor by means of a simple screw mechanism, which converted the rotational output of the motor into the linear motion required to move the indentor (figure 2.8). A

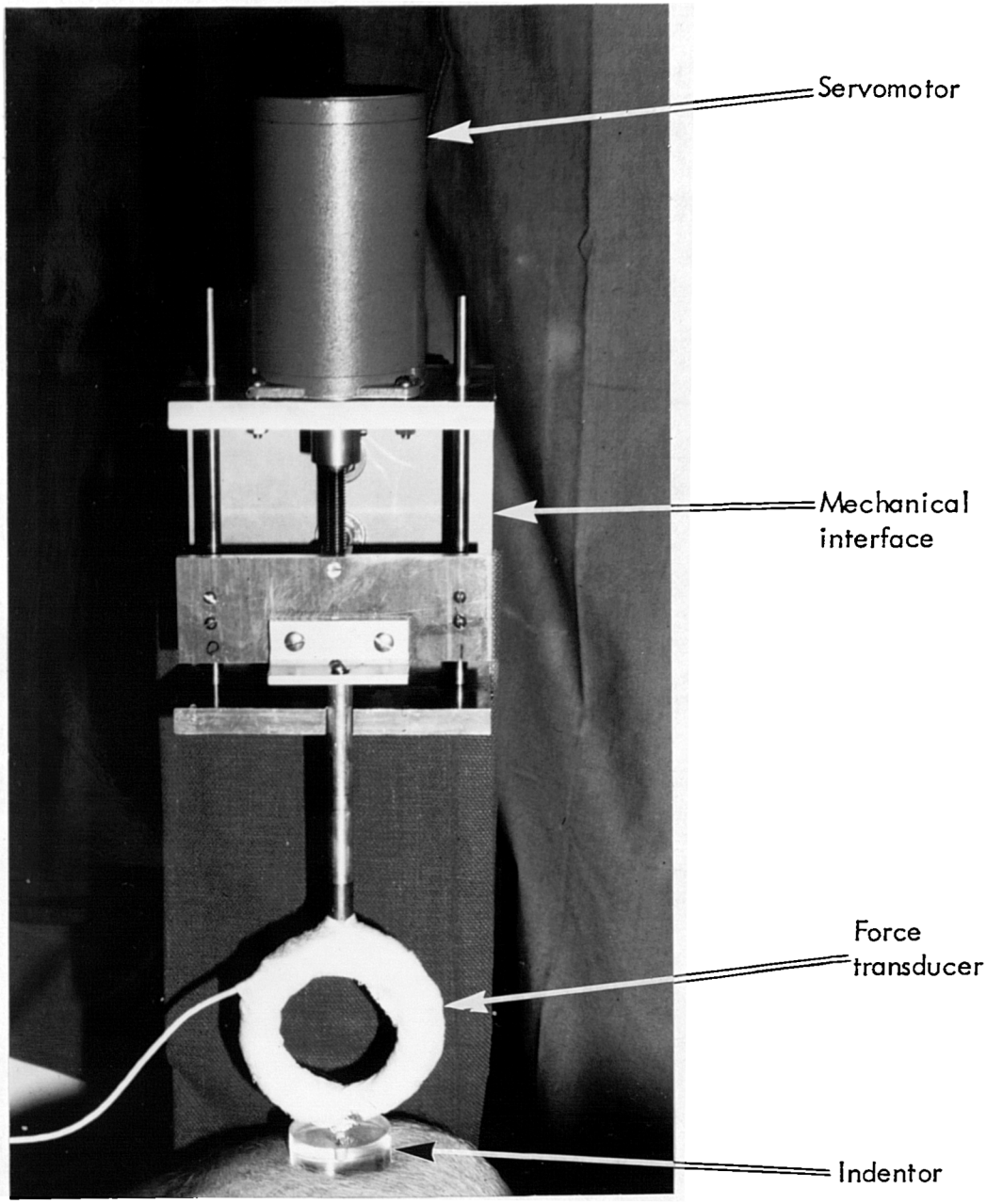


Figure 2.8 The pressure applicator in use.
(after Daniel et al, 1981)

force transducer, in the form of a proving ring, was mounted on the shaft between the mechanical interface and the indentor (figure 2.9). This served to measure the force generated by the motor, leading to a pressure being exerted by the indentor upon the animal. The stepping motor and force transducer were both connected to a microcomputer (Cromenco Z1, Cromenco Inc., California, U.S.A), through suitable electronic interfacing. The system made use of force feedback in that as the motor rotated, the force transducer was providing a measure of the force being applied to the animal. When this reached the desired "target pressure" for that experiment, then the computer halted the stepping motor. The computer was programmed to adjust the stepping motor to maintain the preselected "target pressure". It compared the output from the force transducer to the preselected value. If the discrepancy was larger than 0.7kPa (5mmHg) the computer instructed the stepping motor to rotate until the measured pressure was within 0.7kPa (5mmHg) of the desired pressure. The computer recorded the applied pressure at regular intervals for the duration of the experiment. In addition, the date, duration, "target pressure", time and the data of the experiment were recorded and stored on floppy disk for analysis.

The system was capable of applying pressures up to 160kPa (1200mmHg) under a 50mm disk, for durations in excess of 24 hours.

2.4.3 Experimental protocol

The study made use of Poland-China pigs weighing 13kg $\pm$ 1kg. The animal was starved for 24 hours prior to the start of the experiment, to facilitate anaesthesia. At the time of the experiment, the pig was sedated, intubated and anaesthetised (figure2.10). Gas anaesthesia was utilised, with a mixture of approximately 50% N_2O, 50% O_2, and 0.5%-1% Halothane (Daniel et al, 1981). To prevent dehydration, due to the length of the experiment, fluids were regularly administered, by means of a catheter inserted in the jugular vein. The animal was placed in a lateral position and the indentor of the pressure applicator was positioned above the greater femoral trochanter. Details pertaining to the desired pressure and the duration of its application,

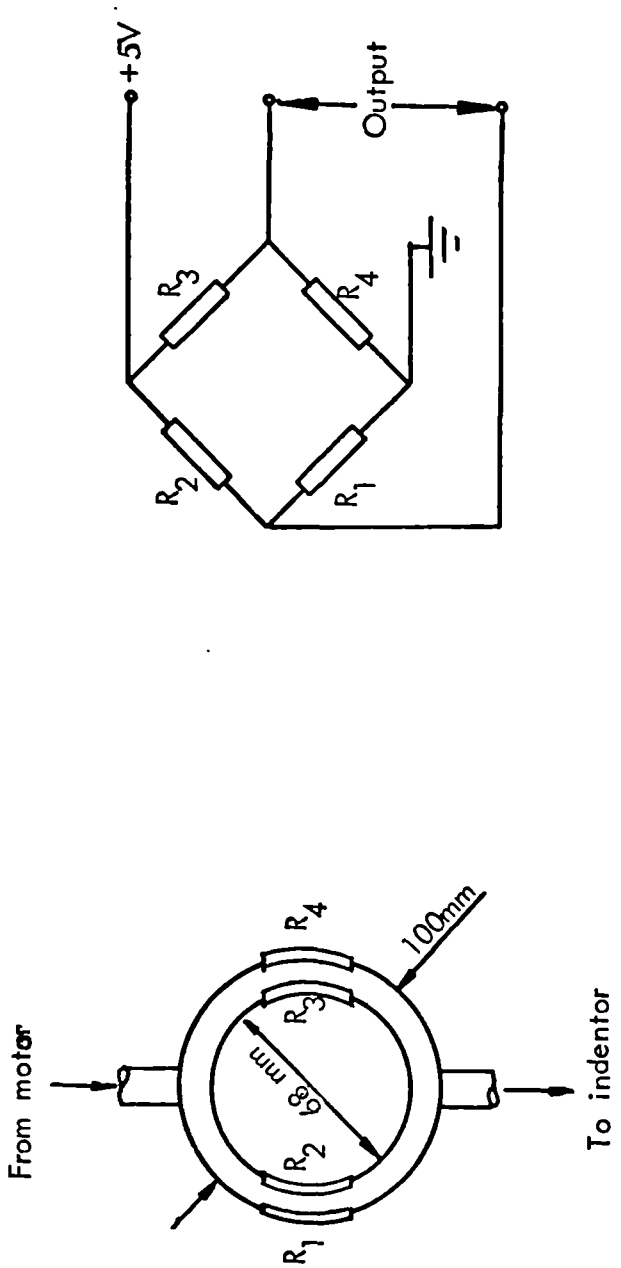


Figure 2.9 Schematic representation of a proving ring, including the strain gauges and their orientation in the Wheatstone bridge.

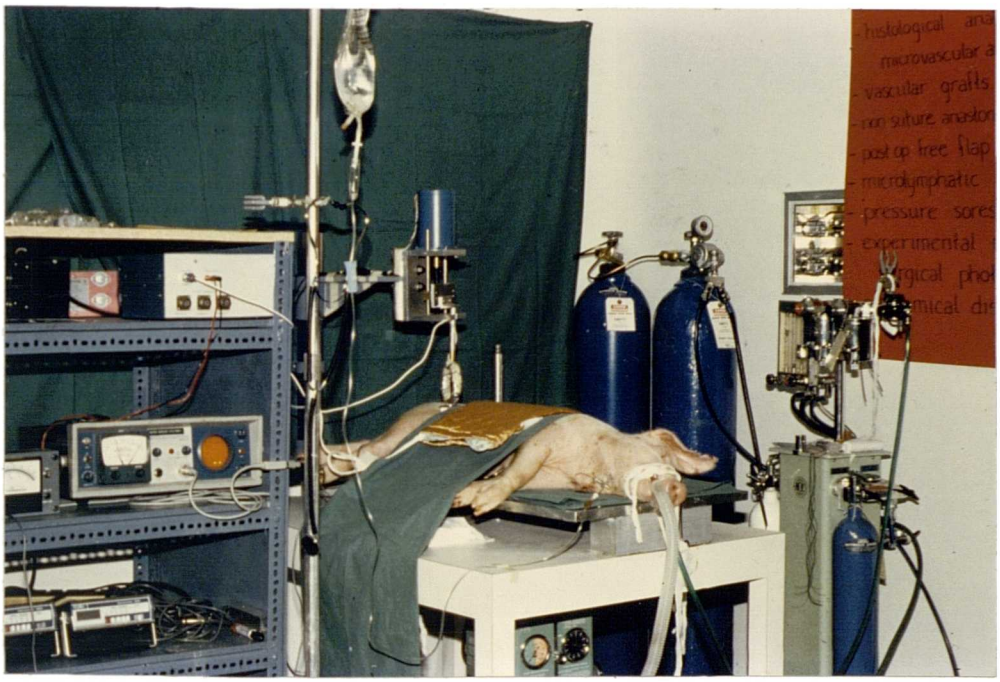


Figure 2.10 An experiment in progress, with pressure being applied to the tissue overlying the trochanter of a pig.

were specified to the computer, and the pressure was applied to the animal. Throughout the experiment, the computer continuously monitored the applied pressure and recorded its magnitude at five minute intervals.

Young pigs (approximately four weeks old) were made paraplegic by transection of their spinal cord, and allowed to grow to a size comparable with that of the normals. At this stage, pressure was applied to the tissues overlying the greater femoral trochanter, in the same manner as was used for the normals.

At the end of the period of pressure application, the pressure was removed, a photograph taken of the pressure site, and the pig returned to its pen. The animal was inspected daily, noting particularly the colour and condition of the skin overlying the greater trochanter. At sacrifice, seven days after the removal of pressure, an incision was made through the skin and muscle of the pressure site down to the bone (greater trochanter). The extent of any tissue damage was visually assessed. A full thickness biopsy, including both normal and affected tissue was taken for histological analysis.

2.4.4 Results

Some of the principal findings of this investigation are summarised in the following subsection.

- There is an inverse relationship between the pressure and duration required to produce pressure sores (figure 2.11). This finding is in agreement with those of Groth (1942), Husain (1953) and Kosiak (1959). However, the duration required to produce an equivalent pressure sore was significantly greater than was previously accepted. This disparity may be explained in part, by the use of different experimental animals. As observed previously (vide 2.4.1) loose skin mammals possess an external tissue coverage, markedly different to that of the pig. In particular, the lack of a subcutaneous fatty layer of tissue reduces the "padding". Thus under an external load, the soft tissues of the loose skin animals do not distribute the pressure as well as those of the pig, rendering them more susceptible to pressure induced damage. In addition, each of the studies utilised differently shaped indentors and it is

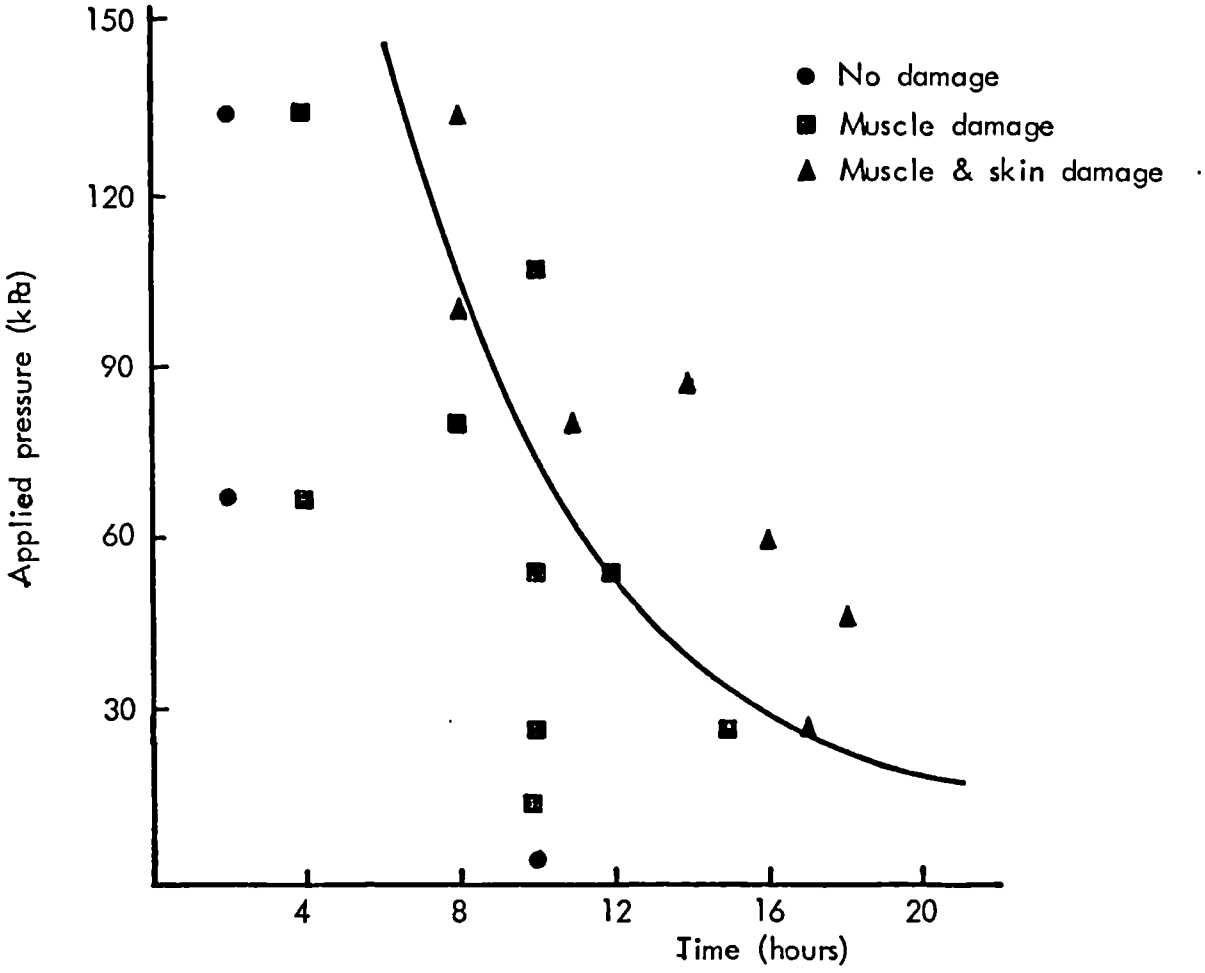


Figure 2.11 Summary of pressure-duration experiments on normal pigs. (after Daniel et al, 1981)

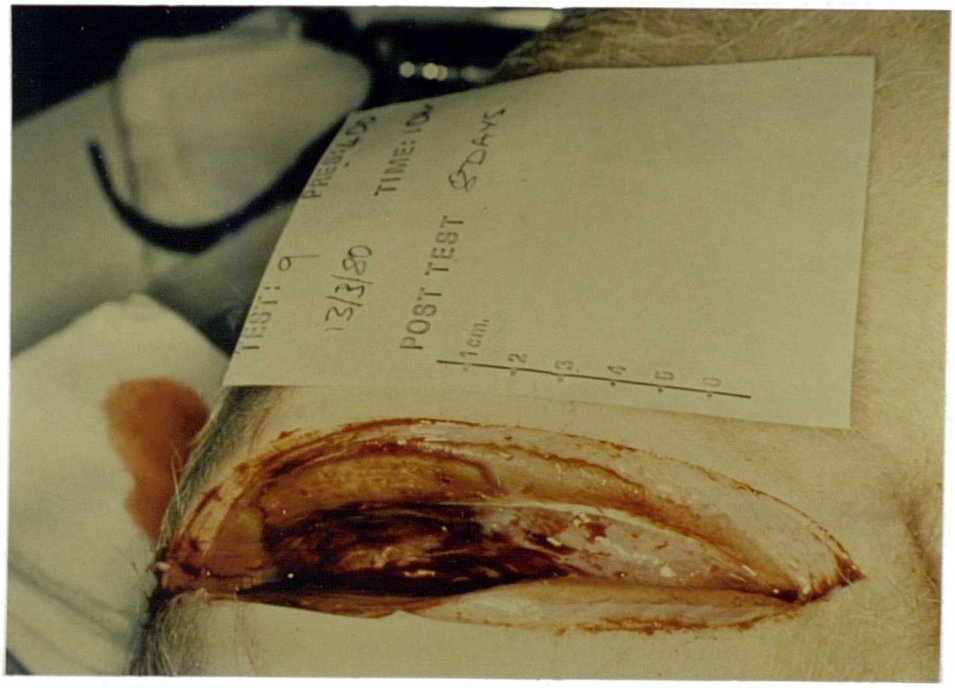


Figure 2.12 A pressure induced lesion, with damage to the muscle overlying the trochanter of a pig.

reasonable to assume that the geometry of the indentor will play an important part in determining the characteristics of the stress field in the tissue under compression. Thus it may be possible to attribute some of the differences between the studies to variations in experimental technique.

Although it is unwise to extrapolate in a quantitative manner, the results obtained from experiments on animals, to man, it might be assumed that animals such as pigs with a similar external tissue structure will provide results which bear the most relevance to man.

- The lesions that formed as a result of the applied pressure were of varying severity, depending upon the magnitude of the pressure and its duration of application. Three pathological groups categorised any resulting lesion.

Firstly, those confined to the muscle, which occurred at pressures as low as 13.3kPa (100mmHg) (for 10 hours) and for durations of four hours (at 133kPa(1000mmHg)). Lesions of this nature ranged from isolated areas of damage to a total necrosis of all the muscle between the skin and the bony prominence (figure 2.12).

Secondly, those relating to muscle and deep dermis damage, which usually consisted of damage to a significant area of the muscle and the lower part of the dermis, with no involvement of the epidermis.

Thirdly, full thickness injury, including damage to muscle, dermis and epidermis. Lesions of this type occurred as a result of high pressure combined with low durations, or of low pressures and long durations (figure 2.13).

- Histological analysis provided further evidence of damage to the external tissues by pressure. The use of a set of critical criteria (Appendix 4) allowed quantitative histological assessment of the damage to both the skin and the muscle. Typical changes in the muscle were, loss of striations, infiltration (giant cells and white blood cells), vessel thrombosis and necrosis. Skin changes included blistering and separation of layers in the epidermis; oedema, haemorrhage and thrombosis of the arterioles and veins; ulceration and loss of the epithelium; and necrosis. There was a close correspondence between the

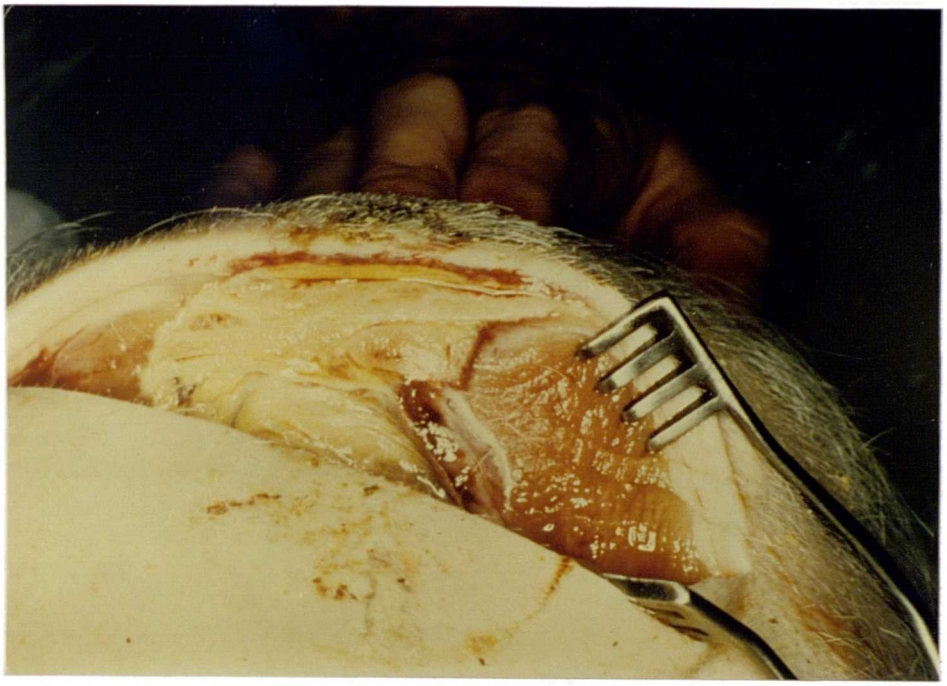


Figure 2.13 A deep pressure sore with damage to all layers of the soft tissue.

(after Daniel et al, 1980)

histological evaluation and the visual assessment of each test.

- Initial pathological changes occurred in the muscle and progressed upwards towards the skin. A cross-section of the tissue from skin down to bone, through a pressure sore, developed using this apparatus, generally showed an inverse cone of damage. That is, a broad area of damage in the muscle overlying the bone, tapering up through the muscle, subcutaneous tissue, to the surface of the skin. This observation is in agreement with Dinsdale (1970), but contrary to the findings of Kosiak (1959), who maintained that degeneration of the different tissues, takes place simultaneously throughout all levels, including the skin. However, the use of a different experimental animal by Kosiak (1959) makes a direct comparison difficult and the reduced soft tissue coverage of dogs, could be expected to affect the internal stress distributions, resulting from the applied pressure.

- The paraplegic group of animals were significantly more sensitive to applied pressure than were the normals (figure 2.14). This result is contrary to those of Groth (1942), Husain (1953), Kosiak (1959) and Dinsdale (1970), who all reported spinal transection as having no effect on the susceptibility of the tissue to pressure sores. However, these authors applied pressure to the animal soon after transection of the spinal cord. Conversely in this study, there was a period of approximately six weeks between cord transection and pressure application. By this stage marked wasting of all the soft tissues was apparent, reducing the coverage over the bony prominence from approximately 30 mm in normal animals to 10-15 mm in the paraplegic animals. This reduction in soft tissue probably accounts for the increased susceptibility of the paraplegic animals to pressure sores.

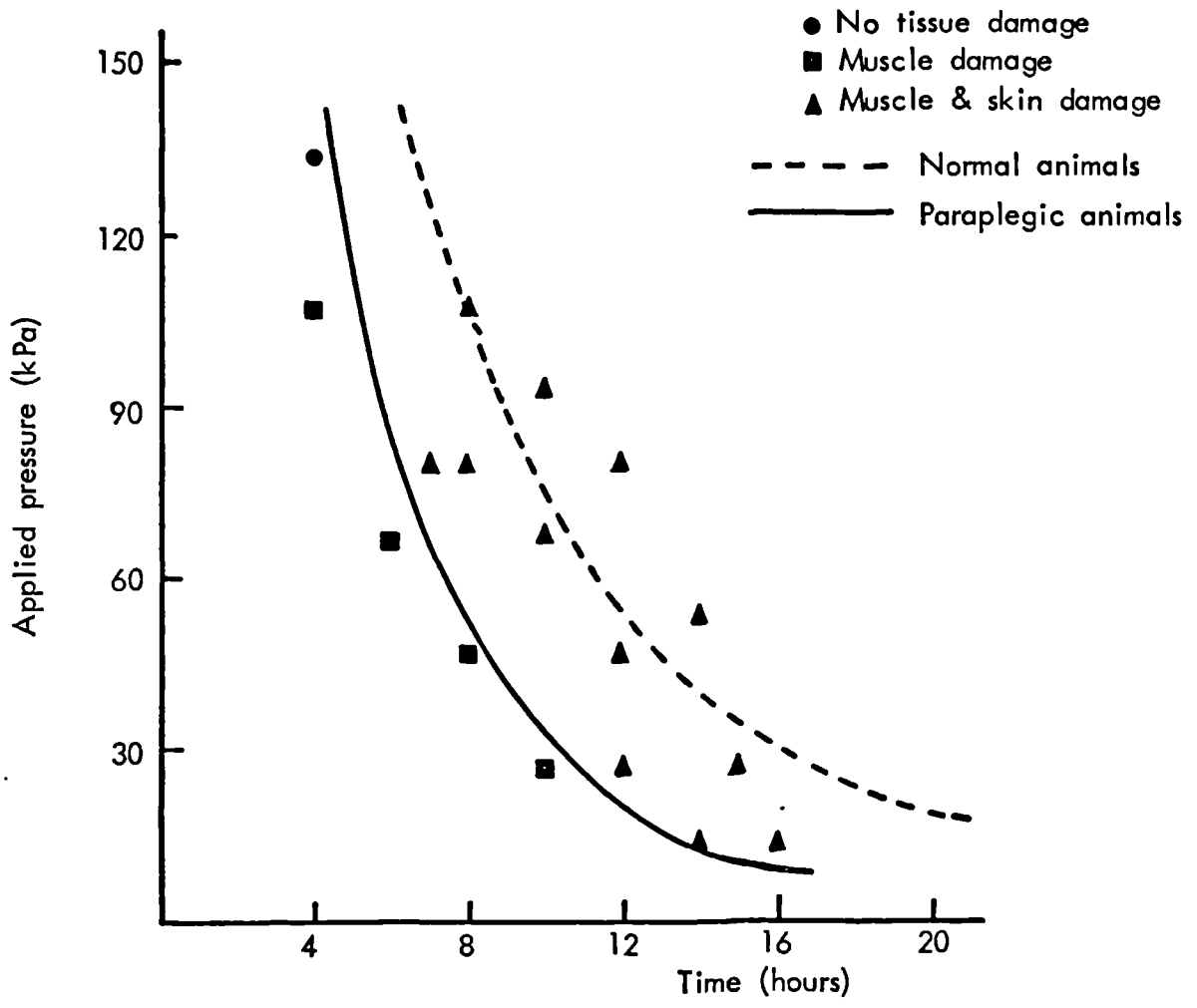


Figure 2.14 Summary of pressure-duration experiments on paraplegic pigs. Demonstrating an increased susceptibility towards pressure sores, relative to normal animals. (after Daniel et al, 1981) Data points relate to paraplegic animals. Dotted line indicates results from normal animals - data presented in figure 5.12.

2.5 SUMMARY

The aetiological factors commonly thought to be responsible for the formation of pressure sores were considered in two groups - primary and secondary.

Primary factors serve as the principal variables involved in the formation of pressure sores. Those sores that start in the deeper tissues (deep) have been attributed to the combination of two primary factors - deformation of the tissue, caused by the transmission of body weight to the support surface, acting both normally and tangentially, and immobility. It is usually considered that the aetiological mechanism responsible for deep pressure sores is one of compression of the soft tissues underlying a bony prominence, leading to ischaemia. If this is not relieved then the tissue becomes anoxic and dies. Alternatively, it has been suggested that repeated deformation of the tissue, retards the healing mechanism, leading to a progressive damage of micro-vasculature. Those sores that start superficially (superficial) are usually related to primary factors of friction and moisture. Friction, acts to abrade the surface of the skin, removing the protective layer of the epidermis to expose the more vulnerable dermis. Moisture increases the frictional coefficient between the skin and the support surface while also softening the skin itself.

Secondary factors were considered to be those which predispose to pressure sores. These include support surface, which is important in determining the interface forces, drugs, which may influence other factors, such as mobility, incontinence, neurosensory system, and weight, which has been related to the magnitude of interface forces. Infection is important in the development of deep pressure sores, as bacteria tend to congregate around the site of tissue deformation and in superficial sores, as it can hasten the deterioration of a sore and complicate treatment.

The most common sites of pressure sores were determined to be the sacrum, trochanters, ischial tuberosities and heels, with the greatest number occurring in the sacral region. Elderly patients were particularly susceptible to pressure sores and, in addition, the diseases most commonly linked with pressure sores were those commonly suffered by the elderly, such as arterio-

sclerosis and peripheral vascular disease. Reduced mobility was identified as being related to an increase in the number of pressure sores, this was particularly evident in the elderly age group.

A number of studies were described which had been performed to establish the relationship between applied pressure and duration in the aetiology of pressure sores. Each of these discussed an inverse relationship between magnitude of pressure and the duration of its application, necessary to develop a sore. Further results indicated the importance of infection. One of the studies, Daniel et al (1981), contested the previously accepted values of pressure and duration needed to initiate a sore, suggesting that increased magnitudes were required. The disparity between results was explained as differences in the experimental animal. However, the similarity between the soft tissue covering of man and that of pigs, suggested that the results of Daniel et al (1981) would compare most closely with the human situation.

The animal studies involving a single application of pressure, provided useful information on the characteristics of soft tissue under compression, and demonstrated the feasibility of producing pressure sores in the controlled environment of the laboratory. However, the application of a single episode of pressure is unrepresentative of the aetiology associated with some pressure sores, which occur as the result of repeated traumas and it is apparent that further research utilising the experimental animal should be conducted in this area.

The importance of prolonged mechanically induced ischaemia, as illustrated by the experimental studies and the particular susceptibility of the elderly age group, indicated by the prevalence surveys, suggested that future research should attempt to combine these factors. That is, an experimental study assessing the interface pressures between the body and support surface, their changes with time related to mobility, and in particular should concentrate on the elderly patient.

CHAPTER 3

MOVEMENT DURING SLEEP

3.1 INTRODUCTION

Rather than simply being an interruption of the waking state, sleep influences some of the most important body functions in man. The study of sleep has been of interest to researchers of many disciplines and has encompassed the relationship between sleep and physical, psychological and physiological factors in both the healthy and unwell subject. Comprehensive reviews of the literature relating to sleep have been undertaken by Kleitman (1963) and Freemon (1972).

In the first half of this century, body movements, their duration, frequency and distribution, were often used as a means of characterising sleep. They provided a simple, reliable and easily measurable variable for studies of the effect of drugs, psychiatric disturbances, age, etc. on sleep. However, with the advent of the electroencephalogram (EEG), interest has been directed towards assessing sleep by electrophysiological means.

An EEG serves to measure small electrical potentials produced by the brain. Detection of these potentials is achieved by the use of electrodes attached to the scalp of the subject. The signal is amplified and recorded on strip chart paper for analysis. A detailed account of the EEG, including information regarding amplification, type and placement of electrodes and possible artefacts, is given by Greenfield and Sternback (1972).

3.2 SLEEP MEASUREMENT USING THE EEG

Berger (1930), utilising Ag-AgCl electrodes inserted epidurally, describes one of the first studies to make quantitative assessment of EEG recordings. He demonstrated that the brain produced measurable changes in electric potential. Particular patterns were isolated, and named, the most common of which were termed alpha and delta. Alpha waves are synonymous with wakefulness and typically have a frequency of 8 - 13 Hz, with a magnitude of 30 - 50 μV. Delta waves are closely associated with deep sleep having a low frequency of 0.5 - 3.5 Hz and a high amplitude of 200 - 300 μV.

In 1955 an important addition was made to the study of sleep when Aserinsky and Kleitman recorded the presence of rapid eye movements, lasting for approximately ten minute periods and occurring approximately every 90 minutes throughout the night. At the same time as the eye movements, the EEG record showed increased arousal, although the subject remained asleep. After questioning the subjects by awakening them, in a period of rapid eye movement, it was shown that in such periods, dreaming occurred. These results led to the concept of two different "classes" of sleep, termed REM (rapid eye movement) and NREM (no rapid eye movement).

3.2.1 Characterisation of EEG sleep records

A number of different assessment techniques of EEG records have been suggested in the past 50 years. One of the earliest of these was by Davis et al (1938) who recognised five stages of sleep which were designated by the initial five letters of the alphabet. A, (interrupted alpha pattern) observed when the subject was awake. B, (low voltage pattern) the regular alpha rhythm was replaced by a signal characterised by small undulations, observed at the onset of sleep. C, (spindle pattern) a characteristic waveform, typically 12 - 15 Hz, superimposed upon an irregular pattern of slow waves. D, (spindle plus random pattern) similar to C with the addition of delta waves and E, (random pattern) similar to D but without any spindles. Additionally, the letter K was used to represent a characteristic waveform (diphasic) of about one second duration, which was apparent in stages B and C. K complexes appear as a result of the

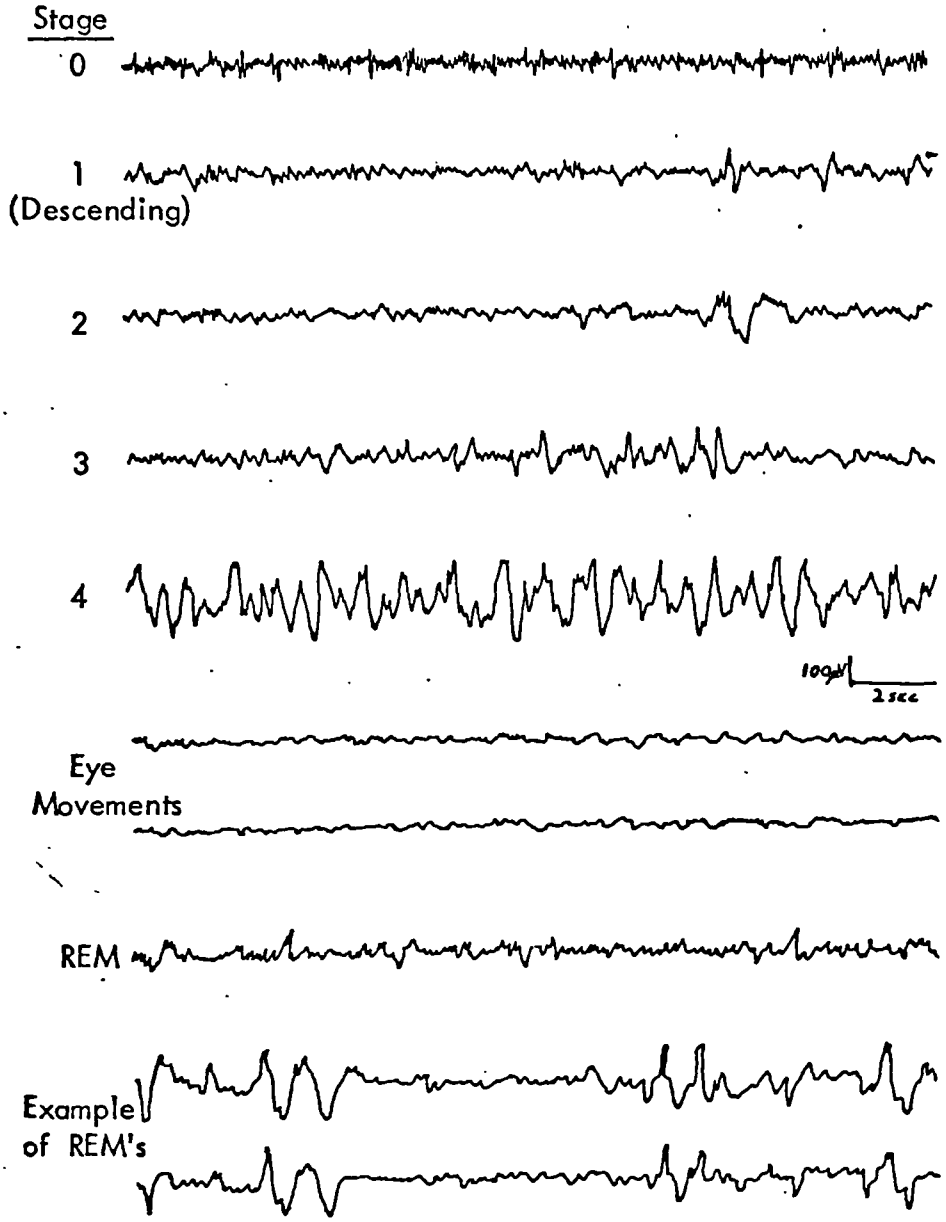


Figure 3.1 EEG waveforms associated with the scoring system developed by Dement and Kleitman (1957).

activation of any of the body's sense modalities (Roth et al, 1956). Depth of sleep was considered to increase from wakefulness A, to deep sleep E. Such a system proved to be applicable to the majority of EEG recordings, although the complexity and quantity of the data meant that a number of errors in classification occurred (10 - 15%) (Kleitman, 1963). For this reason each night's recording was assessed separately by two independent observers and their results compared and combined. This alphabetical system was employed by the majority of sleep researchers until Dement and Kleitman (1957) proposed a simplication, characterising the sleep pattern into four groups, (figure 3.1).

Stage 1 (Modified alpha): A somewhat irregular alpha pattern which occurred at the onset of sleep and may be considered as a combination of A and B (Davis et al, 1938).

Stage 2 (Spindling): This group is synonymous with stage C (Davis et al, 1938).

Stage 3 (Delta with spindling): Typical waveforms in this group contain within 20% to 50% of large amplitude low frequency delta waves.

Stage 4 (Slower delta without spindling): This group contains waveforms exhibiting 50% or more delta waves and is synonymous with stages D and E of Davis et al (1938).

When the existence of REM sleep was recognised, it became apparent that the current classification technique, used for EEG's was unsatisfactory. This resulted in a profusion of different techniques, all designed to include REM sleep. However, the most commonly accepted method of characterisation of EEG sleep records remained that of Dement and Kleitman (1957). To account for the two classes of sleep, the four stages previously described were attributed to NREM sleep and a further stage was added to represent REM sleep (figure 3.1). The continued use of the classification of Dement and Kleitman (1957) was potentially ambiguous and meant that some results, which did not distinguish between REM and NREM sleep were of limited value. For example, Corletto et al (1967) who studied the evoked responses during Stage 1, did not delineate between REM and NREM sleep and as a result their work cannot be used in comparison with other evoked response studies.

3.2.2 Typical sleep cycle - normal subject

The subject is monitored by means of an EEG, electromyograph (EMG) and electro-oculargraph (EOG) with electrodes placed on the scalp, chin and near the eyes respectively. As the subject falls asleep the waking voluntary eye movements, evident from the EOG, are reduced as is the recording from the EMG. The EEG displays a reduction, followed by a disappearance of the alpha rhythm and the signal decreases in frequency while increasing in amplitude. At this point the subject was said to be in Stage 1, NREM sleep, (Dement and Kleitman, 1957). With the appearance of K complexes and 14 Hz spindles, the subject was considered to have moved into Stage 2 of the NREM sleep cycle (Roffwarg et al, 1966). The K complexes may occur following an external stimulus (such as noise) or be the result of "internal autonomic events", for example, bladder contractions (Johnson and Karpan, 1968). Sleep spindles are thought to occur as a result of "thalamocortical interrelations" (Freemon, 1972). Large delta waves proceed to enter the record indicating firstly Stage 3 and then as they increase in number, Stage 4 of the sleep cycle. All four of these stages were considered to be NREM sleep. REM sleep first occurred after approximately 50 - 70 minutes (Roffwarg et al, 1966) or two hours (Freemon, 1972). In the period immediately before this, the EEG record indicated a waveform similar to that of Stage 1 NREM sleep. However, distinction between NREM and REM was provided by the EOG and EMG recordings. The EOG became active due to the eye movements, whereas the EMG was reduced to virtually zero. A normal subject was expected to maintain their first REM sleep period for approximately 10 - 20 minutes, before returning to NREM. Thereafter REM and NREM sleep were expected to alternate for the course of the night, at 80 - 90 minute intervals. The duration of REM sleep periods changed throughout the night, with those in the early part of the night being shorter than those occurring towards the end of the night. Typically there were 4 - 6 periods of REM sleep during the night, leading to a total of a fifth or a quarter of the overall time asleep. The majority of NREM sleep, Stage 4, was accomplished in the period before the first episode of REM sleep. Between periods of REM, NREM Stage 2 sleep predominated with its

characteristic K complexes and spindles.

3.2.3 Limitations of characterisation techniques

One of the major disadvantages of experiments involving the use of an EEG, is the quantity of data produced. For example, the "common slow chart speed" for a sleep experiment is 15 mm/sec (Freemon, 1972). If the subject sleeps for eight hours, the total length of the sleep recording is in excess of 400 metres. Such a record is then divided up into 20 second epochs. Thus, approximately 1500 separate epochs must be classified for each test. Each interval being scored as waking, REM or NREM Stages 1 to 4. In an effort to provide a reproducible system for characterisation, Rechtschaffen and Kales (1968), produced a manual of "standardised terminology", based on the technique of Dement and Kleitman (1957), outlining each of the criteria and providing details of exceptions and borderline cases.

Such a system of analysis is long and tedious and partially subjective, all of which tend to introduce errors. Particular difficulty has been observed in delineating between Stages 2, 3 and 4 of NREM sleep. This problem arises if the percentage of delta waves, present in the record, is close to the maximum or minimum of that defining Stage 3. That is, if approximately 20% of the epoch contains delta waves should it be classified as NREM Stage 2 or Stage 3 ? A similar case is apparent when approximately 50% of the epoch contains delta waves, in choosing between Stages 3 and 4 (Monroe, 1969). For this reason, data analysis is often simplified by combining Stages 3 and 4 together and calling them "slow wave sleep" or SWS.

Numerous attempts have been made to score the sleep records by the use of computer techniques. A review of some of these methods has been provided by Smith (1978). The major advantage of such techniques is the speed of analysis of the records. However, as yet no program has been devised which is capable of attaining perfect agreement between human and automatic detection, although Smith (1978) reports work conducted by Johnson et al (1976), who were able to achieve a 92% correspondence.

3.2.4 Use of the EEG to monitor body movements

It has been demonstrated that it is possible to detect the occurrence of movements during sleep, by the presence of artefacts in the recording of the EEG of a subject (Dement and Kleitman, 1957). This method proves to be sensitive to movements of the head, but unsuitable for providing a measure of gross body movement (Sassin and Johnson, 1968). However, in an effort to provide a technique capable of measuring general body movement Dement and Kleitman (1957) combined the method of EEG artefacts with that of the measurement of the displacement of a bedspring (vide 3.4.1). They claimed to be able to detect all major body movements.

Further examples of the combination of an EEG with other monitoring techniques are those of, Sassin and Johnson (1968), who combined the use of EEG, EOG and EMG; Naitoh et al (1973), who monitored heart rate and finger pulse amplitude in addition to the EEG; and Metz and Muzet (1975), who combined multiple techniques to monitor overnight mobility including those of EEG, EMG, heart rate, finger pulse amplitude and ultrasonic radiation.

The use of an EEG to study sleep requires significant complexity and has generally been limited to use in specifically designed sleep laboratories. Studies of sleep using EEG's are notorious for the quantity of data produced, and analysis is a long tedious process, to be carried out by skilled personnel. For these reasons EEG's tend only to be used specifically for identification of body movements, when it is desired to compare them with other changes in sleep characteristics. For example, changes in sleep stage, characteristic waveforms etc. (vide 3.3).

3.3 STUDIES OF MOVEMENT DURING SLEEP

3.3.1 Movement in the sleep cycle in relation to K complexes

Sassin and Johnson (1968) quantitatively compared body movements during sleep with the occurrence of K complexes. This study was based upon the works of Gastaut and Broughton (1965), who demonstrated an association between K complexes and body movements, and of Brazier and Beecher (1952), who reported arousal like waveforms appearing on the EEG before movements (this was of particular importance when it was considered that K complexes themselves were thought to be related to an arousal stimulus (Roth et al, 1956)). Sassin and Johnson (1968) measured sleep and mobility using an EEG, EMG and EOG and attempted to compare electrophysiological brain waveforms, measured by the EEG, in particular the K complex, with changes in the EOG and EMG. Sleep was characterised by means of the standardised scoring system of Rechtschaffen and Kales (1968). Stages 3 and 4 were combined and termed SWS (slow wave sleep), K complexes were identified in Stage 2 only, due to the difficulty of their discrimination in SWS. Movements were found to be significantly associated with spontaneously occurring K complexes, although no measure of the significance was reported. A move was generally followed by a K complex. Although there were no alpha waves observed before a move (cf. Brazier and Beecher, 1952), they were sometimes evident following a move. Gastaut and Broughton (1965) and Sassin and Johnson (1968) were of the opinion that K complexes occurring during SWS were also related to the occurrence of body movements, although they could not produce conclusive evidence of this, because of the difficulty of identification of K complexes in SWS.

3.3.2 Body movements and their relationship to the sleep cycle

Position: Sassin and Johnson (1968), proposed that the majority of moves in SWS occur at the end of the period and were indicative of an imminent change of stage. Moves in Stage 2 being grouped together at the beginning of the period, particularly after a change from SWS. Stage 1 and REM sleep having a relatively even distribution of moves throughout the period

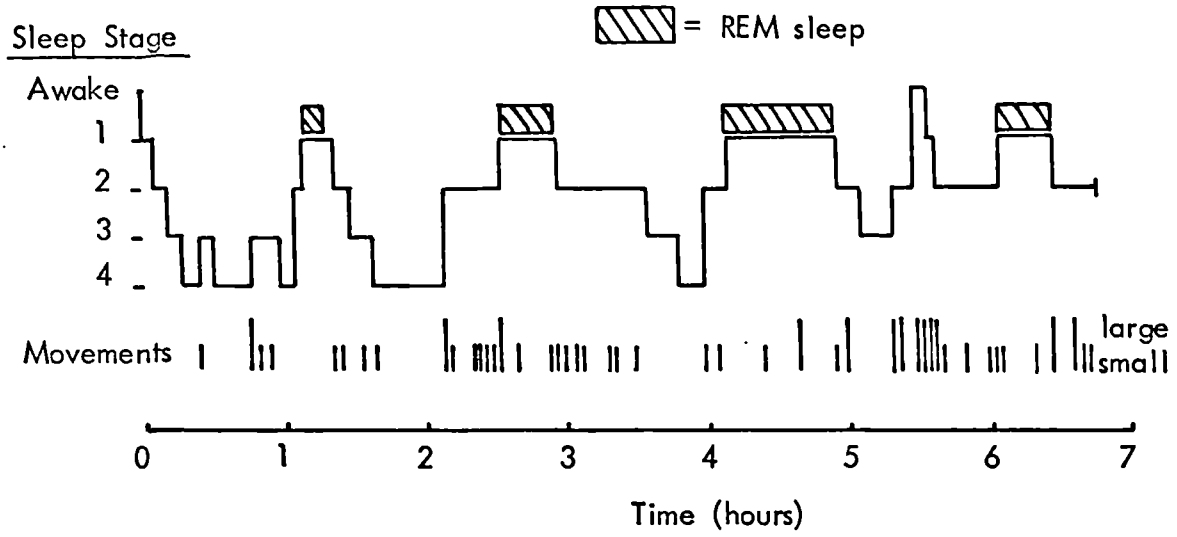


Figure 3.2 Occurence of body movements during the sleep cycle.
(after Greenfield and Sternbach, 1972)

(figure 3.2). In addition Muzet et al (1972) have observed that, before entering SWS, there is an absence of body movements, whereas prior to entering REM sleep body movements may occur often.

Body movements occurring in REM sleep have been associated with dreaming. Dement and Wolpert (1958) provided evidence that the presence of a body movement in REM sleep indicated the end of a dream sequence. Subjects in REM sleep awakened after a body movement were found to have fragmented dream episodes. However, it was not possible to associate the dream content with any movements made during this period.

Size of movements: Sassin and Johnson (1968) divided the measured body movements into three groups relative to their magnitude. Type A, (small) movements of the head, face or mouth only, Type B, (medium) movements of an extremity only, and Type C,(large) movements involving the head, extremities and trunk simultaneously. Sleep Stages 1 and 2 were found to contain approximately equal numbers of Type C and Type A movements with no Type B size moves. The majority of movements in SWS sleep (80%) were Type C moves.

Studies on REM sleep have observed a supression of muscle tone during this stage, the only movements being twitches of the face and extremities (Berger, 1961 ; Jacobson et al, 1964). Moves of this nature were classified as Type B and provided the only significant number (10%) of medium size movements recorded.

Duration and frequency of moves: Naitoh et al (1973) reported rates of body movement of 3.78 (movement/hour), 9.3 (movement/hour), 12.18 (movement/hour) and 9.6 (movement/hour) for SWS, Stage 2, REM and the total sleep period respectively. With a statistically significant difference in the rate of movement between SWS and all the other sleep stages and between REM and Stage 2 sleep. These results are in agreement with those of Dement and Kleitman (1957) and Kamiya (1961), but are in disagreement with Monroe (1967) and Sassin and Johnson (1968), who obtained no significant difference between REM and Stage 2 sleep. This discrepancy may be attributed to differences in the subjects and the experimental method used for detecting the movements employed

by each of the authors.

The duration of the movements were assessed by Sassin and Johnson (1968) who determined that the "large" body movements (Type C) were significantly longer (mean 16.6 seconds), accounting for the long average duration of moves in SWS (17.4 seconds). The most rapid movements being Type B, which occurred during REM (2.65 seconds). Comparison was made between the three different groups and their association with a change in sleep stage: 0% Type B, 5% Type A and 27% Type C movements being related to a change in sleep stage. These Type C movements were found to be significantly longer than those unrelated to a stage change ($p < 0.001$).

The distribution of movements throughout the night was closely associated with the distribution of sleep stages. Section 3.2.2. described a typical night's sleep, consisting of an initial period (1 - 2 hours) of SWS, changing to REM or NREM Stage 2, which then proceed to alternate for the remainder of the night, with occasional short duration return to SWS. Suckling et al (1957), Sassin and Johnson (1968), Muzet et al (1972) all observed a smaller number of moves occurring at the beginning of the night, during SWS. However, the number of body movements increases later in the night during REM, Stage 1 and Stage 2 sleep. Studies by Naitoh et al (1977) observed an increase in SWS on the first recovery night after a period of sleep loss. As might be expected, on the basis of the results of Suckling et al (1957) and Sassin and Johnson (1968), there was a concomitant reduction in the overall number of body movements.

3.3.3 The effect of endogenous factors on movements during sleep

Sleep, both its duration and quality have long been associated with the action of a number of endogenous physiological variables.

Age: Much research has been conducted into the effect of age on sleep. A far smaller proportion has concentrated on the relationship between age and body movements occurring during sleep, and of these studies, the emphasis has been on neonates and young children.

Roffwarg et al (1966) determined a higher proportion of REM sleep in

young children, largest when the child was newborn and decreasing as they grew older. This was thought to be directly related to the length of time the child spent awake, the newborn children, sleeping for protracted sessions with brief periods of awakening. Later, as the growing child achieved longer periods of wakefulness, the amount of REM diminished. The significant increase in the rate of movement during REM sleep (vide 3.3.2) and the occurence of long periods of REM in children, helps to explain an observation by Garvey (1929), that the overall rate of movement of children is greater than that of adults.

Those studies that have involved elderly subjects have concentrated on changes in the proportions of sleep stage, with increasing age and disability (Agnew et al, 1967; Kales et al,1967; Kupfer et al, 1978). Thus, any conclusions about the effect of age on bodily movement must be inferred from this data using the already derived relationship between sleep stage and movement (vide 3.3.2).

Agnew et al (1967), conducted a study into the characteristics of sleep in a group of 50 - 60 year olds. They found a marked reduction in Stage 4 sleep with a small increase in Stage 1. There was an overall decrease in the total time spent sleeping. On the basis of the work of Sassin and Johnson (1968) it is known that body movements occur more frequently in Stage 1 than in Stage 4 (vide 3.3.2). Thus, it may be postulated that increased age implies an increased mobility. However, as noted by Bardsley (1977), it is apparent that the relationship between increasing age and body movements during sleep is not well defined and requires further research.

Heart rate: It is well established that heart rate changes during the course of sleep (Kleitman, 1963). These variations may be related to changes from NREM to REM sleep, with a small increase in heart rate during REM sleep (3% - 10%), (Aserinsky and Kleitman, 1953). In addition to variations due to changes in sleep stage, it has been shown that body movements also affect the heart rate. This manifests itself in the form of an anticipatory cardiac acceleration. According to Jackson (1941) and Brazier and Beecher (1952), the acceleration begins about six minutes before a body movement, increasing

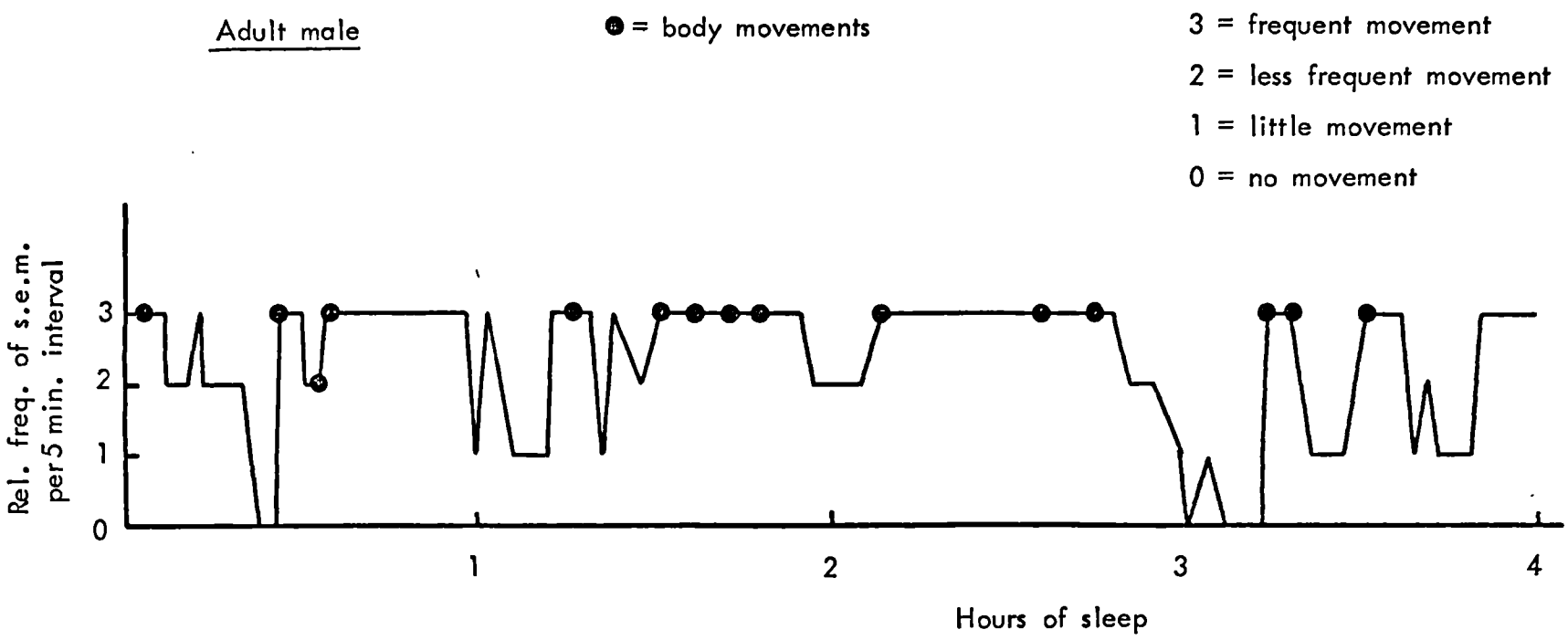


Figure 3.3 Distribution of slow eye movements (s.e.m.) and body movements during sleep.
(after Aserinsky and Kleitman, 1955)

rapidly up to the time of the movement. Townsend et al (1975) noted an increase in heart rate approximately eight seconds before a move occurring in Stage 2 or REM sleep. Schieber et al (1971), measured an increase in cardiac acceleration in a period several seconds before a movement, and a reduction in heart rate following the movement. In their study this characteristic was observed during all of the sleep stages.

Slow eye movements: Lawson (1950), employing a method of direct observation, noted the presence of slow, drifting, pendular movements of the eye of a resting subject. As the subject fell asleep the movements stopped. Andreev and Ivanov (1950) confirmed the presence of slow eye movements and considered them to be related to the onset and awakening of sleep. These results were similar to those from earlier studies of Miles (1929) and de Toni (1933). Aserinsky and Kleitman (1955) attributed slow eye movements not only to the start and end of sleep, but to body movements occurring during sleep (figure 3.3).

It is possible that slow eye movements occur at a low level of arousal during sleep. Such a level would be expected at the beginning and end of sleep and the results of Brazier and Beecher (1952) indicated that the depth of sleep decreases prior to and immediately following a body movement.

Respiration: It has been demonstrated that the overall respiration rate falls continuously for the period of sleep (Schaff et al, 1962; Phillipson, 1978). Bülow and Ingvaar (1963) correlated decreased ventilation with EEG tracings of sleep. They obtained a close correlation for even transient periods of sleep. Roffwarg et al (1966) measured the rate of respiration during the sleep cycle and noted a difference between NREM and REM sleep. On entering REM sleep the respiration rate increased by approximately 18%. However, the breathing became irregular, leading to a significantly greater minute to minute variation in REM relative to NREM sleep. It was also observed that on return to NREM, the respiration rate was lower than the previous period of NREM sleep, leading to a reduction in rate over the total night's sleep.

Østergaard (1944), measured an increase in the level of alveolar CO_2

during sleep, with a corresponding decrease in oxygen consumption. Krausse (1935) noted an inverse relationship between the percentage of CO_2 alveolar air and the frequency of movement during sleep.

Blood pressure: Snyder et al (1963) measured changes in blood pressure and changes in sleep stage. They described a rapid decrease in systolic blood pressure in the period immediately before onset of sleep, continuing for approximately 1 - 2 hours, after which there was a gradual, but irregular increase for the rest of the night. A period of REM sleep was found to be coincident with a rise in blood pressure (up to 4kPa) greater than the NREM level. Gross body movements also served to cause transient increases in systolic blood pressure, which then returned to its previous level at the end of the movement. Naitoh et al (1973) and Muzet et al (1974), demonstrated a decrease in finger pulse amplitude, implying vasoconstriction, and a correspondence with the findings of Snyder et al (1963).

Gastric activity: Wada (1922) conducted studies on a number of young adult normal subjects, monitoring muscular contractions of their stomach, and measuring their body movements during sleep. She observed that gastric activity exhibited a regular periodicity, consisting of an alternation between a quiescent stage, lasting 30 - 45 minutes, and a stage during which there is an increasing tonus of the stomach muscles, lasting for 1 - 1½ hours. It was discovered that body movements took place simultaneously with contractions of the stomach, and further, that the majority of the movements occurred at the point of maximum stomach activity. McGlade (1942) measuring body movements, observed a number of fast movements occurring after about three hours in 12% of his subjects. These moves took place only when particular foods had been eaten and they were found to be synchronous with the opening of the pyloric sphincter.

Urinary bladder pressure : A study by Laird (1935) showed a relationship between the occurrence of body movements and the urinary bladder pressure of the subject. It was discovered that on occasions where the subject had an increased urinary output, there was a corresponding increase in the number of body movements. The movements took place in the period previous to a

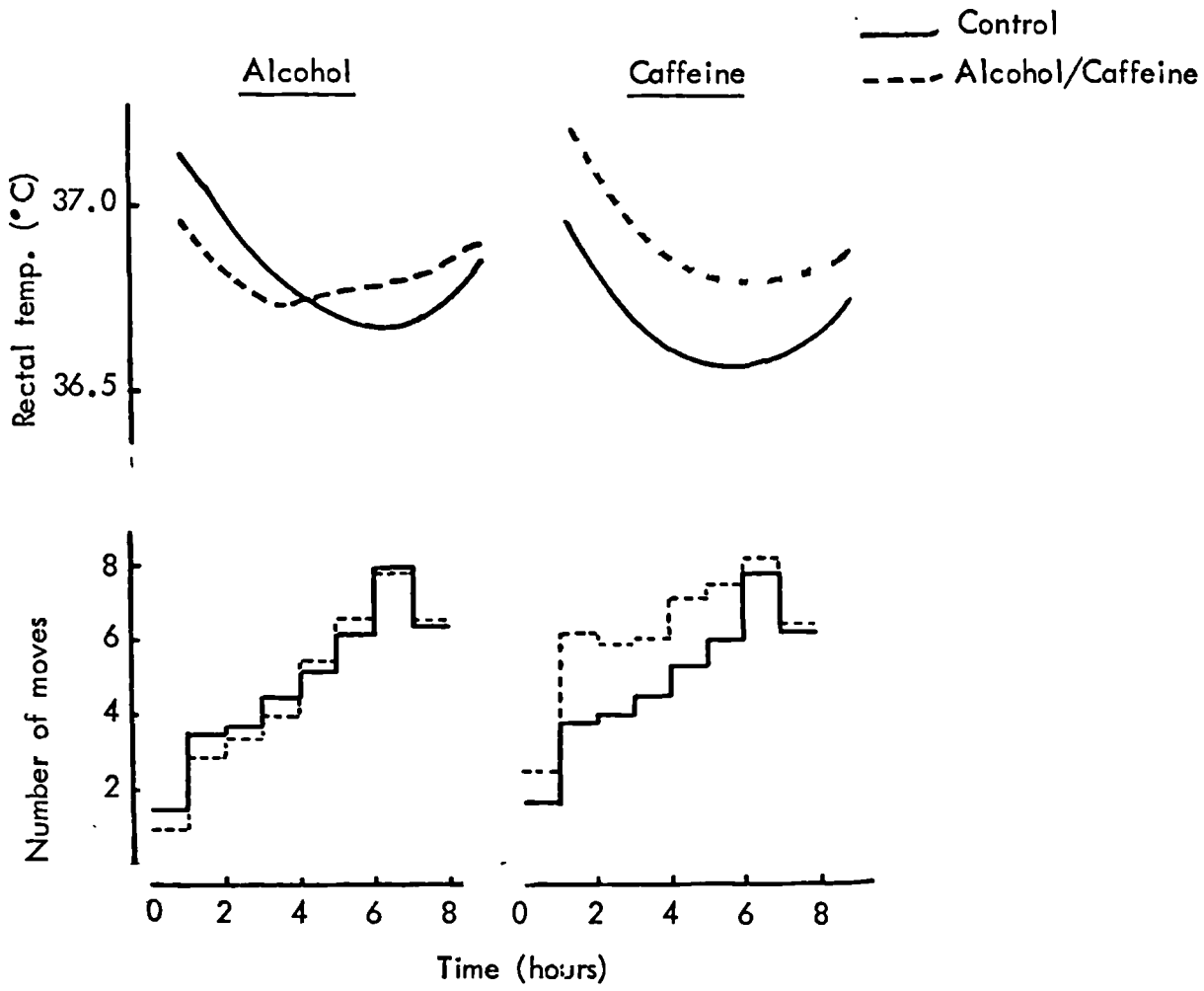


Figure 3.4 Effect of alcohol and caffeine on overnight mobility and rectal temperature.

(after Kleitman, 1963)

measured high bladder pressure.

Body temperature: No direct relationship between changes in body temperature during sleep and changes in motility for the normal subject have been observed. Karger (1925) and Kleitman (1963) noted that the motility of their subjects (children) was unchanged even at high body temperature (fever).

Body temperature may be significantly increased following changes in metabolic activity associated with the consumption of stimulants such as alcohol or caffeine (vide 3.3.4). Measurements of rectal temperature and body movement following the consumption of alcohol and caffeine,show corresponding changes in core temperature and motility as these substances are metabolised (Kleitman, 1963) (figure 3.4).

Hellmuth and de Veer (1936) compared the difference in interface temperature between the skin and the bed with motility. In the normal situation, the skin temperature was 4 - 5°C higher than that of the bed and motility was unaffected. However, if the difference was reduced to approximately 1°C there was a corresponding increase in motility.

3.3.4 The effect of exogenous factors on movement during sleep

Drugs: It has been proposed that a number of drugs, in particular sedatives, such as barbiturates("hypnotics")serve to provide an increase in the sleep duration, while reducing the spontaneous movement of the subject (Brazier and Beecher, 1952; Cox and Marley, 1959; Hinton, 1961; Exton-Smith, 1967; Brocklehurst et al, 1974; Oswald, 1974). This observation will be of particular importance in the case of a patient already suffering from predisposing factors to pressure sores (vide 2.1.3), such as old age, incontinence, reduced blood pressure, deficiency in nutrients etc., who, if prescribed a barbiturate sleeping drug, will exhibit a reduction in motility, further increasing the likelihood of the development of a pressure sore.

Meyers et al (1940) reported that the use of a variety of hypnotic drugs produced no change in sleep motility, except in the case of congestive heart failure, where motility was actually increased.

The magnitude of reduction in motility has been identified as being a

function of the type and amount of the drug given to the patient, in association with other variables influencing the effect of the drug, such as body weight (Hinton, 1961).

Oral consumption: Studies have been conducted on the effects of different fluids taken immediately before retiring, and movements occuring during sleep.

Southwell et al (1972) carried out a study on four male students, subjecting each of them to, a drink of water, warm milk containing Horlicks or no drink at all (control), in the period immediately before retiring to bed. They discovered that the water and the Horlicks had no noticeable effect on the movements during sleep, except that the Horlicks resulted in a reduction in the rate of small movements occuring during the night. Březinová and Oswald (1972) also conducted work on the effect of Horlicks taken immediately before bedtime. They determined a reduction in the number of body movements towards the end of the night, in young adult subjects. Further studies using older subjects, 42 - 66 years old, indicated that sleep after Horlicks, was of a longer duration with a reduction in the periods of wakefulness and Stage 1 sleep. They concluded that Horlicks, although weak in effect compared to barbiturate type sleeping drugs, may be of use in long - term applications.

Mullin et al (1933) studied the effect of alcohol and caffeine on motility during sleep. They concluded that alcohol did not effect the total number of body movements, but served to redistribute them, with a decrease in motility for the initial half of the night and an increase in motility in the second half. Caffeine however, increased the overall motility for the duration of sleep. These results are in agreement with those of Kleitman (vide 3.3.3).

Weight: Crisp and Stonehill (1976), studied the effect on sleep of weight changes of the subjects. Two groups were selected, overweight obese and underweight thin patients. The overweight subjects were fasted, while those who were underweight were encouraged to eat. The group of dieting

overweight patients were observed to have an increased body motility, whereas the underweight patients exhibited a decrease in the number of body movements.

Maliniak (1934) also studied the effect of fasting upon the motility of sleeping subjects. She observed a reduction in motility on the first night, after which it returned to its previous level.

Environmental influences: Townsend et al (1973) observed a relationship between body movements and tone pulses. The tone acting to stimulate a movement which occurred approximately seven seconds after the tone. Muzet et al (1974) conducted a test whereby they exposed a number of sleeping young adult volunteers to tone pulses of a fixed intensity, 90dB(A), for varying periods of different frequencies. There was no apparent change in the overall number of movements made during sleep, although it altered the distribution of the moves throughout the night. Williams et al (1964) found that prolonged sleep deprivation (64 hours) served to almost eliminate the body's normal behavioural response to an aural stimulus, irrespective of its intensity. Bardsley (1977) reports work of Metz and Muzet (1975) and Scott (1972) who both showed that a continuous loud noise (93dB(A)) lightened sleep for the first night. However, subsequent nights exhibited a more normal sleep pattern. This being indicative of a period of adjustment.

Kleitman et al (1937) reported a study of the effect of different environmental conditions on sleep. These included outdoor and indoor temperatures, humidity and rain. However, it was not possible to establish a significant relationship for any of the variables. The effect of the long term seasonal variations on body motility during sleep has been investigated by Kleitman (1963). He concluded that the general pattern of motility and the distribution between periods of movement and immobility, remained unchanged for each of the four seasons of the year. Although there was a variation in the magnitude of the movements and the duration for which they occurred.

Bed-mattress interface: Suckling et al (1957) measured the body movement of four subjects sleeping on a range of mattresses, each of a different hardness ("hard", "medium" and "soft"). They found that differing support surfaces did influence movement during sleep, with the "hard" surface

promoting an increase in motility, although the variations were not always statistically significant.

A report prepared for the National Health Service (1968), indicated that an alteration in the body-support surface was capable of changing some of the characteristics of movement during sleep. However, as indicated by Bardsley (1977), shortcomings in description of experimental technique and method of analysis, meant that the results were of limited value. Bardsley (1977) assessed the effect on movement during sleep of three different mattresses (bead, foam on springs, foam on board). Each subject slept for a period of four or more nights on each mattress. He observed a statistically significant difference in the total number of moves made during the night between subjects sleeping on the bead mattress and those sleeping on the foam mattress on board, for all subjects. The difference between the foam mattress on springs and the other two support surfaces was not found to be significant for all of the subjects. This result further supports those of Suckling et al (1957) indicating that the mattress is a relevant variable when considering body movement during sleep.

Emotional state: The effect of changes in emotional state in the period before sleep, such as shock, apprehension, anxiety, psychosis and pleasant anticipation, have all been shown to affect motility during sleep. (Maliniak, 1934; Giddings, 1936; Dement and Roffwarg, 1965; Feinberg et al, 1965).

3.4 METHODS OF MONITORING MOVEMENT DURING SLEEP (OTHER THAN EEG)

Bardsley (1977) provides a comprehensive review of techniques evolved for monitoring body movement during sleep. The techniques that have been employed include, measurement of the displacement of all or part of the bed, observation, ultrasonic radiation, measurement of body section displacement and measurement of mechanical forces generated in,or by, the support structure. Each of these monitoring principles and their associated instrumentation are reviewed below.

3.4.1 Measurement of the displacement of all or part of the bed

The majority of the methods previously used for the assessment of body movements during sleep, have relied on this principle. Perhaps the earlist use of such a system was by Szymanski (1914) who studied the characteristics of animal and human motility. The apparatus consisted of a combination of pulley, string and recording equipment. The string was attached to a bedspring or, for animals, a suspended cage, leading via a pulley to recording apparatus. Kleitman (1963) likened the principle of operation to that of a seismograph and claimed that, with suitable adjustment, the system could be made sensitive enough to pick up the effects of respiration and pulse.

Renshaw and Weiss (1926) also employed the principle of the deformation of a bedspring. However, they made use of a system which relied upon the making and breaking of electrical contacts. A metal bar containing alternate inserts of brass and bakelite was attached to one of the bedsprings in the centre of the bed. A small brass wheel was positioned against the rod, while also being electrically connected to the recording device, termed a "hypnograph". This consisted of 24 stylus pens (allowing the simultaneous recording of up to 24 beds), which wrote on a uniformly travelling recording paper. When the subject moved, the rod was displaced, causing contacts to be made and broken, resulting in electromagnets on the hypnograph marking the chart.

Kleitman (1932) reviewed the methods available for measurement of movement during sleep. He described their principle of operation as direct

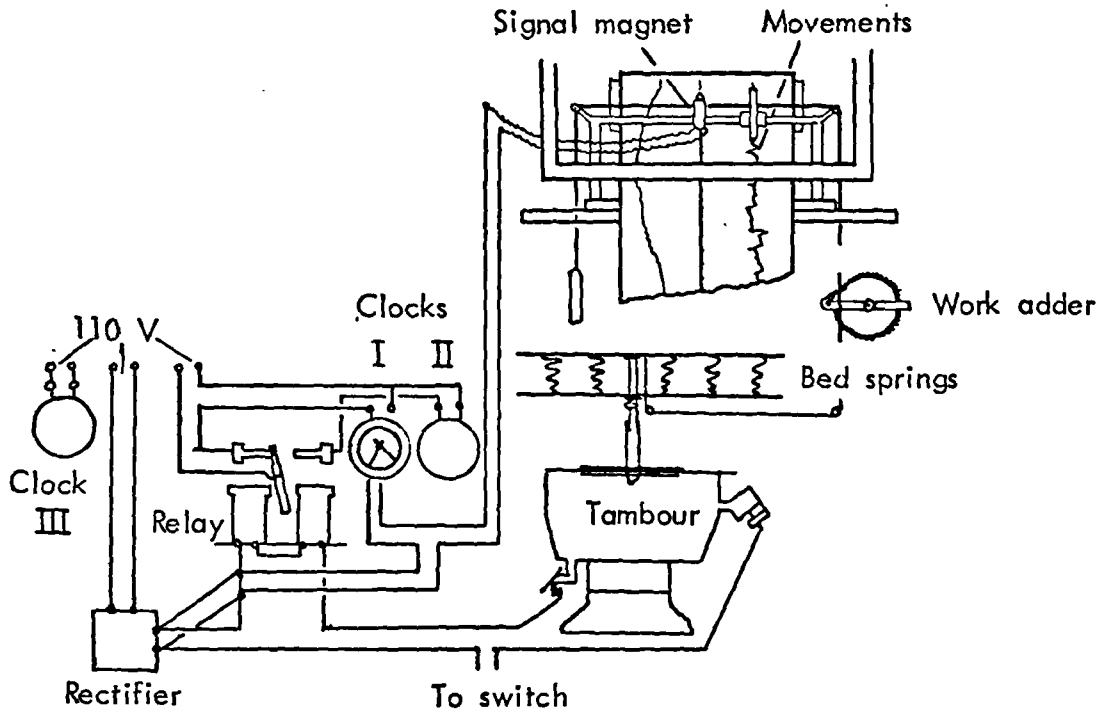


Figure 3.5 Apparatus for determining the duration and occurence of the movements made by a subject in bed, utilising the deflection of a bedspring.

(after Kleitman, 1932)

transmission to a recording lever, pneumatic transmission to a recording tambour and electrical transmission to a signal magnet. The latter records the frequency of movements only, the others additionally recording the magnitude of the move. All had an output format which produced a continuous recording on a kymograph of the position of the bedspring (or whole bed). The technique of direct transmission to a recording lever has been illustrated by the method of Szymanski (1914) while that attributable to Renshaw and Weiss (1926) is illustrative of transmission to a signal magnet. Pneumatic transmission to a recording tambour was the method selected by Kleitman himself to measure body motility in bed. His device is illustrated in figure 3.5. A rigid connection was made to a bedspring, the opposite end of which was attached to the rubber surface of a tambour. Outlet and inlet valves to the tambour were connected to an electrical relay. Three clocks (I, II and III), were employed to provide a measure of the duration of the body movements. Two of these (I and II) were controlled by the relay, the third (III) being separate and running continuously. Any change in pressure in the tambour, caused by a movement in the bed, resulted in the relay contact "breaking", which cut the power (disabled) to one of the clocks (II), and applied power (enabled) another (I). At the end of the movement, the relay was "made" enabling clock II and disabling clock I. At the conclusion of the experiment, the disparity between clocks III and II should have been equal to I, and representative of the sum total duration of the body movements performed by the subject during the night. However, repeated switching on and off of the clocks (I and II) led to inaccuracies as a result of their inertia. However, by calibration it was possible to provide separate correction factors for each of them, compensating for the error. Measurement of the magnitude of a move was accomplished by a "conventional" pulley and string system, combined with a slow moving kymograph.

When considering the design of a motility measuring system Laird (1935) was critical of all previous techniques. He claimed that many were functions of the mechanical characteristics of the mattress and springs, making these methods inaccurate. Additionally. some of the techniques were said to

be aperiodic and to suffer from phase-effects. Those methods utilising electrical signals were deemed insensitive, on occasion requiring a movement of nearly 6.5 mm of the bottom of the mattress before an event was noted. As a result, Laird (1935) designed a device to circumvent these problems. A small brass plate was placed on top of the mattress in a position approximating to the centre of the bed. From this plate a 1.6 mm metal rod passed through the mattress to a fishline and then to a recording instrument. He claimed that it was possible to adjust the system to account for differing body weights, and quoted a system sensitivity, with the subject supine, of 1.6 mm deflection in the record line for a 51 mm movement of the forearm. No comment was made with regard to possible artefacts arising from the subject having to lie on a brass plate or of errors attributable to any non-linear mechanical properties of the mattress.

The continuing popularity of motility measurement by means of assessing the displacement of a section, or sections, of the bed is indicated by the work of Sherwin et al (1961) who used mattress displacement during a movement, to activate an inertia switch via a rack and pinion type device. An electrical impulse counter activated by the switch then served to count the number of movements. The output of the system being the total number of movements executed by the patient during the night. Although this technique proved satisfactory for its intended application, the nature of its output was restricted to the number of movements only. It provided no information on the time when the movements had taken place, the rate of movements, duration of movements, or the magnitude of the movements, other than that they were capable of triggering the inertia switch. Crisp et al (1970) employed a device comprising a long brass tube with slits at preselected intervals, attached to the bed springs. The tube contained an incandescent light bulb and was encased in the manner of a piston in a cylinder. Mounted inside the casing, also at chosen intervals, was a set of photodiodes. Thus, when a movement occurred and the tube was displaced, if one of the slits in the tube passed one of the photodiodes a voltage pulse was transmitted to the recording apparatus. By knowing the intervals between slits and photodiodes, it was possible to calculate the size of the movement. Crisp et al (1970), recognised that their

system was weight dependent and by careful calibration were able to formulate an equation allowing them to compensate.

3.4.2 Observation

Of all the mobility assessment techniques, observation of the subject is the simplest, and requires only an observer to watch over the sleeping subject, noting changes in posture and time of occurrence. Unfortunately it is difficult for such an observer to remain alert for the whole of the sleeping period. As a result, methods have been developed whereby the subject is photographed by a cine camera (Branton and Grayson, 1967; Southwell et al, 1972), or movement triggered still camera (Muzet et al, 1972; Bardsley 1977), allowing analysis of the night's record at the convenience of the investigator. One of the drawbacks of these types of assessment are the restrictions imposed on the subject and thus the fear of artefactual data. As an example, Southwell et al (1972) required the subject to wear dark pyjamas with no covering on the bed, and to provide sufficient illumination, the room was lit by two tungsten bulbs of 100W each. Also, analysis of the filmed records is extremely laborious, requiring projection of the film, frame by frame, while manually counting and recording changes in position. Close circuit television systems, linked to time lapse video tape recorders, have also been used to monitor movement during sleep (Ryan, 1979). Although this technique allows direct observation, with analysis at the experimenter's leisure, such analysis is tedious and necessarily subjective.

3.4.3 Ultrasonic radiation

Bardsley (1977) suggested that techniques utilising this principle are of limited value unless costly and sophisticated systems are employed. The major restriction being that only gross body movements can be detected, with no information regarding the magnitude of a movement or the body segment(s) involved. However, he further commented that should these limitations prove to be acceptable, there are cheap but effective systems commercially available; burglar alarms are an example and may be used with only minor modifications.

3.4.4 Body section displacement

Techniques utilising this principle have been employed by several investigators to provide an assessment of movement during sleep. Such methods involve the attachment of transducers to one or more of the "body sections" (arms, legs and trunk). The transducer may be in the form of weighted electrical potentiometers (Kresse and Rettenmaier, 1973) or accelerometers (Korn et al, 1974) the only real specification being an output related to motion/displacement. The transducers need to be physically attached to the body sections in question. However, this results in a disruption of the normal environment, especially if there are long, trailing leads between the transducer and the recording equipment, increasing the possibility of artefacts. Although miniature transducers have been fabricated, there is still concern that they disrupt normal sleep due to discomfort. O'Brien (1976) has developed a small cassette recorder which may be worn by the subject, preventing trailing leads and allowing continuous recording of a complete night's data. Andrews et al (1978) made use of posture switches, attached to the skin overlying the sternum. Recording the output on a miniature cassette and playing back the recorded signal into a microcomputer, it was possible to obtain information relating to the occurrence of a move and any resulting change in posture. Such a system proved simple and easy to use. However, it still required the attachment of transducers to the subject and did not provide information on the magnitude of a move.

3.4.5 Measurement of mechanical forces generated in, or by, the support structure

Techniques involving this principle, invoke two of the basic laws of statics. That of equilibrium, whereby a system of forces is said to be in equilibrium when the resultant of all the forces, and the resultant of all the moments at one point are equal to zero, and that of action and reaction, whereby any load on a support causes an equal and opposite load to be applied to the support, so that action and reaction are two equal and opposite forces. Using these principles, it is possible to monitor "a structure" in such a way as to be able to calculate any change in position of load upon that structure.

Dotzler (1972) used an "instrumented bed" to determine the change in three dimensional forces in the bedframe, which occurred as the result of a movement. Recordings of the forces were made simultaneously with conventional EEG tracings.

Fernie (1973) constructed a system which, by means of a load cell, measured the force in one of the legs of the bed, variations in this force being a direct function of the size of the move. By means of suitable amplification, monitoring the output with an ultraviolet galvanometer recorder, he was able to attain sufficient sensitivity to distinguish "even the movement of an arm". From a study involving a small number of subjects, he deduced that his technique was capable of reliably monitoring mobility, although the results were biased in favour of the body segment nearest the load cell. Bardsley (1977) extended this technique to involve the use of two load cells, placed under the bed, providing a uniform sensitivity over the complete bed surface (vide 4.2).

When measuring movements of seated subjects, Rieck (1969) and Bardsley (1977) used four load cells, one placed beneath each leg of a chair. After amplification and processing of the outputs from the transducers, they were able to determine the magnitude and duration of movements of the seated subject.

3.4.6 Indirect methods

The most important of these techniques involves the study of EEG artefacts. Sections 3.2 and 3.3 discuss the EEG, its characteristics and its use in studies of mobility during sleep.

Oswald (1974) employed an innovative technique, utilising the sound of the bed itself as an indicator of movement. He attached a microphone to the "central bedsprings" amplified the output and recorded it. It was claimed that the method was able to distinguish both the occurrence of a move and to make crude approximations relating to the magnitude of the move. He used this technique to compare the mobility between groups of "normal" and "mentally-ill" patients .

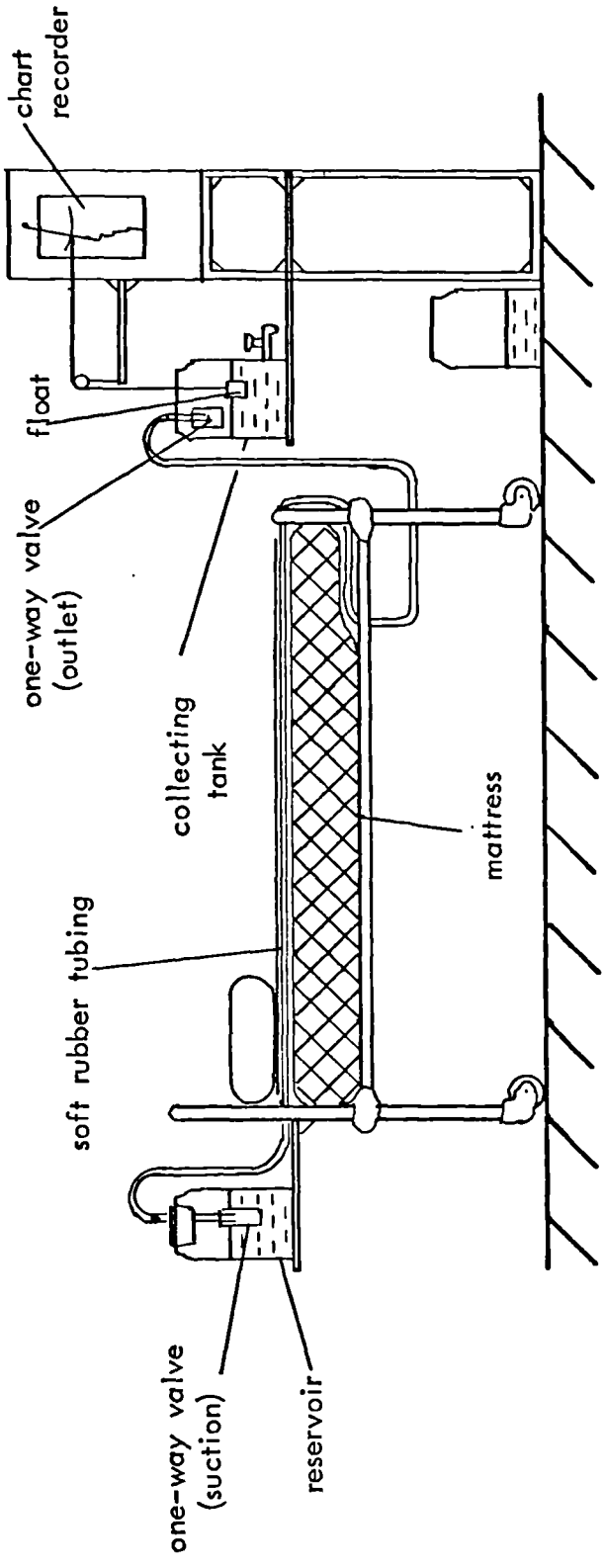


Figure 3.6 Technique for monitoring overnight mobility, utilising the displacement of a liquid from a tubular rubber sheet. (after Brocklehurst et al, 1974)

Brocklehurst et al (1974), employed an Archimedian type method to the measurement of "restlessness in sleep" (figure 3.6). A multi-tubular, water filled, sheet was placed on top of the mattress. Each tube was fitted with non-return valves, so that when the subject moved, water was ejected into a receiving tank and an equivalent amount drawn from a supply tank. The volume of water in the receiving tank was recorded continuously, by means of a simple float mechanism. Brocklehurst et al (1974), postulated that the amount of water collected in the receiving tank was approximately equivalent to the "acquired area" of the subject after moving. He used this system as the basis of a limited drug trial. The techniques developed by Oswald (1974) and Brocklehurst et al (1974) can only be considered as qualitative in nature, providing information relating to a move "event" only.

Alihanka and Vaahtranta (1979) proposed a method for recording body movements during sleep, by means of a static charge sensitive bed. Two metal plates, separated by an insulator (wood), were placed beneath the mattress of the subject's bed. A sensitive differential amplifier was employed to measure the potential difference between the plates. As the subject moves in bed, static charges are induced in the clothing and in the mattress, these charges being sensed as a potential difference between the two metal plates. To obtain any reliable measure of the movement it was necessary to calibrate the system by having each subject perform a set of "typical movements" before each experiment. Provided this regime is followed, the technique is claimed to be extremely sensitive, detecting pulse fluctuations and even the movement of a finger. Unfortunately, this method is easily prone to artefact. For example, the output is affected by anyone moving close by the bed and thus requires a Faradic cage, or equivalent, to electrically shield the bed. In addition, humidity changes may significantly alter the charge build up requiring controlled environmental conditions.

3.5 SUMMARY

Physiologically, two different types of sleep have been identified. Rapid eye movement (REM), during which period dreaming occurs and non-rapid eye movement (NREM) which constitutes the majority of the sleep period (80%).

NREM sleep is commonly classified into four stages, related to the level of arousal of the subject. Stage 1, associated with a low arousal threshold, or light sleep, to Stage 4, associated with deep sleep. The type and stage of sleep changes throughout the sleep period. Typically, the early part of the night is spent in deep sleep (Stages 3 and 4), after which REM sleep alternates regularly with NREM sleep at 90 minute intervals.

Body movements occurring during sleep have been shown to be related to the sleep cycle, with their rate, distribution and magnitude varying accordingly. The deep sleep at the beginning of the night contains a small number of large magnitude moves, whereas REM and Stage 2 sleep include an increased number of medium and small magnitude moves. Body movements have also been closely correlated with the occurrence of particular waveforms in the sleep record, most notably the K complex.

Sleep may affect, or be affected by, a range of factors both intrinsic, eg. age, heart rate, respiration, blood pressure, gastric activity, urinary bladder pressure, and emotional state; and extrinsic, eg. drugs, oral consumption (fasting), environmental conditions and support surfaces. By changing sleep characteristics, these variables also serve to modify the pattern of body movements.

Techniques for the measurement of body movements of a sleeping subject in bed have employed a number of different principles. The measurement of the deflection of the support surface has been widely used, employing a range of suitable instrumentation systems. Other principles include direct observation, ultrasonic radiation, body section displacement and measurement of mechanical forces generated in or by the support structure.

CHAPTER 4

EXPERIMENTAL EVIDENCE FOR A RELATIONSHIP BETWEEN CHANGES IN INTERFACE PRESSURE AND BODY MOVEMENT

4.1 INTRODUCTION

Chapter 2 described the aetiology of pressure sores. The importance of patient movements in the relief of pressure induced ischaemia and thus the prevention of pressure sores was emphasised. However, no experimental data exists to provide quantitative information about the acknowledged relationship between mobility and pressure relief of the tissues.

In earlier research programmes at the Bioengineering Unit, University of Strathclyde, techniques have been developed to assess, the mobility of normal subjects (Bardsley, 1977) and interface pressures between the body and the support surface (Ferguson-Pell, 1977), during sleep. By the use of these two techniques working simultaneously, it was possible to assess the association between movement and corresponding interface pressure variations.

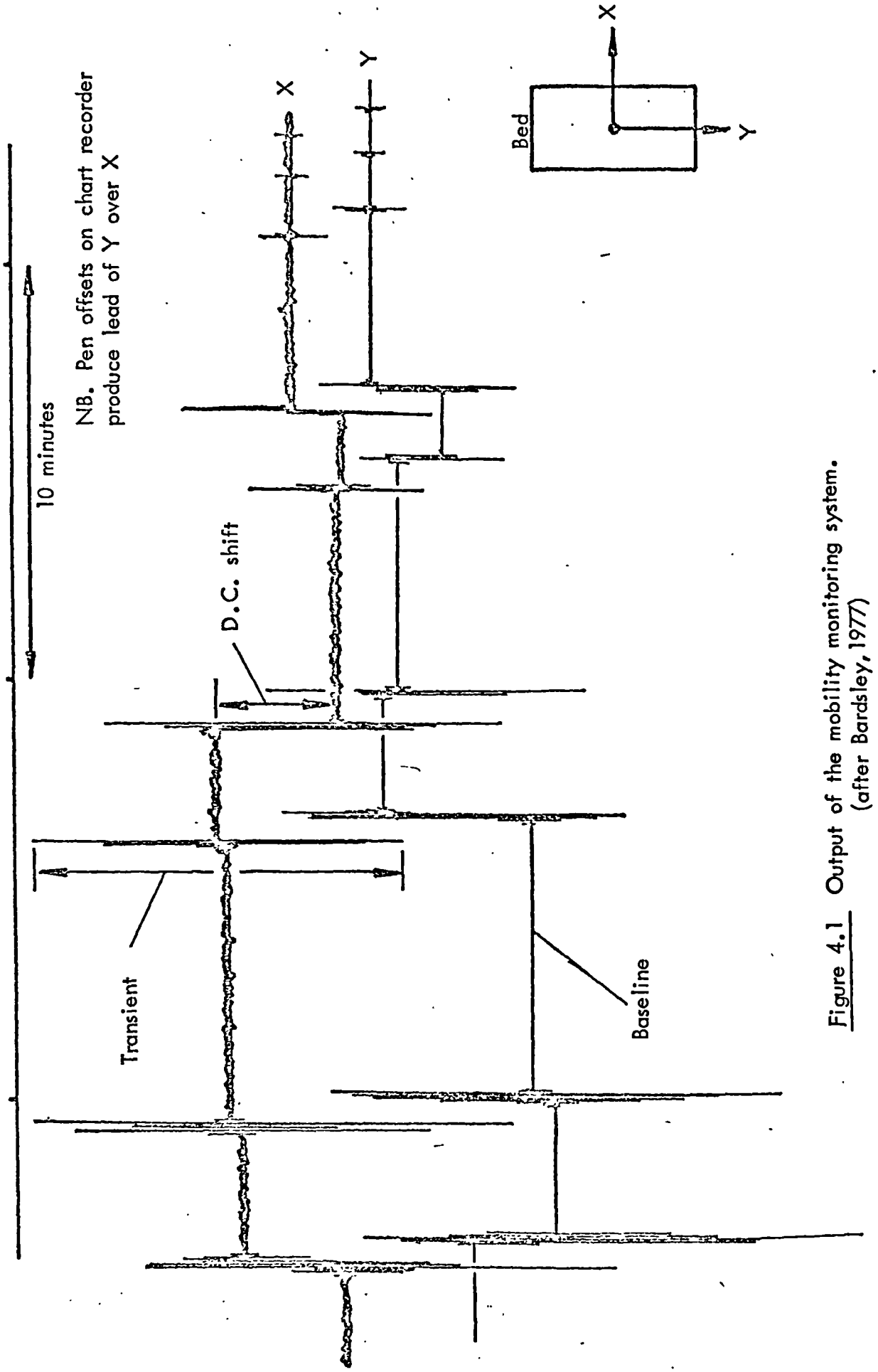


Figure 4.1 Output of the mobility monitoring system. (after Bardsley, 1977)

4.2 MOBILITY MONITORING APPARATUS

This system was designed and used by Bardsley (1977) to study the characteristics of the mobility of normal subjects during sleep. His equipment was designed for use in a "sleep laboratory", situated in a local hospital.

Bardsley based the design of his system on the measurement of the reaction forces generated between the bed and the floor, (vide 3.4). He employed a three point support system, with load cells under two of the supports. The output from the transducers was amplified and provided the input to an analogue computation system, which determined the position of the subject's centre of gravity in the bed. The system was sensitive to changes in body weight between subjects. However, this was accounted for by the use of a calibrated scaling potentiometer, adjusted prior to each experiment. The system was capable of measuring the movement of a subject in bed (with reference to the position of their effective centre of gravity), in two dimensions, to within an accuracy of $\pm$ 4 mm.

The output of the analogue circuitry was recorded by means of a three channel pen recorder Type Watanabe MC641 (Environmental Equipment, Nantwich,U.K.). A typical example of the recorded output is illustrated in figure 4.1. The two channels represent the X and Y co-ordinates, which defined the position of the subject's centre of gravity in bed. A change in voltage level of either one or both of these channels was indicative of a move by the subject. The output produced by the mobility monitoring system was considered to be the combination of three components. Firstly, an unchanging steady state voltage (baseline), forming the majority of the signal and occurring when the subject was lying still, in the bed. Secondly, high frequency voltage transients and thirdly a low frequency change in voltage level (d.c. shift). The latter two components were produced as a result of a movement by the subject. That is, each move was composed of high frequency (dynamic) and low frequency (quasi-static) components.

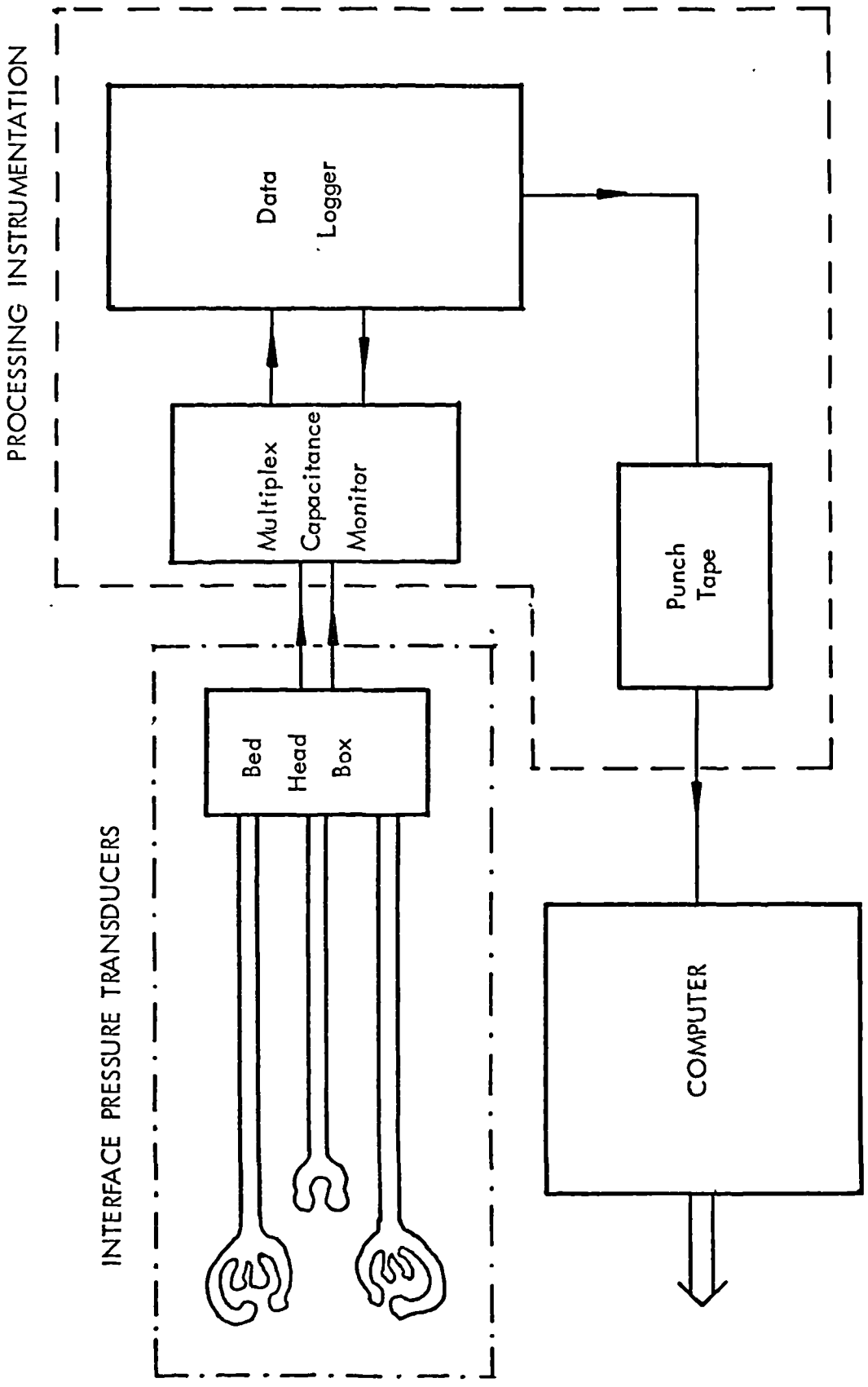


Figure 4.2 Schematic diagram of the interface pressure monitoring system.
(after Ferguson-Pell, 1977)

4.3 PRESSURE MONITORING APPARATUS

An apparatus was designed, to measure the interface pressures between the body and the support surface, by Ferguson-Pell (1977). The system may be considered in three parts (figure 4.2).

- Interface pressure transducers, placed between the body and the support surface.
- Processing instrumentation, amplifying the signal from the transducers and manipulating it into a form suitable for recording on punched tape.
- A computer with associated software, to linearise, list and plot the data.

4.3.1 Interface pressure transducers

There have been numerous studies involving the measurement of body-interface pressures. (Kosiak, 1959; Lindan, 1961; Houle, 1969; Reswick and Rogers, 1976; Shaw, 1979). One of the major limitations in all of these studies has been the existence of artefacts, caused by the presence of the pressure sensor itself. In an effort to reduce these artefactual results, it was desirable that the transducer be as thin as possible.

Ferguson-Pell (1977) developed a transducer which utilised the concept of the parallel plate capacitor with a mechanically compressible dielectric. Two thin layers of dielectric (polyester adhesive), were sandwiched between three layers of thin metal foil (figure 4.3). The outer foils were earthed and an energisation voltage of 1V, peak to peak operating at 1k Hz, was applied to the central foil. A load applied to the transducer deformed the dielectric, producing a change in capacitance, which was measured by associated processing instrumentation. A direct, although non-linear, relationship was established between the magnitude of the force applied and the changes in the capacitance of the transducer. The device had a load sensitive area of $100mm^2$ and an overall thickness of approximately 0.4mm. This advantageous width to thickness ratio was considered acceptable for use as an interface pressure sensor. Details of manufacture, design and specifications are described by Ferguson-Pell (1977).

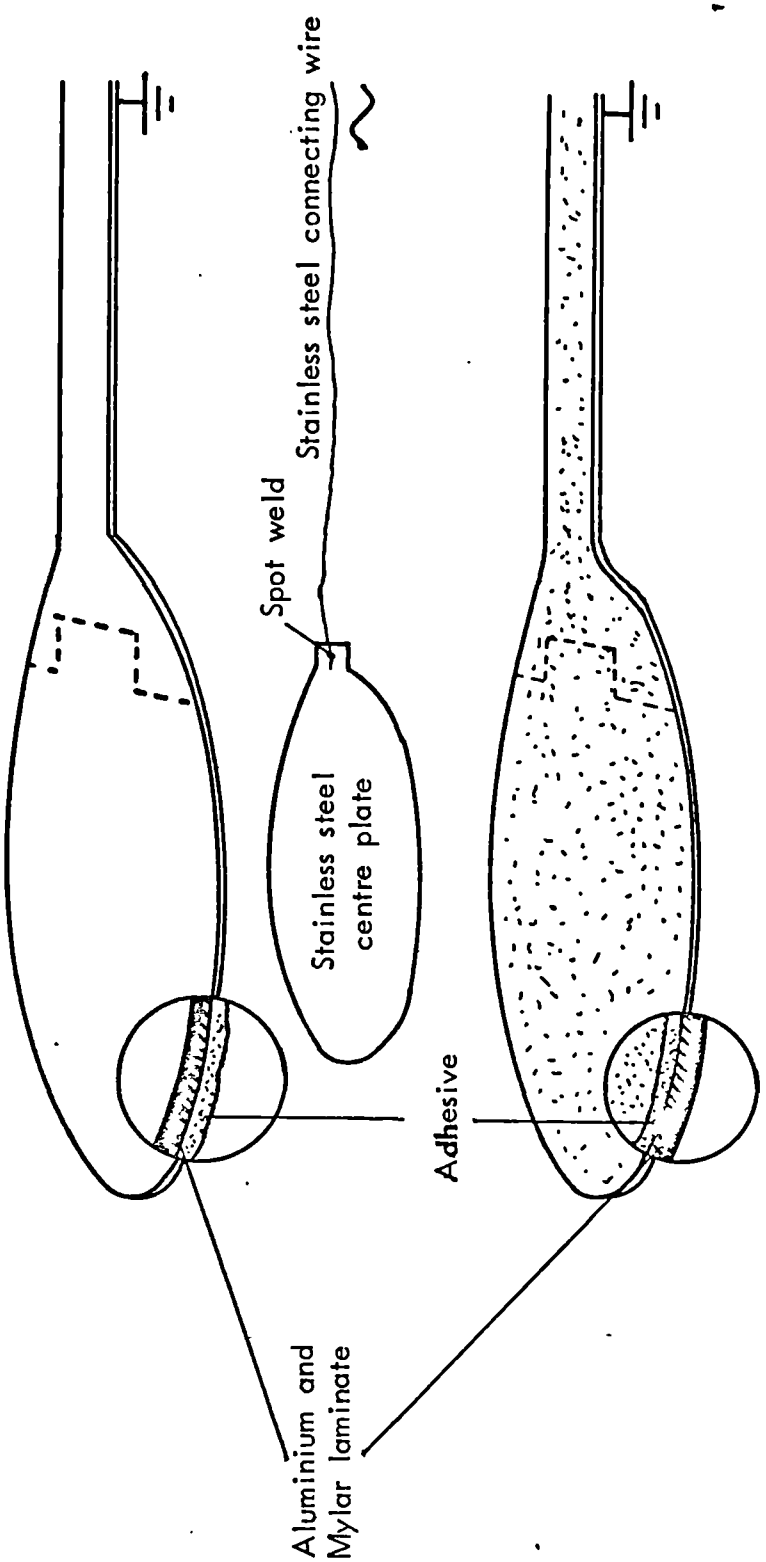


Figure 4.3 Schematic diagram of the interface pressure transducer.
(after Ferguson-Pell, 1977)

4.3.2 Processing instrumentation

In his study, Ferguson-Pell (1977) used a matrix of pressure transducers, attached to a number of different sites on the body surface.

A multiplexer served to produce a single channel signal containing sequential information relating to its multichannel input, which was provided by the output of the pressure transducer. At any instant a transducer formed one arm of a capacitance bridge. Any change in capacitance of the device, resulted in an imbalance of the bridge which, in a similar manner to a Wheatstone bridge, resulted in a change in output voltage. A Mycalex Series 5 data logger was used to convert the analogue signal into digital form, suitable for recording on punched tape. The data logger scanned the output from the multiplexer continuously, or at preset intervals, ranging from 1 minute to 30 minutes.

4.3.3 Computation and output format

An I.C.L. 1904S digital computer was utilised to process the "raw data", as collected on the punched tape. By the use of calibration constants, assessed for each transducer and information regarding zero level output and electronic drift, the computer calculated the output of each transducer, producing results in suitable units of pressure.

An example of the systems output format is shown in figure 4.4. The output of the interface pressure monitoring system was presented in two forms, firstly as a matrix of measured pressure against scan number and secondly in graphical form, providing a visual representation of the processed data.

4.3.4 Link with the mobility monitoring system

To provide temporal synchronisation between the interface pressure and mobility monitoring systems, the chart record produced by the latter was annotated. The data logger operating an event marker pen on the chart recorder, each time the pressure transducers were scanned. A typical chart recording, inclusive of annotation, is shown in figure 4.1.

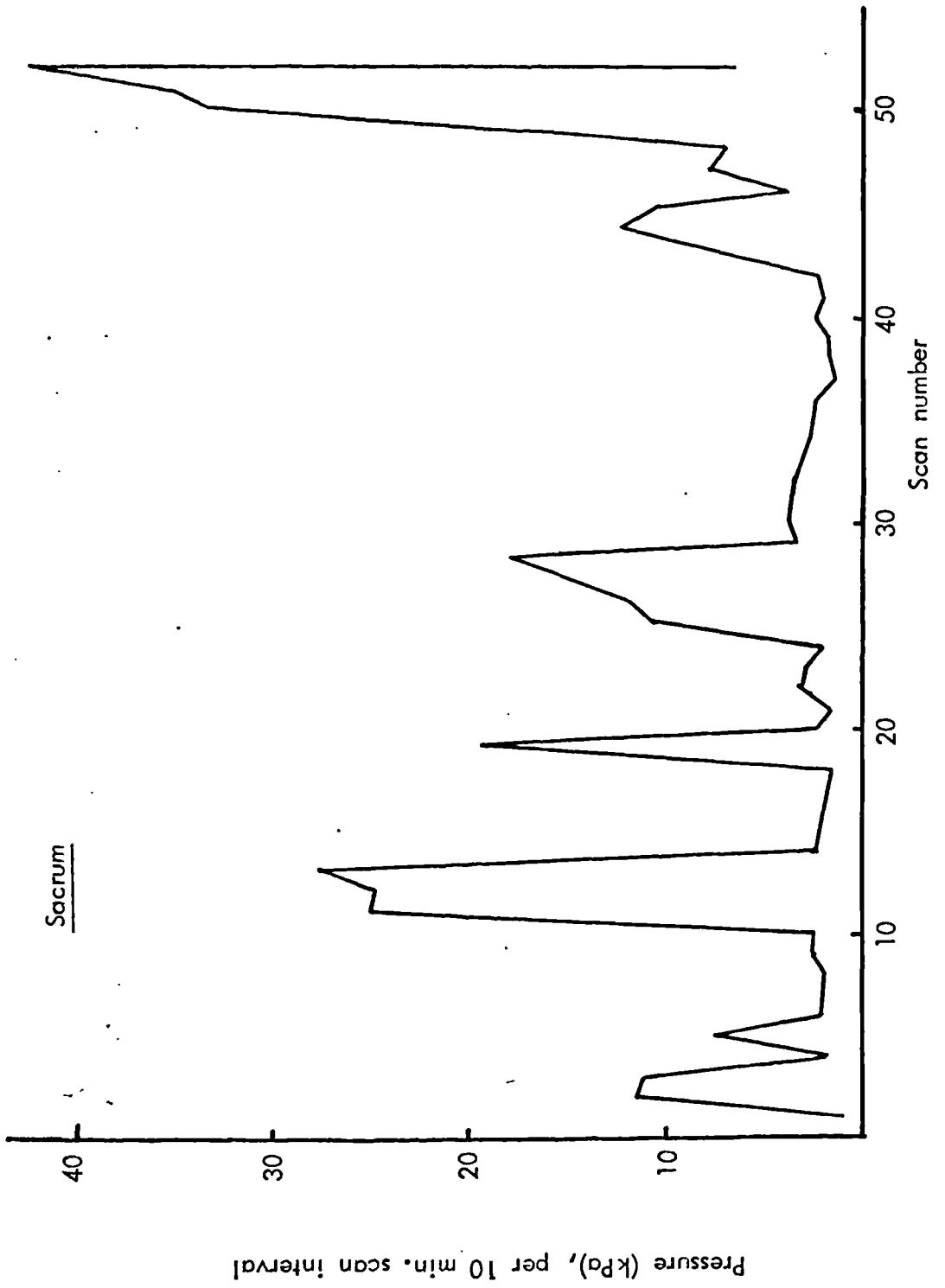


Figure 4.4 Graphical output of the interface pressure monitoring system.
(after Ferguson-Pell, 1977)

4.4 EXPERIMENTAL PROTOCOL

4.4.1 Selection of subjects

The nine subjects selected were normal, healthy adults, drawn from the student population of Strathclyde University and ranged from 22 to 28 years in age, 2 (22%) of these were female. The equipment was established at a local hospital requiring the subjects to sleep in an unfamiliar environment.

Two or three tests were conducted on each subject, usually on consecutive nights and always within the same week. Bardsley (1977) commented on the importance of a period of acclimatisation (usually one night) to allow the subject to adjust to a new sleeping environment. However, in this study, the primary interest was the relationship between mobility and pressure relief and not in establishing patterns of normal sleep. Thus, every night's recording was analysed and the concept of a period of acclimatisation was ignored.

4.4.2 Test environment

Use was made of a sleep laboratory, comprising a suite of three rooms, established at Philipshill Hospital, Glasgow. One room served as a bedroom for the subject. Load cells for the mobility monitoring system, were placed underneath the legs of the bed. A camera and flash unit were positioned directly over the bed in order to photograph the subject throughout the course of the night. The three matrices of pressure transducers were connected to a junction box, placed at the head of the bed. Environmental conditions, primarily those of temperature and light, were adjusted to suit the individual preference of the subject. An adjoining room contained the instrumentation, which could be monitored and adjusted during the test, without disturbing the subject. The punch tape machine, which was noisy during its operation, was acoustically insulated.

4.4.3 Interface pressure measurement

Site: For each subject, the interface pressure between the body and the support surface was measured. The sites selected were the sacrum, left trochanter and right trochanter. A matrix of four transducers was attached to

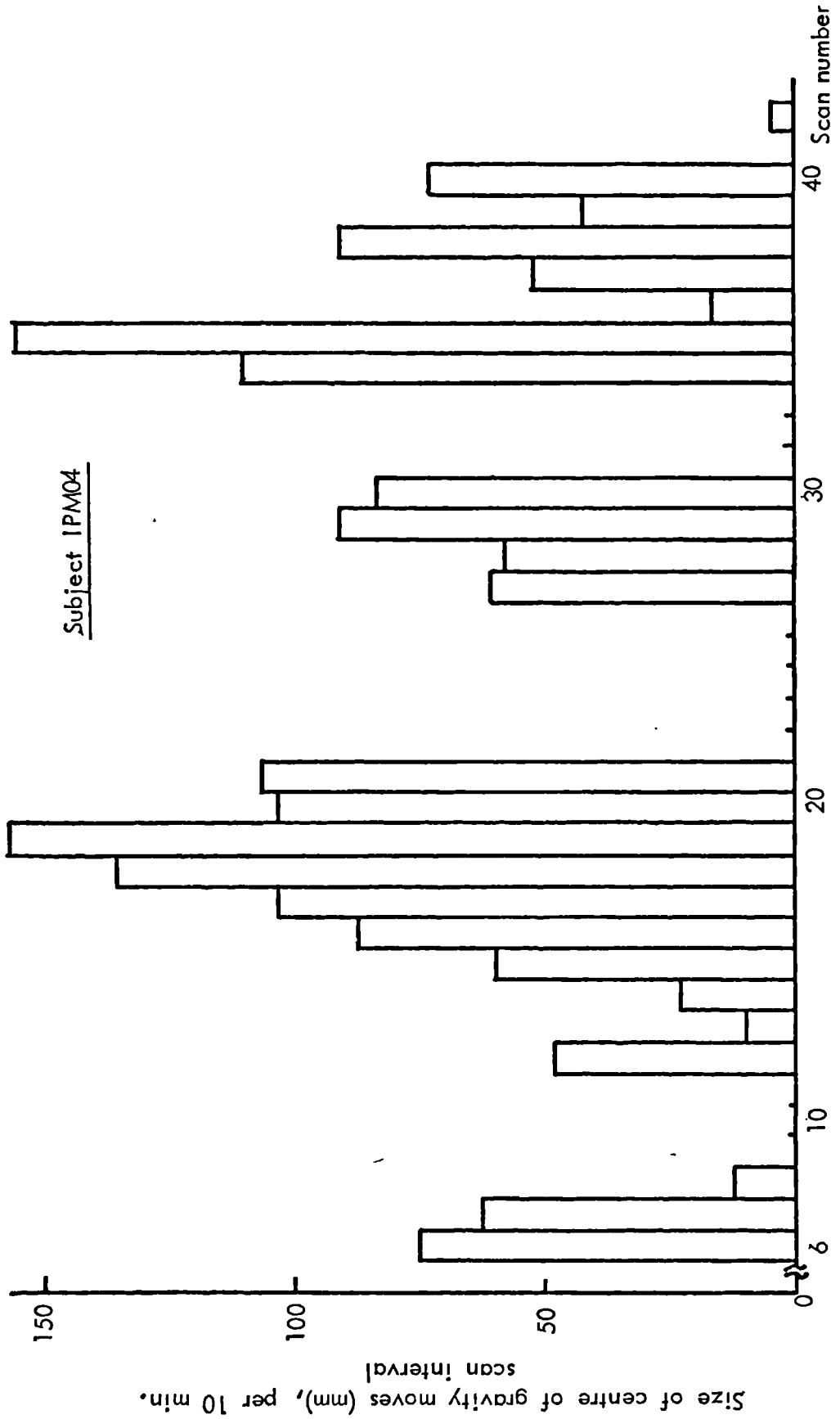
the test, indicated that the transducer had been damaged and that it should be replaced. The results obtained from transducers that proved to be faulty after the test, were discarded.

Approximately fifteen minutes before the test was due to begin, the attendant switched on the instrumentation. The data logger and the multiplexer were synchronised, by adjusting the 32 trigger pulses, provided by the data logger to be in phase with the multiplexer, ensuring a correct discrimination between each of the transducers. The mobility monitoring apparatus was then adjusted. The scaling potentionmeter was adjusted toaccount for the body weight of the subject. The output of the system was zeroed, with the subject lying centrally in the bed.

At this stage the matrices of pressure transducers were attached to the subject. To improve repeatability from subject to subject the matrices were aligned using anatomical landmarks, in combination with a stencil. The leads from the transducers were all brought to the small of the back and secured to the skin by means of adhesive tape. The leads were then run up along the back of the subject to the base of the neck, to be connected to the junction box at the head of the bed. The subject was requested to wear pyjamas, since this was thought to reduce the likelihood of the transducers becoming detached and of entanglement with the leads during the course of the night.

Having attached the three matrices of transducers, the subject was instructed to lie in a prone position on the bed. The output from the transducers was then scanned by the data logger and recorded on punched tape. If the subject was lying prone, no load was applied to any of the transducers. Thus, the output of the transducers was equivalent to a zero load condition. When processing the data, this punched tape recording was added to that produced during the night and provided the computer with information regarding the resting condition of the transducer.

On the morning following the test, the data was collected and later analysed as outlined in the following section.



M^c_{SL} = signal level change (m^c_{SL}) between the start and end of a scan.

Figure 4.5a Size of centre of gravity moves occuring during the night (M^c_{SL}).

4.5 ANALYSIS

The data produced by each experiment was in two parts, a chart recorder tracing, illustrating the output of the mobility monitoring apparatus, and a punched tape recording of the output of the interface pressure system (vide 4.2. and 4.3.3). The chart recording and punched tape were analysed separately. Comparison was then made between the two sets of analysed data.

4.5.1 Mobility monitoring

Section 4.2 indicated that the output of the mobility monitoring system could be considered to be the combination of three components, steady state (baseline), high frequency transients (m_T) and low frequency shifts in signal level (m_{SL}). The latter two components were associated with a move by the subject in the bed and the mobility of each subject was analysed with regard to each of these.

By measuring the chart recording, the magnitude and time of occurrence of each transient and low frequency shift, occurring during the test, was established.

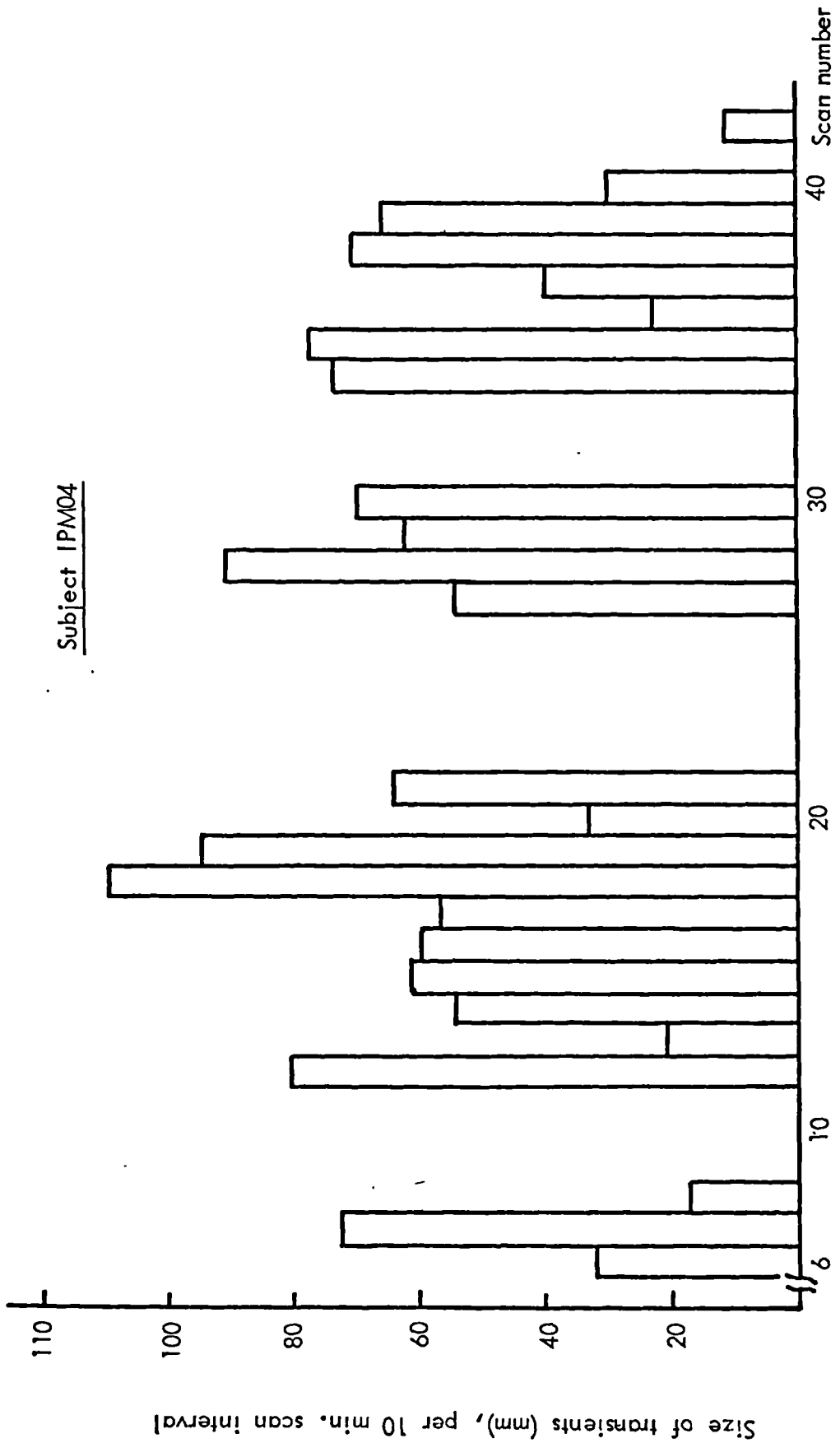
The mobility record consisted of two traces relating to a co-ordinate system (X and Y) established earlier (vide 4.2, figure 4.1). In the case of m_{SL} these were combined to form the magnitude of their resultant, that is

$$\sqrt{X^2 + Y^2}, (m_{SL}^c) \quad \text{and for} \quad m_T \text{ as} \frac{X+Y}{2} (m_T^c).$$

To aid in comparison with interface pressure data, the mobility trace was further divided into time intervals, corresponding to the scan interval of the data logger. The magnitude of the largest transient (M_T^c), within each interval was noted, as was the change in signal level (M_{SL}^c) between the beginning and end of the interval. Figures 4.5a and b are examples of typical results for M_{SL}^c and M_T^c, respectively.

4.5.2 Interface pressure monitoring

The output format of the data produced by the pressure monitoring system, with a scanning interval of ten minutes, was described in section



M_T^c = the largest transient (m_T^c) occuring within a scan.

Figure 4.5b Size of move transients occuring during the night (M_T^c).

4.3.3. However, analysis of those experiments employing a 1 minute scanning interval, produced a large volume of data and demanded "pre-processing". This involved dividing the total length of punched tape, collected each night, into a number of smaller parts, which were then input to the computer. Thus the complete night's recording was stored in a number of small data files. Software was developed to join all of these together, providing editing facilities to remove the gaps in the recording and finally to produce a graphical representation of the data.

The processed data of both the ten minute and one minute scanning interval tests, contained the output of the ten interface pressure transducers, which were attached to the body of the subject. Each transducer was considered individually and the measured pressure before and after each scan was compared. The magnitude of the change ($|\Delta P|$) and the scan number were recorded. Results produced by transducers that exhibited different pre and post calibration characteristics, usually as a result of damage to leads or failure of the adhesive bond between the metal plates, were discarded.

The transducers were arranged in the form of three matrices overlying the left and right greater trochanters and the sacrum (vide 4.4.3). Thus, for each scan, the change in pressure representative of each matrix, was considered to be the peak change in pressure $(|\Delta P|)_{max}$ recorded by any one of the transducers in the matrix. A combination of,

$$(|\Delta P_S|)_{max} + (|\Delta P_{LT}|)_{max} + (|\Delta P_{RT}|)_{max} = \Sigma\ (|\Delta P_{max}|)$$

represented an overall change in interface pressure, measured at the sacrum, left trochanter and right trochanter during the time interval between scans. Figure 4.6 illustrates an example of the combined change in pressures versus scan number.

For the purposes of this analysis, it was necessary to derive a single value of ΔP for each epoch, which could then be compared to the mobility record of the same interval. To achieve this, the largest change in pressure $\Sigma(|\Delta P_{max}|)$ was selected. In addition, it would be expected that this would be related to the largest physiological effect, although it was under-

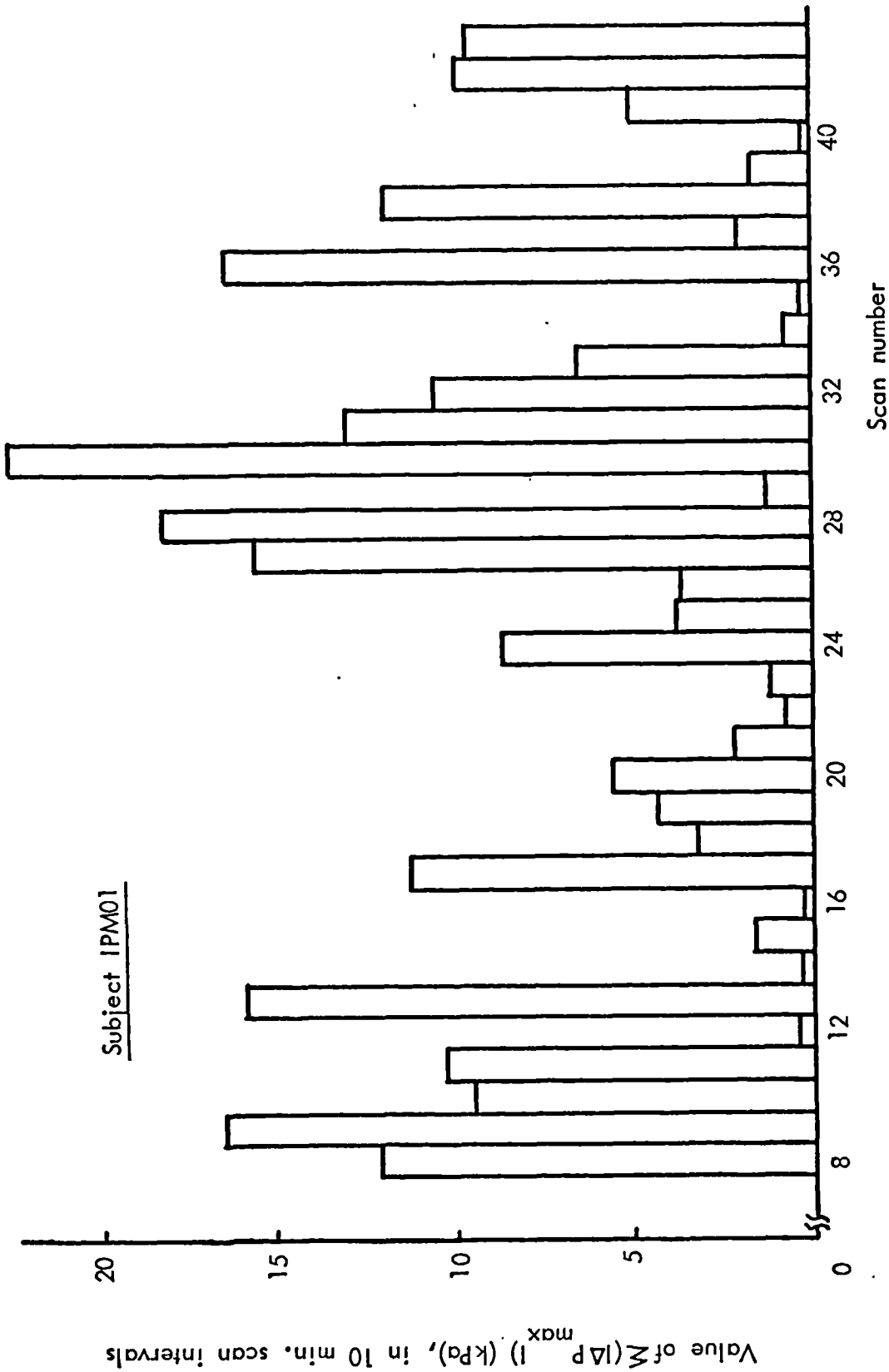


Figure 4.6 Body-support surface interface pressure during a typical night's sleep.

stood that smaller changes in pressure, measured by the other transducers in the matrices could also have some physiological influence.

4.5.3 Comparison of mobility and interface pressure data

The aim of this analysis was to establish the optimum correlation between changes in interface pressure and corresponding changes in mobility. The mobility data was processed in relation to two parameters M_{SL}^{c} and M_{T}^{c}. Each of these was compared separately to the interface pressure data. Thus, at this stage the analysis may be considered as having been in two parts, comparison of interface pressure changes with:

- the magnitude of signal level changes (M_{SL}^{c})
- the magnitude of transients (M_{T}^{c}).

Interface pressure threshold: Selection of threshold pressure change included consideration regarding accuracy and likely experimental artefacts of the monitoring system.

Ferguson-Pell (1977) explains the inaccuracy of the pressure monitoring system as primarily that attributable to the pressure transducers. Calibration of these devices indicated that, due to hysteresis, their output was subject to approximately 2.7 kPa (20mmHg) "uncertainty". Experimental artefacts were related to the bed clothing, flexion of the transducer or residual outputs, due to a raised skin temperature (thermal effect) at the site of the pressure transducer. Experimental results suggested that 1.3 kPa (10mmHg) be considered as the minimum pressure, capable of being measured without significant influence by artefacts.

To avoid inaccuracies of the measuring system, both due to the pressure transducer and due to artefacts, a threshold pressure change of 4 kPa (30 mmHg) was selected.

For analysis $\Sigma(|\Delta P_{max}|)$ within each scan interval was compared to the threshold value. If the pressure change was larger than 4kPa (30mmHg) then it was recorded as 1 and if the change was less than 4kPa (30mmHg) it was denoted as 0 (Table 4.1). In this way, the data was reduced to binary form.

Mobility threshold: The mobility data was treated in a similar manner to the interface pressure data. The use of a threshold value, enabling it to be reduced to binary format. However, unlike the interface pressure, no obvious threshold was apparent and so a range of values were used. All were compared with interface pressure data. This procedure was performed for both M_{SL}^{c} and M_{T}^{c}.

Utilisation of a threshold served to alter the data from a continuous function (M_{SL}^{c} and M_{T}^{c}) with a continuously variable parameter (magnitude), (figures 4.5a and b) to a discontinuous function ($M_{SL}^{c} > T$ and $M_{T}^{c} > T$) with discrete parameters (figures 4.10a and b).

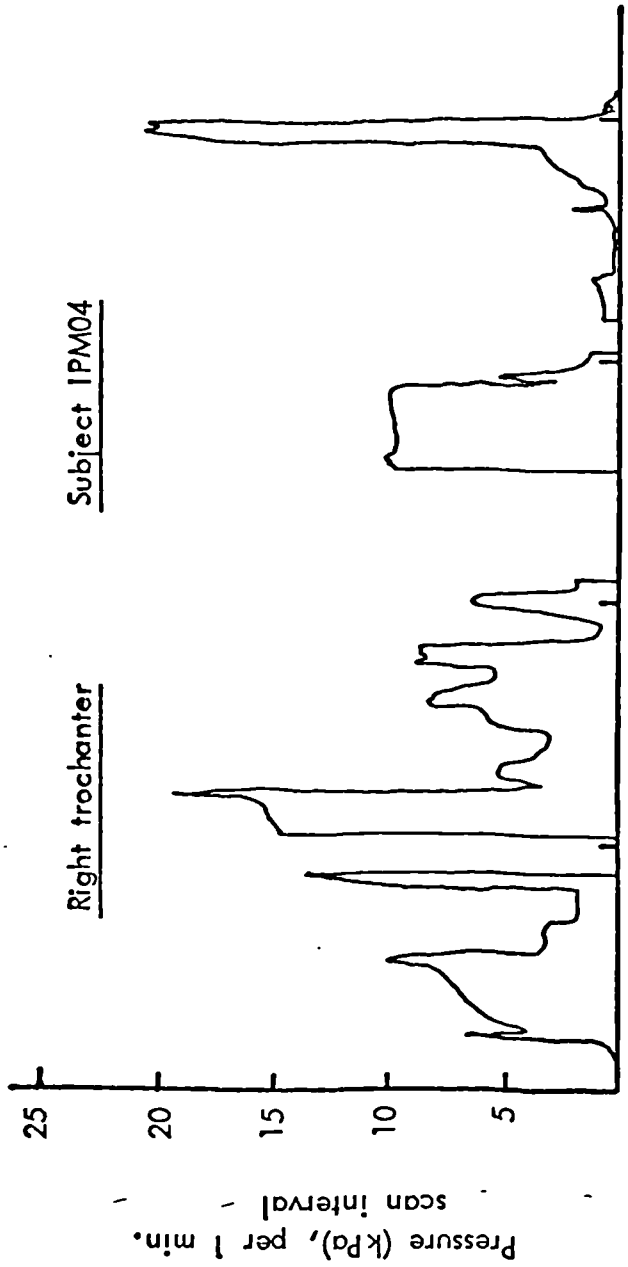


Figure 4.7 Body-support surface interface pressure, measured at 1 minute intervals throughout the night.

4.6 RESULTS

The data produced by the mobility and interface pressure monitoring systems, was analysed as described in section 4.5. After analysis, statistical methods were employed for the comparison of results.

4.6.1 Interface pressure

An example of the data produced by the interface pressure apparatus, using a ten minute scanning interval and analysed as described in section 4.5 is shown in figure 4.6. The fluctuation in pressure can be considered indicative of changes in interface pressure, occurring throughout the course of the night. By reducing the scanning interval to one minute, it was possible to provide more detailed information (figure 4.7). As a contrast with the ten minute scanning interval, this data was analysed with respect to every tenth interval, disregarding the samples inbetween (figure 4.8). Comparison between the two graphs (figures 4.7 and 4.8), indicated that although the one minute scanning interval provided "fine detail", the results obtained using the ten minute scanning interval, yielded information about the major variations in interface pressure. This result confirmed the earlier assumption that the use of a ten minute scanning interval was capable of producing information relating to significant changes in interface pressure (vide 4.4.3).

Data supplied by the interface pressure monitoring apparatus was analysed with reference to a threshold of 4kPa (30mmHg) (vide 4.5.3), (Table 4.1). Figure 4.9 is representative of the data collected relating to subject IPMO1 earlier described in figure 4.6, after threshold analysis.

4.6.2 Mobility

Typical results of the mobility data, analysed with reference to;

- changes in signal level greater than a threshold ($M_{SL}^{c}>T$),

versus scan number are described in Table 4.2a, figure 4.10a.

- the largest transient greater than a threshold ($M_{T}^{c}>T$),

versus scan number are presented in Table 4.2b, figure 4.10b.

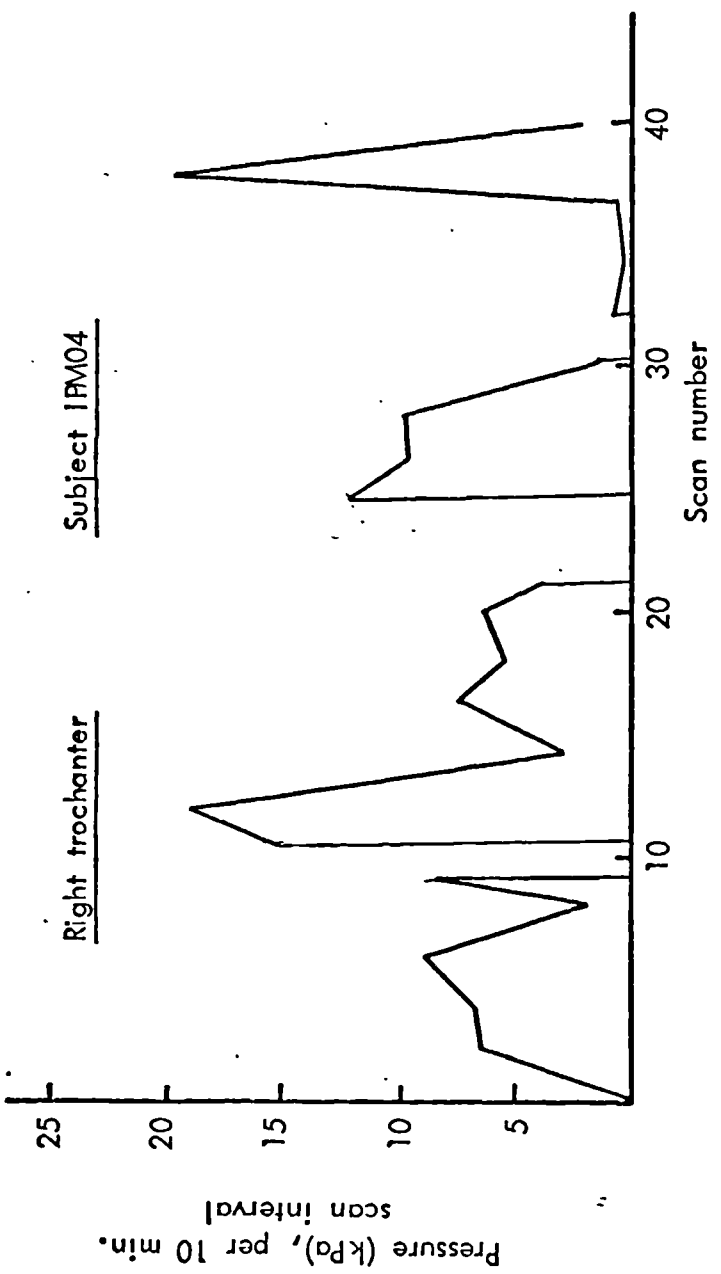


Figure 4.8 Body-support surface interface pressure, measured at 10 minute intervals throughout the night. Data calculated from 1 minute scanning interval test.

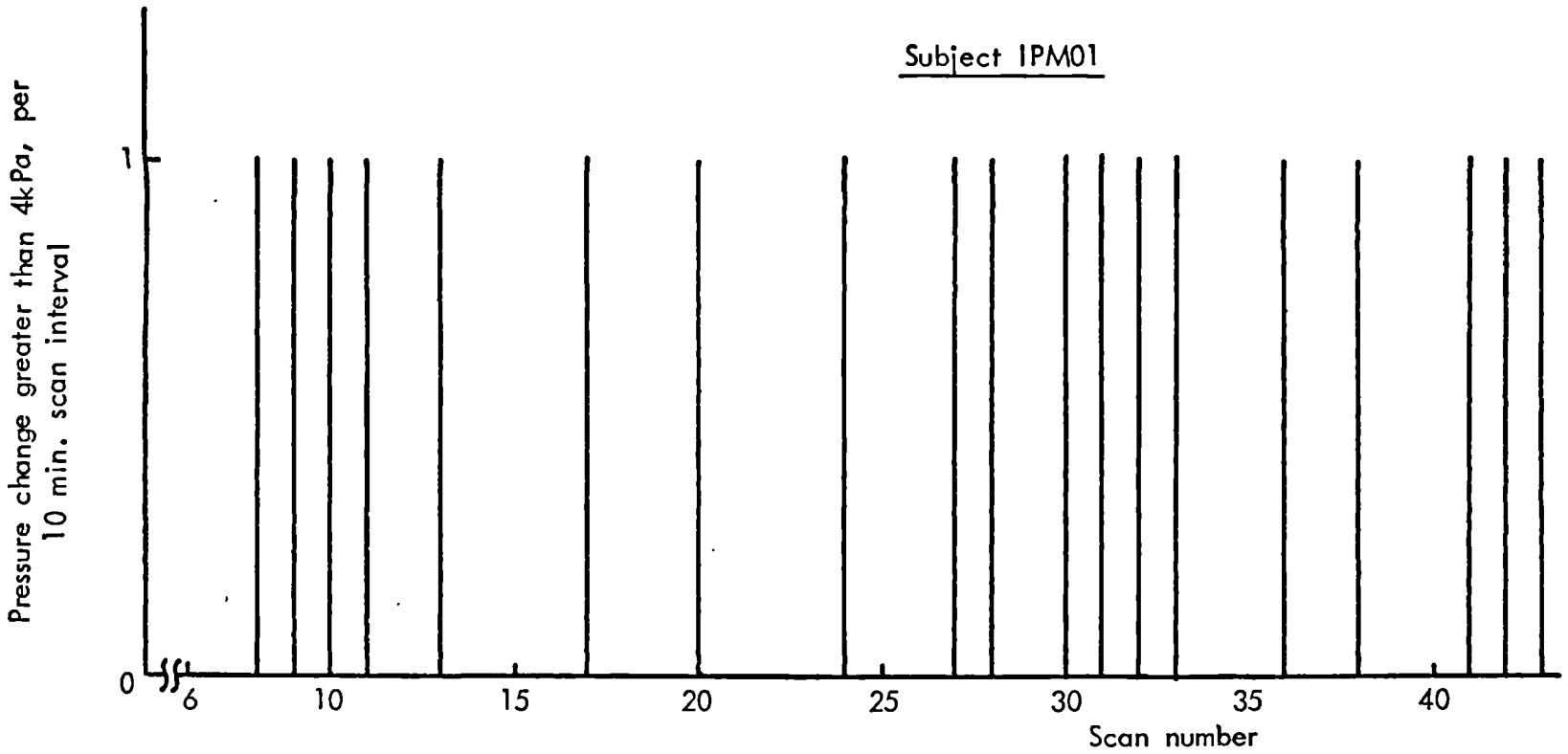


Figure 4.9 Scan intervals containing a pressure change greater than 4 kPa ($\lesssim(|\Delta P_{max}|)>T$).

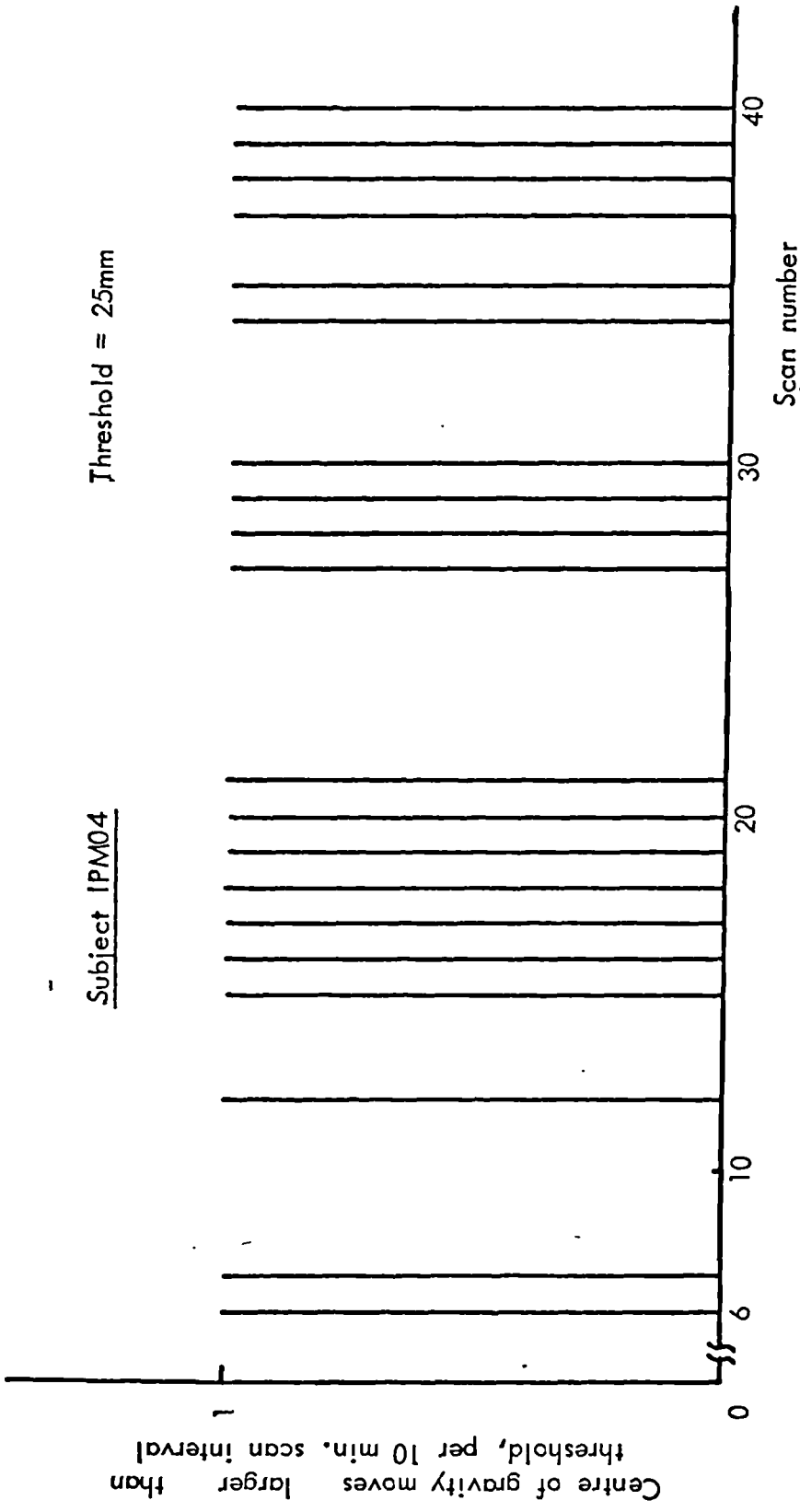


Figure 4.10a Scan intervals containing centre of gravity moves larger than a threshold ($M^c_{SL} > T$).

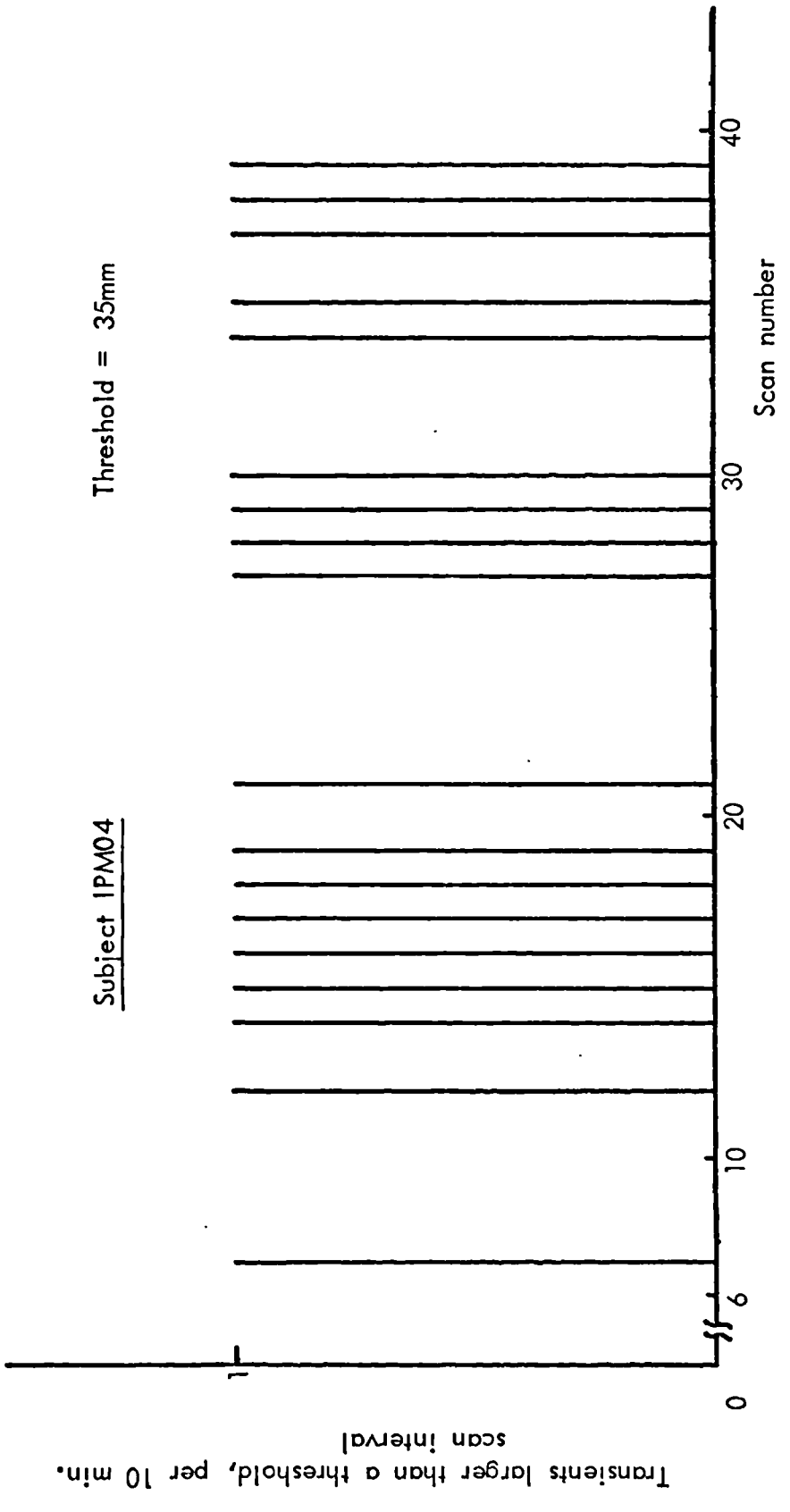


Figure 4.10b Scan intervals containing transients larger than a threshold ($M_T^c > T$)

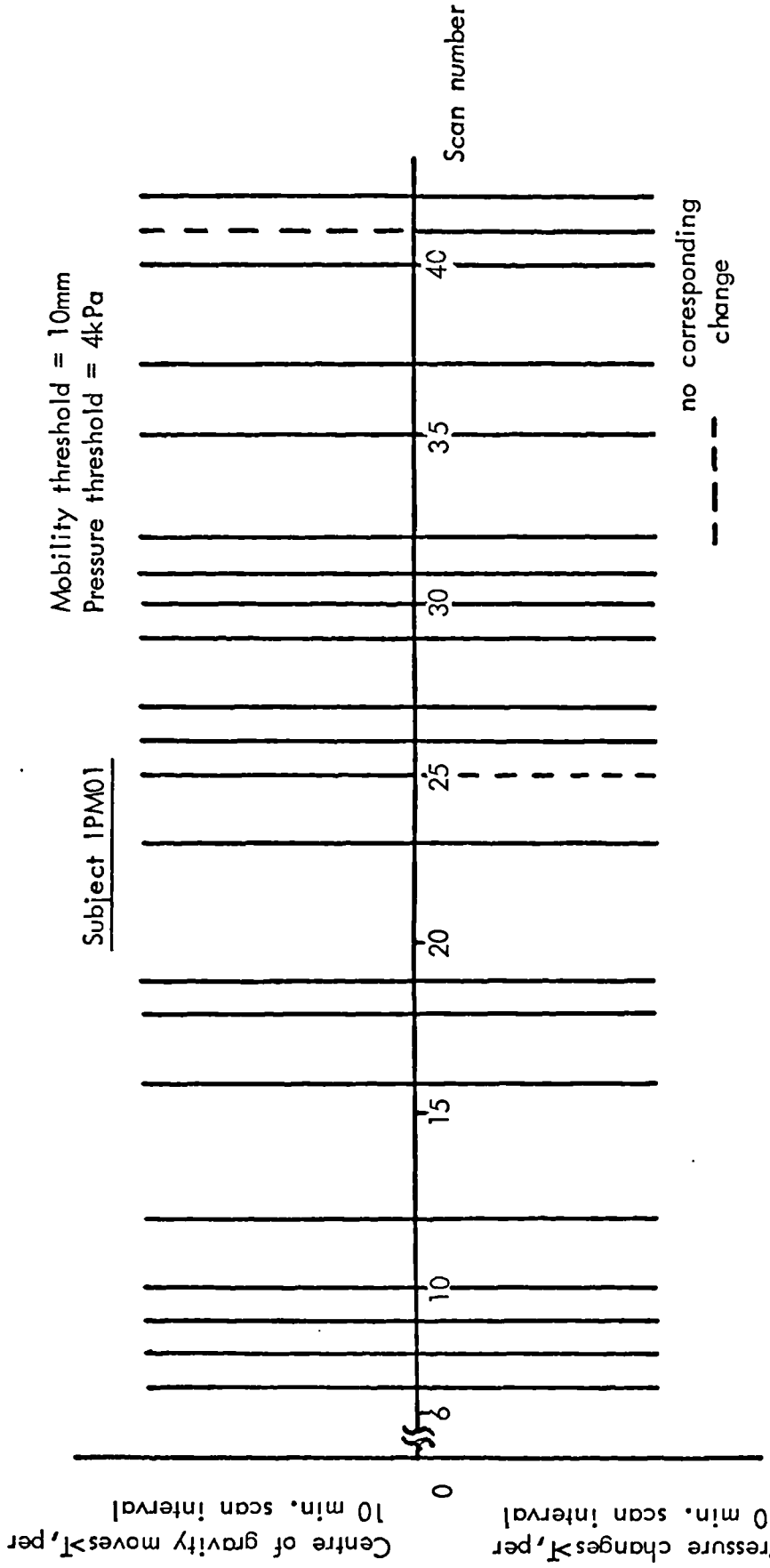


Figure 4.11 Comparison between centre of gravity moves greater than a threshold and interface pressures greater than a threshold, indicating the high correspondence between the two variables.

4.6.3 Interface pressure compared to mobility

Interface pressure and overnight mobility results were compared in the form,

$$\Sigma(\text{I } \Delta \text{ P max I}) > 4\text{kPa} \quad (30\text{mmHg}) \quad \text{to} \quad M_{SL}^{c} > \text{Threshold},$$

$$\Sigma(\text{I } \Delta \text{ P max I}) > 4\text{kPa} \quad (30\text{mmHg}) \quad \text{to} \quad M_{T}^{c} > \text{Threshold}$$

Threshold values, T, selected were; 5, 10, 15, 20, 25 and 30 mm for M_{SL}^{c} and 7.5, 15, 25 and 35mm for M_{T}^{c}.

For each test and each threshold value, graphs displaying $\Sigma(\text{I } \Delta \text{ P max I}) > 4\text{kPa}\ (30\text{mmHg})$ and $M_{SL}^{c} > T$ were drawn. Similarly graphs comparing $\Sigma(\text{I } \Delta \text{ P max I}) > 4\text{kPa}$ (30mmHg) and $M_{T}^{c} > T$ were constructed. Figures 4.11 and 4.12 are typical results for M_{SL}^{c} and M_{T}^{c} respectively.

These graphs provided a visual representation of the relationship between interface pressure and overnight mobility.

The assessment of the statistical significance of any relationship between the two sets of results was achieved by the use of χ^2 tests. This non-parametric statistic was selected because of the binary format of the analysed data, which precluded the use of other, parametric tests.

The interface pressure and mobility results of each experiment were summarised in a 2 x 2 matrix (contingency table) with the general format

	P_0	P_1	
M_0	O_{00}	O_{01}	R_0
M_1	O_{10}	O_{11}	R_1
	C_0	C_1	S

where M_0 = no change in mobility greater than the threshold

M_1 = a change in mobility greater than the threshold

P_0 = no change in interface pressure greater than 4kPa(30mmHg)

P_1 = a change in interface pressure greater than 4kPa(30mmHg)

O_{00} = the number of simultaneous occurrences of M_0 and P_0 in a scan interval.

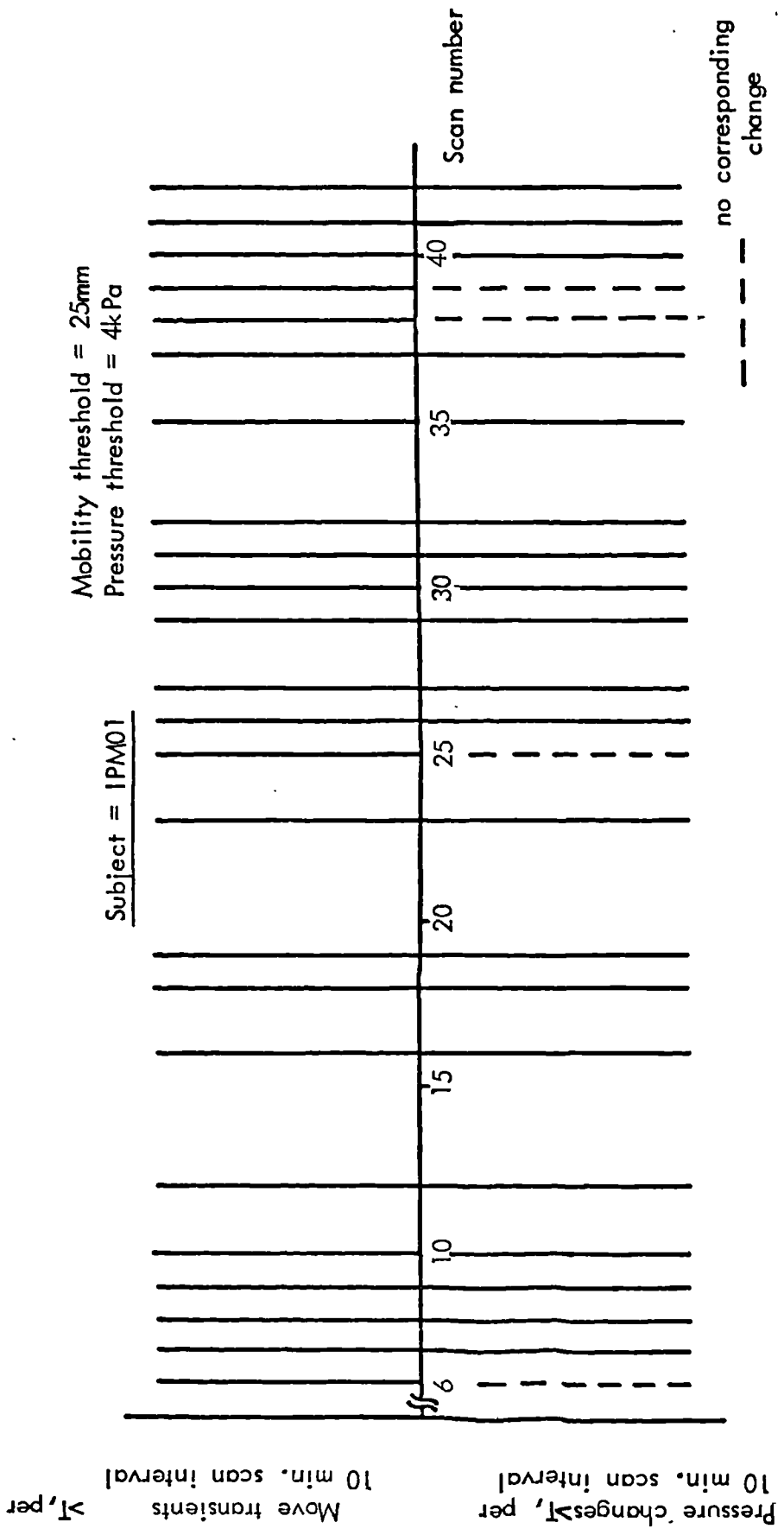


Figure 4.12 Comparison between transient move size larger than a threshold and interface pressures greater than a threshold, indicating a high correspondence between the two variables.

O_{01} = the number of simultaneous occurrences of M_0 and P_1 in a scan interval.

O_{10} = the number of simultaneous occurrences of M_1 and P_0 in a scan interval.

O_{11} = the number of simultaneous occurrences of M_1 and P_1 in a scan interval.

$R_0 = O_{00} + O_{01}$

$R_1 = O_{10} + O_{11}$

$C_0 = O_{00} + O_{10}$

$C_1 = O_{01} + O_{11}$

$S = R_0 + R_1 = C_0 + C_1$

The χ^2 statistic was then represented as

$$\frac{(\text{Observed frequency} - \text{Expected frequency})^2}{\text{Expected frequency}}$$

or, in general terms

$$\frac{\left(O_{ij} - \frac{R_i \times C_i}{T}\right)^2}{\frac{R_i \times C_i}{T}}$$

By this means a χ^2 value was obtained for each element of the contingency table, the overall χ^2 being the sum of these.

In the application of the χ^2 test it was desirable that each of the elements of the matrix be greater than 5, to prevent unwarranted biasing of the statistic (Mounsey, 1964). To avoid this difficulty the results from all of the experiments were pooled together (Tables 4.3a and b).

Thus, contingency tables were constructed comparing Σ(I Δ P max I) > 4kPa (30mmHg) with ($M^c_{SL} > T$) and Σ(I Δ P max I) > 4kPa (30mmHg) with ($M^c_I > T$) for varying thresholds.

The results of the χ^2 tests, indicated that all of the paired comparisons

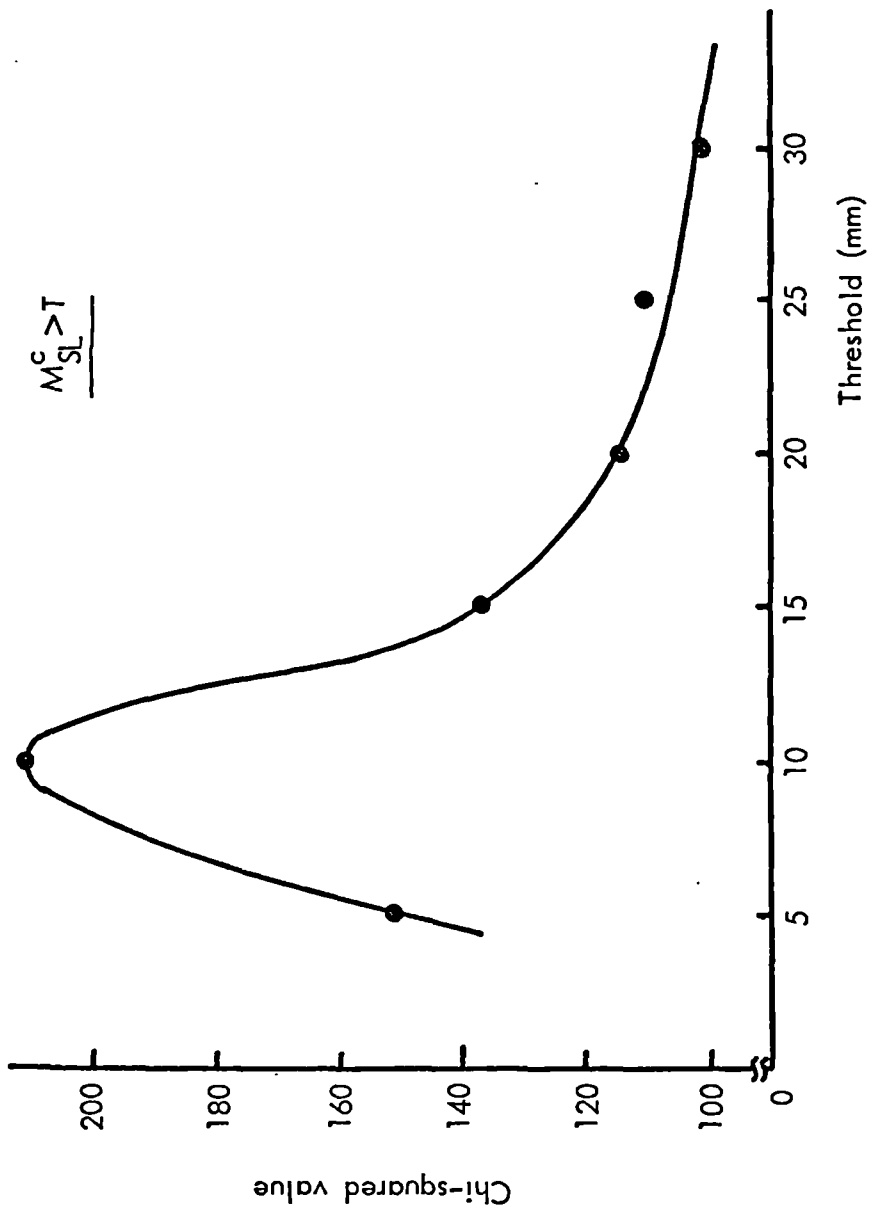


Figure 4.13a Chi-squared statistic comparing interface pressure changes with centre of gravity movements greater than a range of thresholds.

were significant($p < 0.001$) (Tables 4.4a and b). To assess the optimum combination of mobility parameter and threshold, that produced the largest degree of association with changes in interface pressure greater than 4kPa (30mmHg), the data was plotted (figure 4.13a and b).

The shape of the curves, with a single maximum, suggested the selection of an optimum threshold level for each variable. From the graphs, values of 10mm and 25mm were chosen for M^c_{SL} and M^c_T respectively.

Comparison of the two mobility parameters was accomplished by examining the association coefficients calculated for each of them. The greatest of these being indicative of the optimum movement parameter. From Tables 4.4a and b for $M^c_{SL} > 10$mm, the coefficient was 212 and for $M^c_T > 25$mm it was 175. Thus, the greatest correspondence between changes in interface pressure larger than 4kPa (30mmHg) measured at sites around the pelvis, and overnight mobility was achieved by analysing the mobility record with reference to changes in signal level of 10mm or greater.

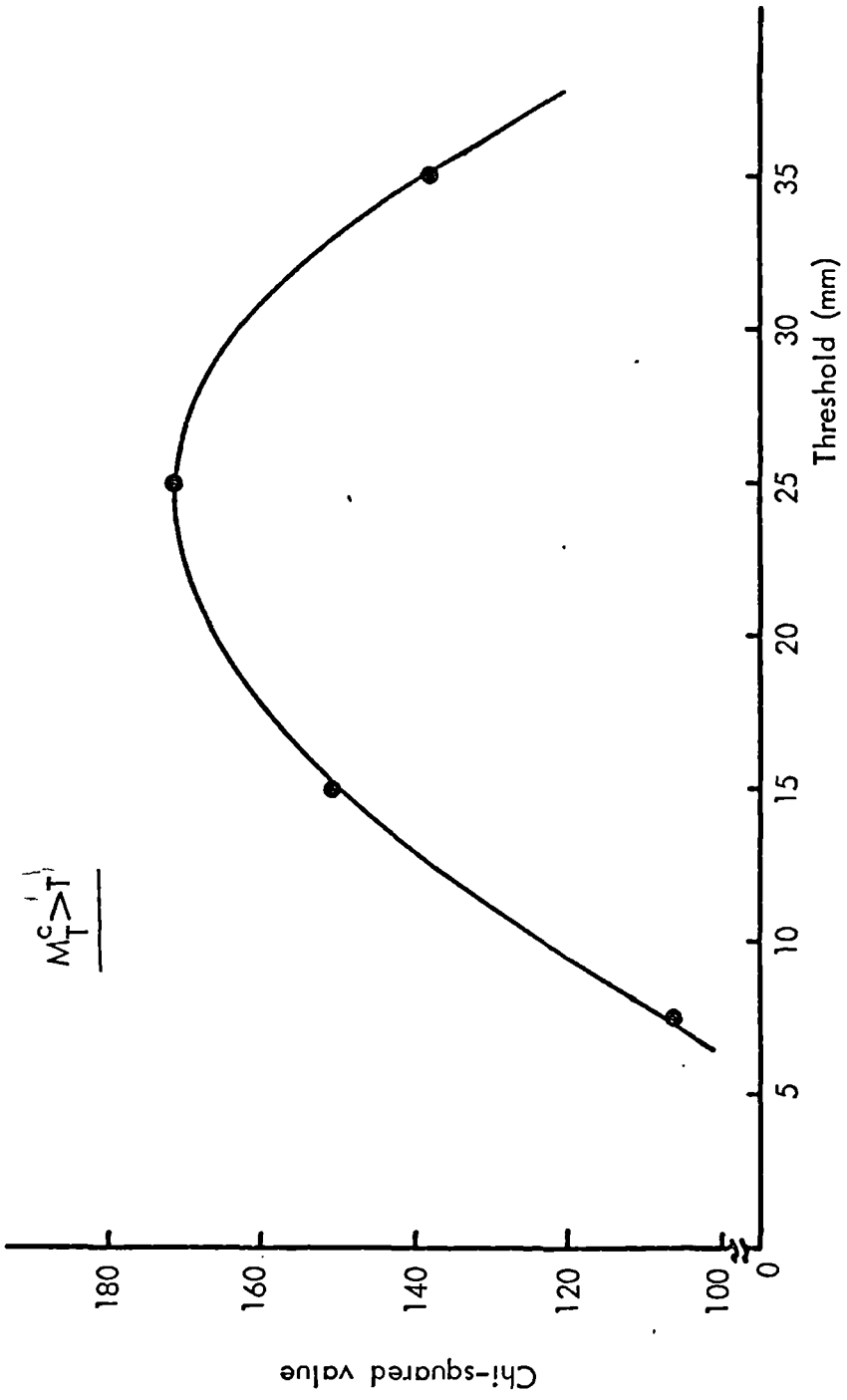


Figure 4.13b Chi-squared statistic comparing interface pressure changes with transient move sizes greater than a range of thresholds.

	Pressure (kPa)				
	$(\lvert\Delta P\rvert)_{max}$			$\Sigma(\lvert\Delta P_{max}\rvert)$	
Scan no.	Sacrum	Trochanter	L. Trochanter	Total	> 4kPa
↓					
20	1.1	2.9	1.7	5.7	1
21	2.0	0.1	0.0	2.2	0
22	0.8	0.0	0.0	0.8	0
23	0.9	0.1	0.0	1.0	0
24	4.2	3.7	0.9	8.8	1
25	3.3	0.1	0.2	3.6	0
26	1.7	1.9	0.2	3.8	0
27	2.8	5.0	7.6	15.4	1
28	13.9	0.4	3.8	18.1	1
29	1.2	0.0	0.2	1.4	0
30	12.5	8.7	3.3	24.6	1
31	6.4	6.3	0.3	13.0	1
32	6.2	3.3	0.9	10.4	1
33	0.6	4.5	1.3	6.4	1
34	0.4	0.3	0.1	0.8	0
35	0.1	0.1	0.1	0.3	0
36	5.7	9.8	0.6	16.1	1
37	1.8	0.4	0.1	2.3	0
38	6.4	4.2	1.1	11.7	1
39	0.4	0.6	0.6	1.6	0
40	0.0	0.1	0.1	0.2	0

Table 4.1 Typical interface pressure readings recorded at the sacrum and trochanters, combined to give $\Sigma(\Delta P_{max})$ and compared to the 4kPa threshold. Sample from subject IPM01.

Scan no..	Size of change in signal level (M_{SL}) (mm) X	Y	$M_{SL}^{c}\left(\sqrt{(X^2+Y^2)}\right)$	Threshold (mm) 5	10	25
↓						
12	48	7	48.5	1	1	1
13	8	2	8.2	1	0	0
14	6	18	19.0	1	1	0
15	69	3	69.1	1	1	1
16	84	9	84.5	1	1	1
17	7	102	102.2	1	1	1
18	13	137	137.6	1	1	1
19	158	4	158.1	1	1	1
20	103	9	103.4	1	1	1
21	106	5	106.1	1	1	1
↓						

Table 4.2a Example of analysis of the mobility data. Change in signal level of channels X and Y, combined to give M_{SL}^{c} and compared to typical threshold levels. Sample data from subject IPM04.

Scan no.	Size of peak to peak transients (M_T) (mm) X	Y	M_T^c $\left(\frac{X+Y}{2}\right)$	Threshold (mm) 7.5	25	35
↓						
12	142	19	80.5	1	1	1
13	29	12	20.5	1	0	0
14	65	43	54.0	1	1	1
15	100	22	61.0	1	1	1
16	90	29	59.5	1	1	1
17	46	47	46.5	1	1	1
18	110	99	104.5	1	1	1
19	110	88	94.0	1	1	1
20	38	28	33.0	1	1	0
21	108	20	64.0	1	1	1
↓						

Table 4.2b Example of analysis of the mobility data. Peak to peak transient values for channels X and Y, combined to give M_T^c and compared to typical threshold levels. Sample data from subject IPM04.

Category	Test number									
	IPM01	IPM02	IPM03	IPM04	IPM05	IPM06	IPM07	IPM08	IPM09	Total
O_{00}	17	18	21	8	16	22	13	15	16	146
O_{01}	1	3	0	1	2	0	1	3	3	13
O_{10}	1	3	5	1	2	2	0	2	3	19
O_{11}	19	17	8	23	14	8	22	19	19	149
Sample no.	38	41	34	33	34	32	33	39	40	327

Table 4.3a Example of the combined interface pressure/mobility results for each of the subjects grouped together.

For M_{SL}^{c}, threshold = 10mm.

Category	Test number									
	IPM01	IPM02	IPM03	IPM04	IPM05	IPM06	IPM07	IPM08	IPM09	Total
O_{00}	14	15	15	8	14	20	13	12	16	127
O_{01}	0	0	0	4	1	0	3	1	1	10
O_{10}	4	6	9	1	4	2	0	5	3	34
O_{11}	20	20	9	20	15	8	19	21	20	152
Sample no..	38	41	33	33	34	30	35	39	40	323

Table 4.3b Example of the combined interface pressure/mobility results for each of the subjects, grouped together.

For M_T^c , threshold = 25 mm.

Threshold(T)(mm)	x^2 Value
5	154.7
10	211.7
15	166.5
20	117.2
25	127.1
30	100.9

Table 4.4a Results of the x^2 tests for different thresholds (T) between mobility, characterised as a change in signal level greater than a threshold ($M_{SL}^{c}>T$) and interface pressure. All coefficients are significant ($p<0.001$)

Threshold (T) (mm)	χ^2 Value
7.5	103.7
15	133.0
25	174.9
35	138.1

Table 4.4b Results of the χ^2 tests for different thresholds (T), between mobility, characterised as peak to peak transients greater than a threshold ($M_T^c > T$) and interface pressure. All χ^2 coefficients are significant ($p<0.001$).

4.7 SUMMARY

Quantitative comparison was conducted between two of the variables commonly accepted as of importance in the aetiology of pressure sores namely mobility and interface pressure. Nine young normal subjects were studied. Apparatus designed for a previous application (Bardsley, 1977) and Ferguson-Pell, 1977) were utilised, to make simultaneous recordings of a subjects overnight mobility and their body-support surface interface pressure, measured at the trochanters and sacrum. The pressures measured at each of these sites were combined together. Inaccuracies and artefacts associated with the pressure monitoring system suggested that the minimum reliable magnitude of the combined pressure change was 4kPa (30mmHg). In general, the interface pressures were scanned and recorded at ten minute intervals. A pilot study, involving a reduced rate of scanning interval (one minute) produced a more detailed record of changes in interface pressure. However, results from the ten minute scans were shown to illustrate all of the major changes in pressure.

To obtain the optimum correlation with the interface pressure data, the mobility record was analysed with respect to two parameters. Firstly, changes in signal level and secondly, the peak to peak magnitude of transients in the signal. A range of mobility threshold values were established for use in comparison with that of the interface pressure system.

The results from both the mobility and interface pressure apparatus were analysed in 10 minute epochs and manipulated into binary form to permit comparison between the real time record of mobility as opposed to the discontinuous nature of the pressure recording. Comparison was then made between changes in interface pressure larger than 4kPa (30mmHg), and variations in mobility larger than a threshold, for each of the characterisation parameters, by utilising statistical analysis. Results suggested that changes in signal level of 10 mm were capable of providing at least a 4kPa (30mmHg) change in combined interface pressure.

This result provided quantitative evidence to indicate that changes in mobility were closely linked to concurrent variations in interface pressure.

The magnitude of the mobility threshold served to emphasise the claim of Fernie (1973) that relatively small changes in position can produce significant variations in interface pressure.

CHAPTER 5

MOBILITY MONITORING APPARATUS

5.1 INTRODUCTION

Chapter 2 discussed the importance of sustained pressure applied to the soft tissues of the body, the particular susceptibility of the elderly to pressure sores and introduced the rationale of a study relating to measurement of the interface pressures of elderly patients in hospital. Experimental techniques to measure interface pressures between the body and the support surface require the attachment of transducers to the body (Kosiak, 1961; Houle, 1969; Ferguson-Pell, 1977), or the use of a modified support surface (Lindan, 1961). The discomfort and disruption to normal nursing procedures caused by the use of such devices, especially when they are connected to associated electronic apparatus, precludes their use for long term monitoring of patients in the clinical environment.

The data from Chapter 4 illustrated that there was a strong correlation between variations in interface pressure and changes in position of a subject lying on a bed. This was observed as an alteration in signal level, representative of a change in position of the subject's centre of gravity and a fluctuation in interface pressures, measured at the sacrum and trochanters. A threshold level of move size was determined, which produced the closest correlation, with a concomitant change in interface pressure. Thus, by monitoring the mobility of a subject, it is possible to obtain information relating to changes in interface pressure, without the need to attach transducers directly to the patient. To achieve this, the concept of monitoring movement by placing force transducers beneath the legs of the patient's bed was developed by Bardsley (1977), and modified and refined in this study to enable the mobility of elderly hospital patients to be monitored during sleep.

5.2. REQUIREMENTS OF THE MOBILITY MONITORING APPARATUS

To provide a basis for selection of the monitoring technique, it was firstly necessary to define the overall requirements and specifications of such a system and using these, to select a design principle.

5.2.1 System specifications

- The device was to be suitable for routine use in the clinical environment, most normally an open-plan ward in one of the local hospitals.
- The output of the monitoring system was to contain information relating to the magnitude and time of occurrence of a movement of a patient's centre of gravity in bed.
- The device was not to be attached to the patient or interfere with normal nursing of clinical procedures.
- The device was not required to be permanently affixed to a bed, but easily transferable, to allow the monitoring of a patient in any bed of the ward.
- The system was required to be capable of monitoring patients over a weight range of 30 kg to 110 kg. In addition the output of the system was to be independent of the patient's body weight.
- The output format of the system was to be electrical in nature, providing for the use of automatic data recording followed by computational analysis.
- The system was to be capable of continuous operation for the period of a night (typically 10 hours).

5.2.2 Evolution of system design

Section 3.4 reviewed previous techniques that have been developed to measure body movements during sleep. However, these do not fulfil all of the required specifications for this application (vide 5.2.1).

Those techniques employing the principle of "measurement of the displacement of all or part of the bed" have usually monitored the deflection of the mattress or bedsprings at one position only, therefore not providing a

uniform sensitivityoverall of the bed surface. Also, systems of this design require, in general, special fixtures to the bedframe, restricting their use to a specific bed.

Methods utilising "measurement of body-section displacement" involve the attachment of transducers to the patient and are considered potentially invasive, the presence of the transducers leading to artefactual data, and inconvenience to the patient.

The principle of "observation" of a patient to assess their mobility is applicable to any bed and requires no attachments to the body. However, the use of cine or still cameras, with their mandatory peripherals, precludes the method from routine use in the clinical environment. In addition, the output is not electrical in nature.

Mobility monitoring systems utilising "measurement of the mechanical forces generated in the support structure" have sometimes employed an "instrumented bed" and as a result have been restricted to a particular bed. Alternative systems which have been developed employing this principle, and that are suitable for use on a range of beds, have either produced an output biased towards a particular body section or have required extra attachments which cannot feasibly be regularly employed in a clinical environment.

Techniques employing "ultrasonic radiation" are, generally, restricted to recording the occurrence of a move only and provide no information as to the magnitude of the move. This limitation precluded them from use in this study.

Other "indirect" techniques are either too approximate, (static charge sensitive bed, water displacement mattress) or too complex (EEG), requiring the attachment of multiple electrodes allied with sophisticated monitoring equipment, to be of use in this application.

However, although none of the existing systems was capable of satisfying the design specification, the technique developed by Fernie (1973) and Bardsley (1977), (vide 4.2) utilising measurement of the reaction forces between the ground and the bedleg, appeared to offer many of the features required, but was limited in several ways for routine clinical research

purposes. The force transducers, placed beneath the legs of the patient's bed, were commercially produced and were not of a suitable profile to maintain the normal stability of the bed. Since only two transducers were used, the system was not independent of the patient's weight. A substantially modified force transducer system was prepared for this study, utilising the experience gained from Bardsley to develop an apparatus that would overcome the limitations of his system capable of routine use in the clinical environment.

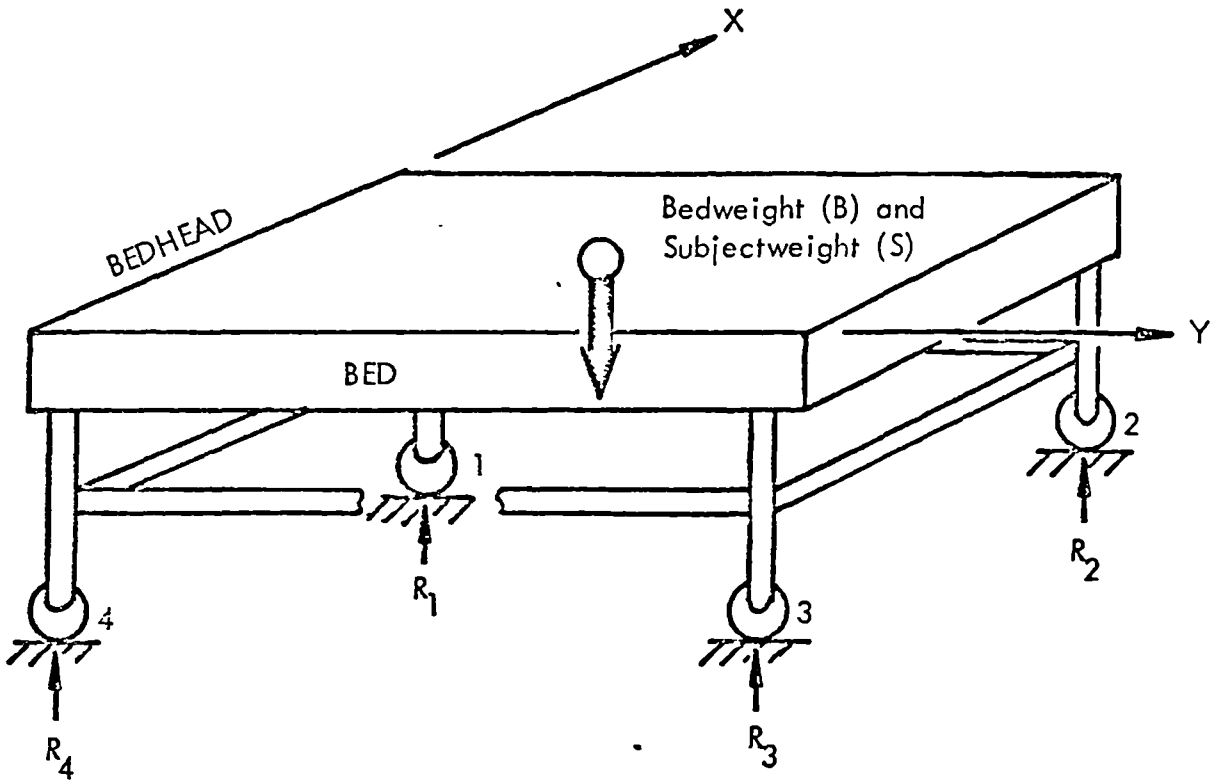


Figure 5.1 Reaction forces (R) at the bedlegs as a result of the bedweight (B) and the subjectweight (S).

5.3 THEORY OF A 4 POINT SUPPORT SYSTEM

Calculation of the load in the legs of a bed may be determined by applying the principles of action and reaction in combination with that of equilibrium (vide 3.4). In practical terms this means that all forces, static or dynamic which are a result of the bed and the subject in the bed, will be transmitted to the ground by any supporting members, these normally being the legs of the bed. The use of force transducers positioned between the ground and the bedleg will give an output representative of the load in that leg due to the weight in the bed. If identical force transducers are placed beneath all four bedlegs, then the sum of their outputs will be a measure of the total weight of the bed. However, due to the differing distribution of weight within the bed, the output from all transducers may not be equal. When a subject moves in the bed, the distribution of weight will be modified, and provided the force transducers are linear, this will result in a directly proportional change in their output. Using the measure of force provided by these transducers, it is possible to identify the position of the centre of gravity of a subject in the bed.

5.3.1 Analysis of forces for a 4 point support system

The bed and subject were considered to have weights B and S respectively. These produced reaction forces R between the floor and each bedleg of R_1, R_2, R_3 and R_4 (figure 5.1).

The bedweight was considered to have components B_1, B_2, B_3 and B_4 at each of the bedlegs. Similarly the subject's weight provided components S_1, S_2, S_3 and S_4. Thus at any instant, the reactive forces were a combination of the two,

$$R_1 = B_1 + S_1$$
$$R_2 = B_2 + S_2$$
$$R_3 = B_3 + S_3$$
$$R_4 = B_4 + S_4$$

Selecting bedleg 4 to be the origin of a coordinate system (figure 5.1), $(x_1, 0)$, (x_2, y_2), $(0, y_3)$ and $(0, 0)$ describe the positions of bedlegs 1, 2, 3

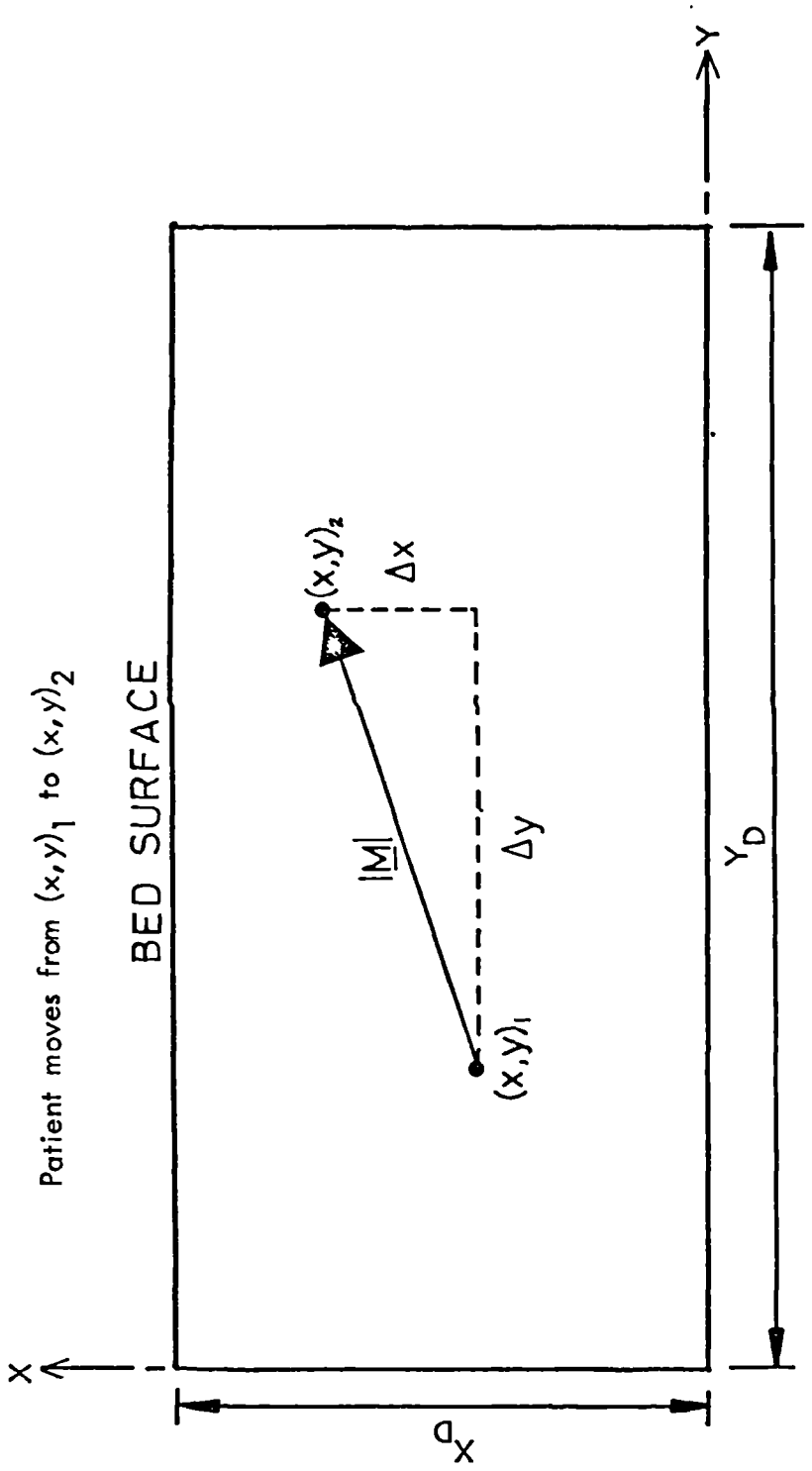


Figure 5.2 An illustration of the magnitude of the movement vector $\underline{M}$, representing the change in position of the patient on the bed surface.

and 4 respectively. In a similar manner the position of the centre of gravity of the bed and of the subject were (x_B, y_B) and (x_s, y_s) respectively.

The magnitude of a move by the subject in the bed, was calculated to be the size of the change in position of the subject's centre of gravity, i.e. $\Delta x_s + \Delta y_s$. The movement vector $\underline{M}$, a measure of the magnitude of the move was calculated as the vector sum (figure 5.2).

$$|\underline{M}| = \sqrt{\Delta x_s^2 + \Delta y_s^2} = |\underline{x}_s + \underline{y}_s| \qquad (5\text{-}1)$$

Assumptions: It was assumed, for the purposes of this analysis, that the bedweight was transmitted to the ground through the bedlegs only and that it was equally distributed between supports with $B_1 = B_2 = B_3 = B_4$, the bed structure was completely rigid and that the force transducers were linear, with identical characteristics measuring only the normal component of force.

Any errors arising from an uneven floor or an unequal distribution of the weight, although ignored in this analysis, may occur in practice. However, the electronic processing involved in the monitoring system (figure 5.3), accounted for this by offsetting (zeroing) the bedweight before the patient was in the bed ($B_1 = B_2 = B_3 = B_4 = 0$). In this way the system became effectively independent of the bedweight and of any unequal distributions associated with it.

Analysis: Consider the situation when the subject is off the bed ($S = 0$)

Moments about the X axis,

$$\sum M_x = B_2y_2 + B_3y_3 - By_B = 0 \qquad (5\text{-}2)$$

Moments about the Y axis,

$$\sum M_y = B_2x_2 + B_1x_1 - Bx_B = 0 \qquad (5\text{-}3)$$

With the subject in bed ($S \neq 0$)

$$\sum M_x = R_2y_2 + R_3y_3 - By_B - Sy_s = 0 \qquad (5\text{-}4)$$

and $$\sum M_y = R_2x_2 + R_1x_1 - Bx_B - Sx_s = 0 \qquad (5\text{-}5)$$

Equating similar terms (i.e. equations 5-2 and 5-4),

$$B_2y_2 + B_3y_3 - By_B = R_2y_2 + R_3y_3 - By_B - Sy_s$$

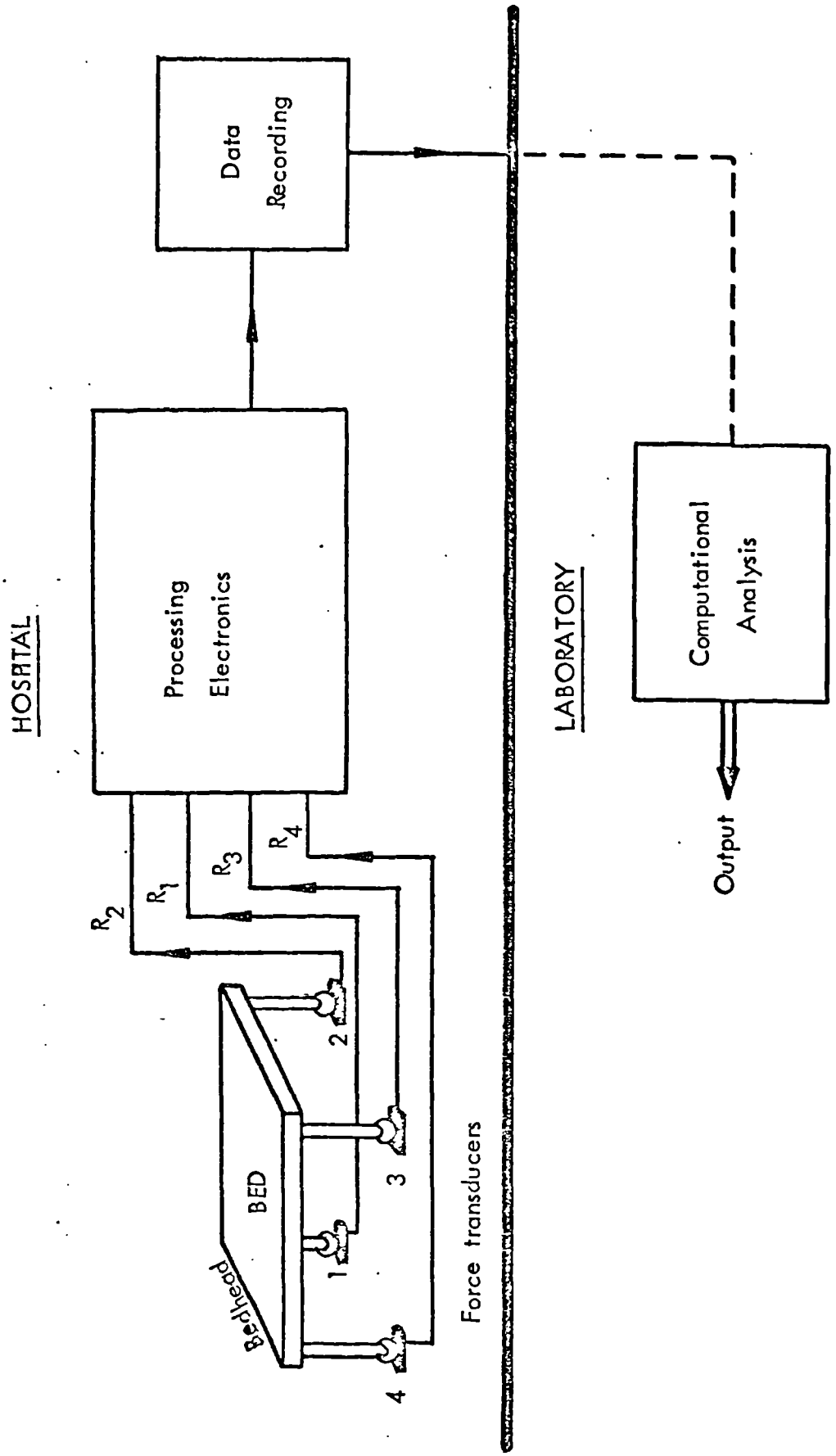


Figure 5.3 A representation of the major elements of the mobility monitoring system, in the hospital and in the laboratory.

after simplification $y_s = \frac{S_2 y_2 + S_3 y_3}{S}$ (5-6)

Equating equations 5-3 and 5-5

$$B_2 x_2 + B_1 x_1 - B x_B = R_2 x_2 + R_1 x_1 - B x_B - S x_s$$

after simplification $x_s = \frac{S_1 x_1 + S_2 x_2}{S}$ (5-7)

However, $y_2 = y_3 = Y_D$ the length of the bed and $x_1 = x_2 = X_D$ the width of the bed (figure 5.2).

Thus equation 5-6 may be rewritten as $y_s = \frac{(S_2 + S_3).\ Y_D}{S}$

and equation 5-7 as $x_s = \frac{(S_1 + S_2).\ X_D}{S}$

We require the resultant of the movement vector $\underline{|M|}$ which from equation 5-1 is

$$\underline{|M|} = \sqrt{\Delta x_s^2 + \Delta y_s^2}$$

X_D, Y_D and S are constants for any particular case.

Thus $$\underline{|M|} = \frac{\sqrt{\left((\Delta (S_1 + S_2)) \cdot X_D\right)^2 + \left((\Delta (S_2 + S_3)) \cdot Y_D\right)^2}}{S} \quad (5\text{-}8)$$

The subject weight S was calculated as $S_1 + S_2 + S_3 + S_4$, the use of four load cells providing for a weight independent system.

Thus, a single channel output device, using the relationship between the load cell outputs expressed in equation 5-8, was designed to measure the size of the movement vector generated by elderly patients during sleep, in the clinical environment. The equipment developed comprise of three elements (figure 5.3):

Force transducers, are placed beneath each bedleg to monitor the reaction force.

Processing electronics, to manipulate the output from the force transducers into a form representative of equation 5-8.

Data recording, to collect and store the data for future analysis.

5.4 DESIGN AND DEVELOPMENT OF A SUITABLE FORCE TRANSDUCER

The force transducers formed the first stage of the system and they were required to be placed underneath the legs of a hospital bed. To aid in the choice of a force transducer, a set of design criteria specific to this application, was established.

5.4.1 Design criteria

Size: Placing force transducers underneath the bedlegs, increases the height of the bed, complicating the nursing routine, particularly when transferring the patient in and out of the bed. For this reason, and to ensure a stable support for the bed, the transducers were required to be of low-profile. It was also desirable that they be as compact as possible, to avoid any obstruction to the hospital staff as they moved around the bed.

Coupling: In the hospital wards in which the work was to take place, all the beds were of the "Kings-Fund" design (Nesbit and Evans Co. Ltd., U.K.) with a castor wheel at the end of each of the bedlegs. As a result the transducers required a reliable coupling mechanism to the wheels.

Although the bed castors were supplied with brakes, on occasion these were found to be faulty and the wheel capable of rotating. Thus, the coupling mechanism was required to be able to prevent the bed simply "rolling off" the transducer. Additionally, the coupling was not to be permanent in nature, allowing the use of any bed in the ward for monitoring. Finally, if the force transducer was position-sensitive, the coupling was required to restrain the wheel repeatably at the same point.

Mechanical strength: When in use overnight, to monitor the force in a bedleg, each force transducer was supporting approximately a quarter of the weight attributable to the combination of bed and patient. However, during the day it was possible that additional weight would be added to this, in the form of hospital staff or relatives sitting on the bed. For this reason, it was necessary to accommodate a significant safety factor when selecting or designing a suitable transducer.

The Kings-Fund design of bed weighed approximately 100 kg without

a mattress and bedclothes. Therefore inclusive of safety factor ($\approx$1.8), the total weight of the bed and clothing was estimated to be 200 kg. The maximum anticipated patient weight was 100 kg and provision was made for three such persons to be on the bed simultaneously. Combination of the loads led to the requirement of 5.0 kN to be supported by the four bed legs.

In the ideal situation each bedleg would be supporting a quarter of the total load (1.25 kN). However, to allow for an unequal distribution of weight, eg. someone sitting at one end of the bed, each force transducer was required to be capable of supporting 2.0 kN.

If the loading on the transducer was large enough to produce collapse it was important that this would not result in a large displacement of the bed, startling or even injuring the patient. As a result the selected force transducer was required to be of low profile so that in the advent of overloading, leading to failure, only a small displacement would occur.

Environmental protection: The force transducer, and connections to it, were required to be sufficiently well protected to prevent damage by extraneous factors. The clinical environment is particularly demanding and the transducer required protection against a wide range of factors, from spilt fluids (water, urine, etc),to accidental kicking or knocking by the hospital staff.

Characteristics: To prevent anomalies and provide an accurate assessment of the force in the bedleg, the transducer was required to be responsive to uniaxial loading exclusively, with a minimum of cross-effects. The castor wheel at the end of the bedleg precluded any idealistic "point loading" and so the force transducer needed to have a large "active loading surface" and its response was required to be consistent, independent of the position of loading on this surface. To allow electronic processing it was necessary for the signal output to be electrical in nature. The transducer was not to be affected by environmental conditions, such as temperature and humidity changes. Finally, the force transducer was required to have sufficient sensitivity to allow the small changes of force in each bedleg, resulting from a movement of the subject in the bed, to be measured.

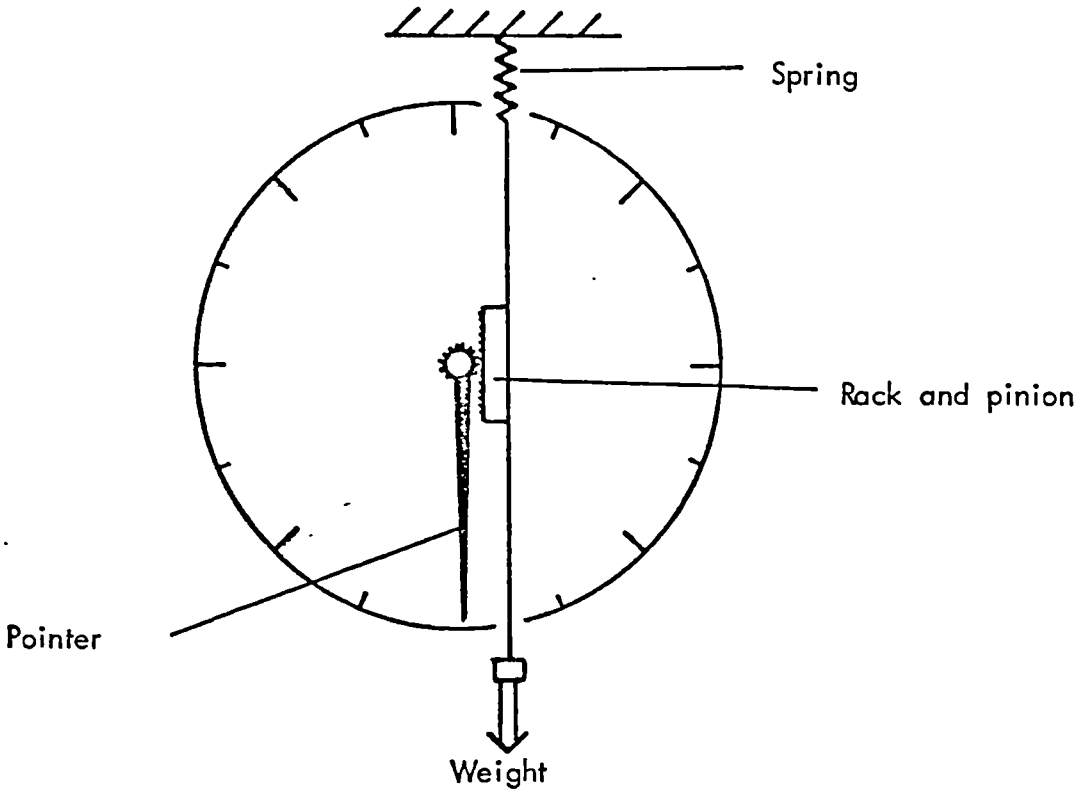


Figure 5.4 Use of a spring with direct readout to measure force(weight).

5.4.2 Conventional force transducer designs

The use of force transducers is widespread in all fields of science and engineering and there are a large number of devices available for this purpose, employing a range of different measurement principles.

Mechanical: Transducers included within this category are based upon the principle of "calibrated deflection" of a mechanical element (spring, cantilevered beam etc). The magnitude of the deflection provides an estimate of the applied force. Measurement of the deflection is commonly achieved by one of two methods:

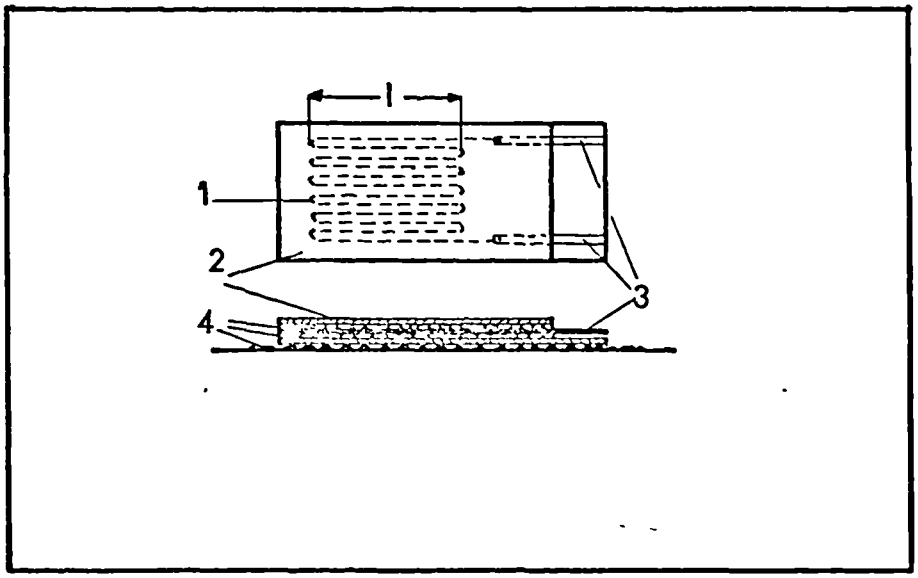
- "direct readout" whereby the physical deflection of the element is transmitted by a lever arrangement directly to a pointer on a measuring scale. This simple principle requires no external power and a minimum of design complexity. However, although such devices can be designed to produce accurate results, they have a restricted resolution.

An example of a "direct readout" device is provided by common weighing scales. These rely upon the deflection of a spring, the extension or compression of which is transmitted to a linear scale whose gradations give a direct indication of the force applied (figure 5.4).

- "strain gauges", typically these consist of a length of fine wire arranged in a grid pattern (figure 5.5). The gauges are bonded to the element that undergoes the deformation. A force applied to the transducer produces changes in the length and cross-sectional area of the gauge, leading to alterations in the electrical resistance. The strain gauges are connected together in the form of a Wheatstone bridge (vide 5.7.1), variations in their resistance producing a change in electrical output from the bridge. This output being proportional to the force applied. The orientation of the gauges on the transducer element determines the primary direction(s) of applied force (moments) to which it is sensitive.

Details of strain gauges, their design, theory of operation and techniques of application are described by Kühl (1976) and Micro-Measurements (1981).

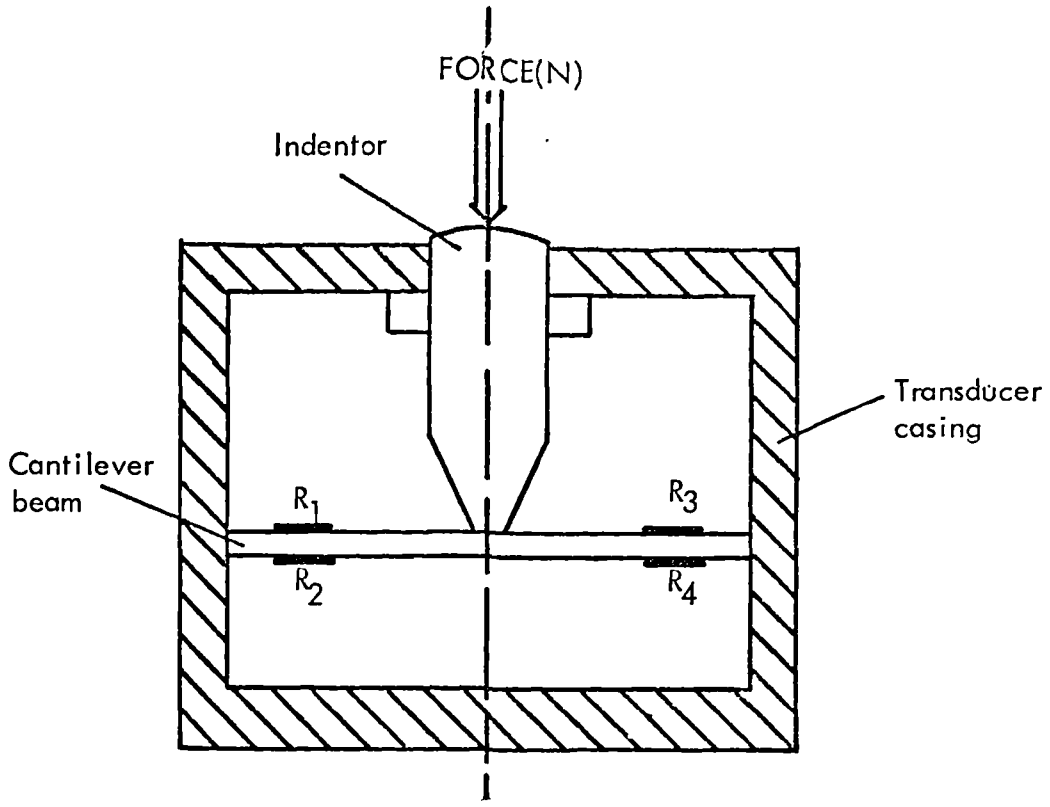
Force transducers employing strain gauges to measure the deformation



l = active gauge length
1 = resistance wire
2 = cover
3 = electrodes
4 = adhesive

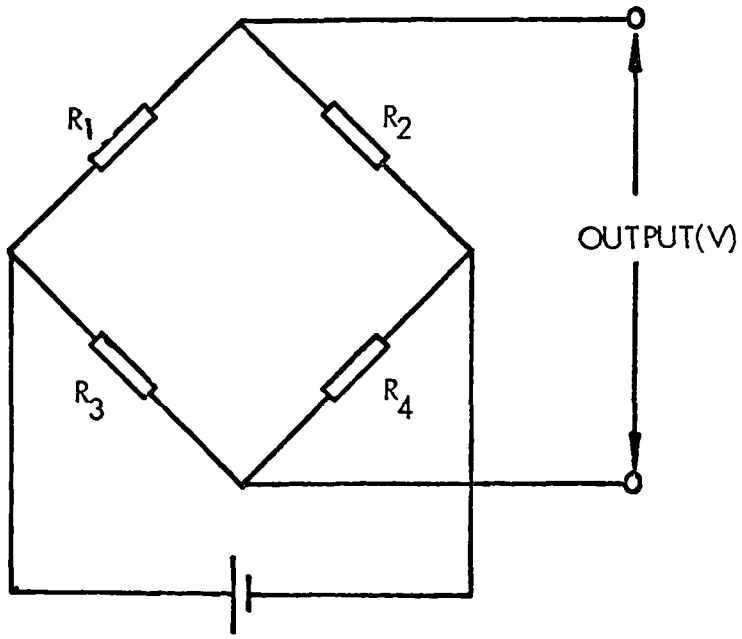
Figure 5.5 Schematic diagram of a strain gauge bonded to a flat surface.

(after Kühl, 1976)



R_1, R_2, R_3 and R_4 = Strain gauges

FORCE(N) $\propto$ OUTPUT(V)



Wheatstone bridge

Figure 5.6 A force transducer employing strain gauges as the measuring element.

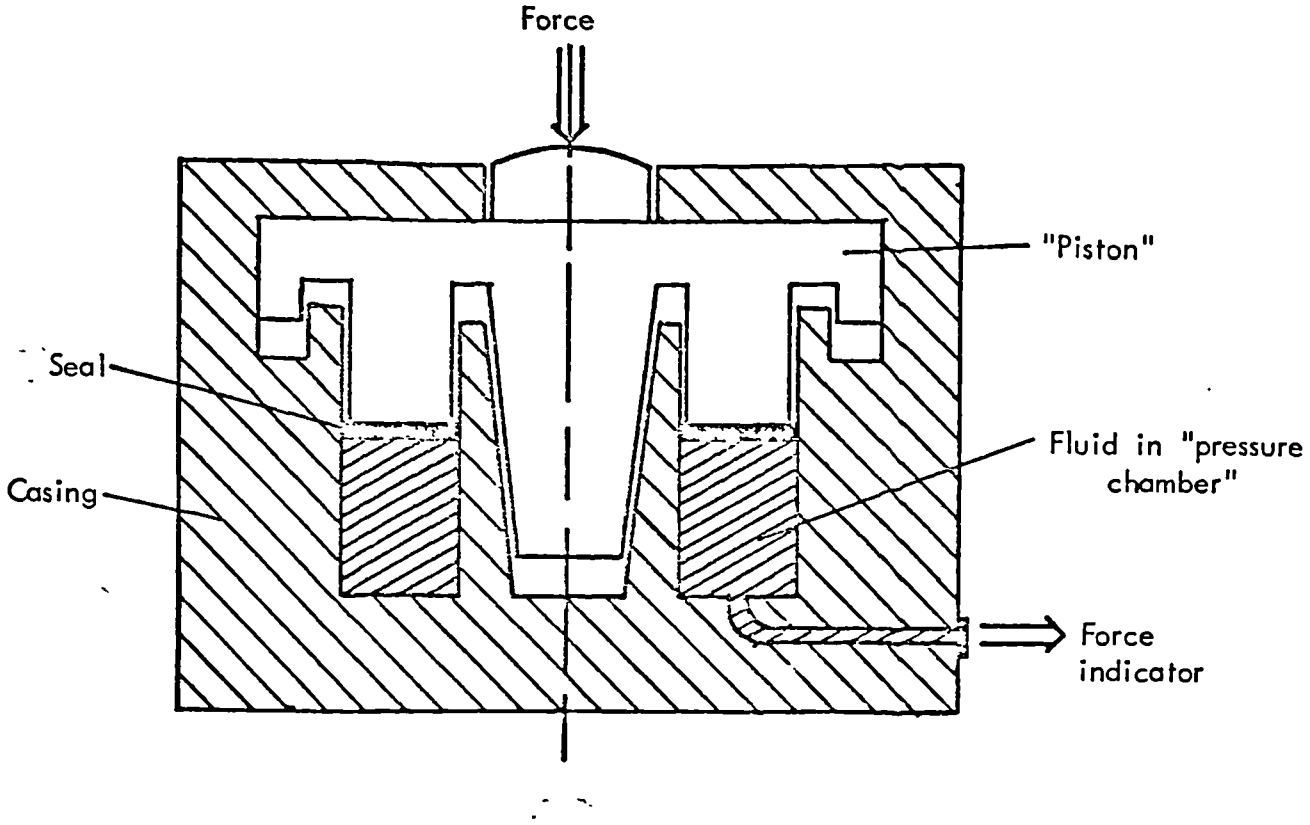


Figure 5.7 An example of a force transducer employing compression of a fluid ("hydraulic").

of a mechanical element under load, provides for an accurate device, capable of measuring a wide range of forces with a typical frequency response of 2kHz. However, unlike direct readout devices, they require electrical energisation along with accompanying amplification of their output. A typical device is illustrated in figure 5.6.

Hydraulic: Force transducers of this design are not as common as the mechanical devices but are capable of an equally large load range and high resolution. Hydraulic force transducers consist of 3 basic elements (figure 5.7). A chamber filled with fluid (assumed to be incompressible), a "piston rod" which fits into the chamber and a pressure gauge (often a Bourdon gauge), which acts to measure the pressure of the fluid in the chamber. Force applied to the piston rod is transmitted through the fluid in the chamber to the pressure gauge. Thus the measured pressure is directly proportional to the force applied.

In contrast to those utilising strain gauges, hydraulic force transducers require no external electrical power making them particularly suitable for use in remote locations. The dynamic response of hydraulic force transducers is usually limited (<10 Hz) and is a function of the viscosity of the hydraulic fluid.

Variable inductance: Transducers are available which operate on the principle of an applied force changing the inductance of a coil, usually by relative displacement of a high permeability core and the coil windings. The application of a force to the armature results in a change in the air gap, varying the current induced in the coils. Thus, by calibrating the change in air gap with variations in inductance of the coil, it is possible to provide a measure of the applied force.

Due to the inherent characteristics of inductance, transducers of this design require an alternating current supply. In addition, they are highly non-linear, subject to hysterisis and require regular calibration. Such factors indicate the unsuitability of variable inductance transducers for this application.

Piezoelectric: There are a number of commercially available force transducers employing piezoelectricity (Kistler, Endevco, Pitran). These

devices rely upon the property of some crystalline substances to generate an electric current (piezoelectricity), when loaded (eg. lead zirconate-titanate, quartz). Their major advantage is rigidity and a correspondingly high natural frequency, determined by the nature of the crystal. This makes them ideal for the measurement of rapid changes in force. However, they have a limited steady-state response, and are not suitable for the measurement of static signals. This characteristic precluded transducers of this design from use in this study.

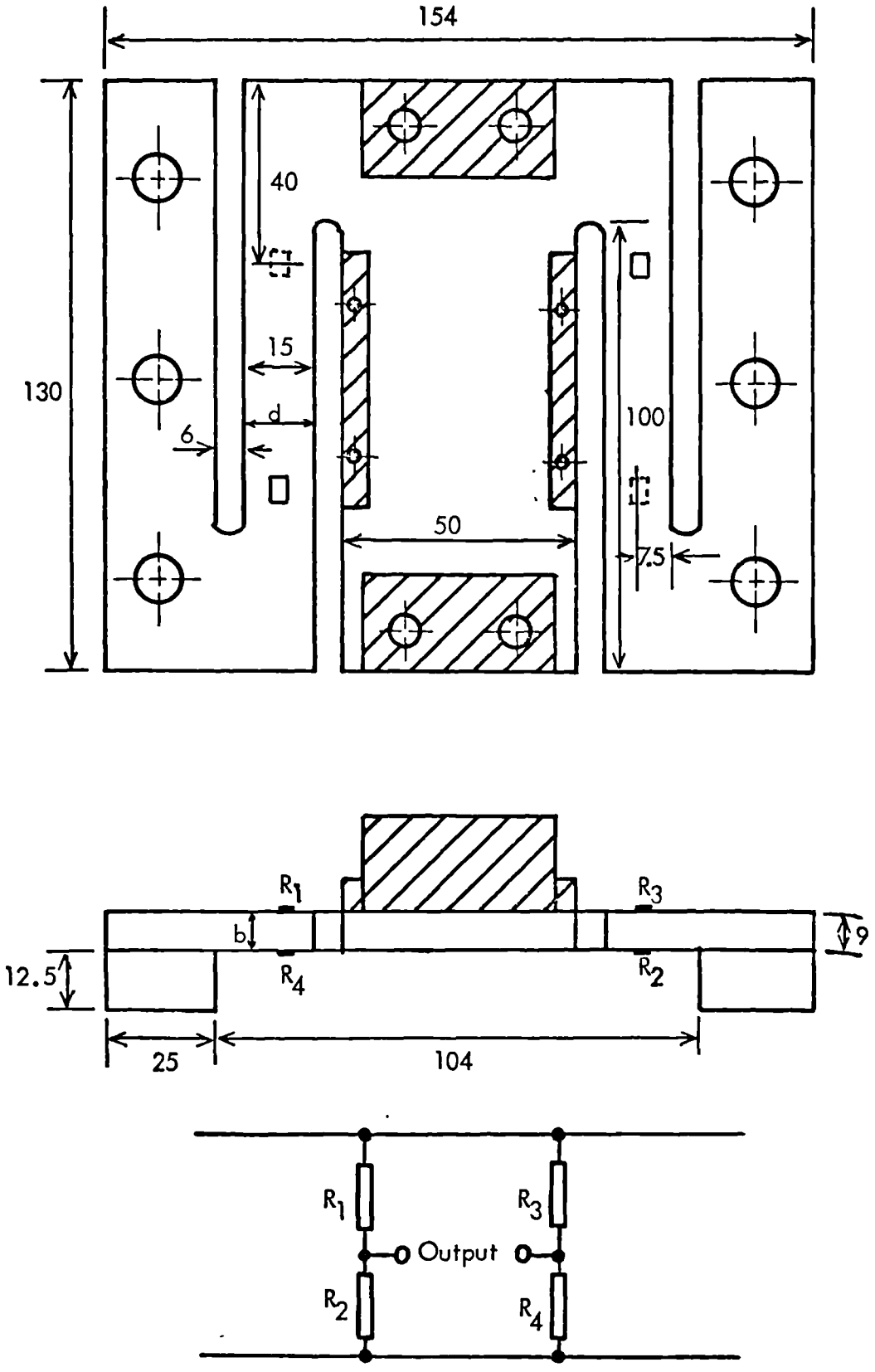
Having reviewed the commercially available force transducers, only one was revealed that complied with the majority of the design criteria as outlined (vide 5.4.1). This device formed part of a system manufactured to provide an accurate assessment of the weight of a subject in bed (Vickers Medical, U.K.) and has been of particular use in the case of renal dialysis patients. The force transducer was capable of stability underneath a bed wheel, although no restraining device was present to prevent the wheel from rolling off the cell, should the bed be pushed. However, these transducers proved too expensive (£560 each).

As no feasible, commercially available transducer was suitable, a new transducer was developed specifically for use in this application.

5.4.3 Choice of design principle for force transducers

Force transducers utilising variable inductance and piezoelectric effects are particularly limited by their non-linearity and a lack of steady state response. Hydraulic force transducers, with the addition of a pressure transducer replacing the dial gauge in order to provide an electrical output, have been used successfully, but the complexity of their design, requiring components to be manufactured to high tolerances, and possible instability of their loading surface under conditions of off-centre loading, precluded this design principle.

Mechanical force transducers with a direct mechanical indicator are simple but lack resolution and an electrical output. Strain gauged devices are accurate, sensitive, have a high frequency response, are capable of high



All dimensions in mm. (strain gauges not to scale).

Figure 5.8 Force transducer dimensions indicating position of strain gauges and of the coupling mechanism.

resolution and have an electrical output, although they require additional instrumentation. Since electronic conditioning of the signal was to be required in order to provide a simple, single-channel output representing the magnitude of a movement, it was decided that a strain gauged transducer would be suitable. To provide for a sensitive device use was made of two strain gauged cantilevered sections, separated by a loading surface. By the correct arrangement of strain gauges on the cantilevers, the output from the transducer was representative of the vertical component of the load, while being independent of its point of application on the loading surface. This design also allowed for a low-profile transducer with a large loading surface, to which coupling devices could be easily fitted. Each transducer was milled out of a solid mild steel plate and two aluminium support surfaces were attached to its underside (figure 5.8) (Wheatley et al, 1980).

5.4.4 Design details

Physical dimensions: The maximum load capacity that each transducer was required to be capable of sustaining before yield was 2.0 kN (vide 5.4.1). With this as the principal determinant, calculations were made to establish the dimensions of a cantilever capable of supporting the load;

The load is supported approximately equally between two cantilevers, i.e. 1.0 kN each.

For convenience the cantilevers are selected to be 100 mm in length. Thus the maximum bending moment K, resulting from the design load applied to the loading surface $= 0.1 \times 1000$ Nm

or $K = 100$ Nm

We know $\sigma_y = \frac{K}{z}$ (Timoshenko and Gere, 1973)

where σ_y = yield stress (Nm^{-2})

z = section modulus

Typically, for mild steel,

$$\sigma_y = 2.8 \times 10^8 \ Nm^{-2}$$ (Oberg and Jones, 1974)

$$\text{Thus,} \quad z = \frac{100}{2.8\times10^{8}} \ m^3$$

$$= 3.5\times10^{-7} \ m^3$$

The section modulus is a function of the physical dimensions of the member, in this case

$$z = \frac{bd^2}{6}$$

where b = the width of the section
d = the height of the section
} see fig. 5.8

b and d define the size of cross-section of the cantilever

Nominally choose b = 10 mm

$$\text{Thus,} \quad d = \sqrt{\frac{6\times3.5\times10^{-7}}{10\times10^{-3}}} = 14.5 \text{ mm}$$

To support a load of 1.5 kN the cantilevered sections indicated in figure 5.8 require a cross-section of 145mm^2.

Coupling: The choice of coupling method was greatly eased by the size of the loading surface and the arrangement of strain gauges on the cantilevers, which allowed a response independent of the wheel position. Aluminium blocks of suitable dimensions were fixed to the loading surface, to prevent the wheel from rolling off the cell. The size of these blocks was determined by the radius of the bedwheel; the Kings Fund bed wheels having a radius of approximately 62.5 mm. As a result, aluminium blocks of 19 mm x 17 mm x 50 mm were bolted to both ends of the loading surface, with blocks of 5 mm x 5 mm x 25 mm attached to either side. This provided a simple, quick and secure means of coupling the load cell to the bedwheel (figure 5.9).

Strain gauges: To provide maximum sensitivity each load cell was fitted with four foil strain gauges, temperature compensated for steel with gauge lengths of 1.57 mm (EA - 06 - 062AA - 120) (Micro-Measurements, Basingstoke, U.K.) connected in a full-bridge configuration to ensure that the vertical component of the load on the loading surface is measured, independent of its point of application.

The dimensions of the cantilever provided a large surface area

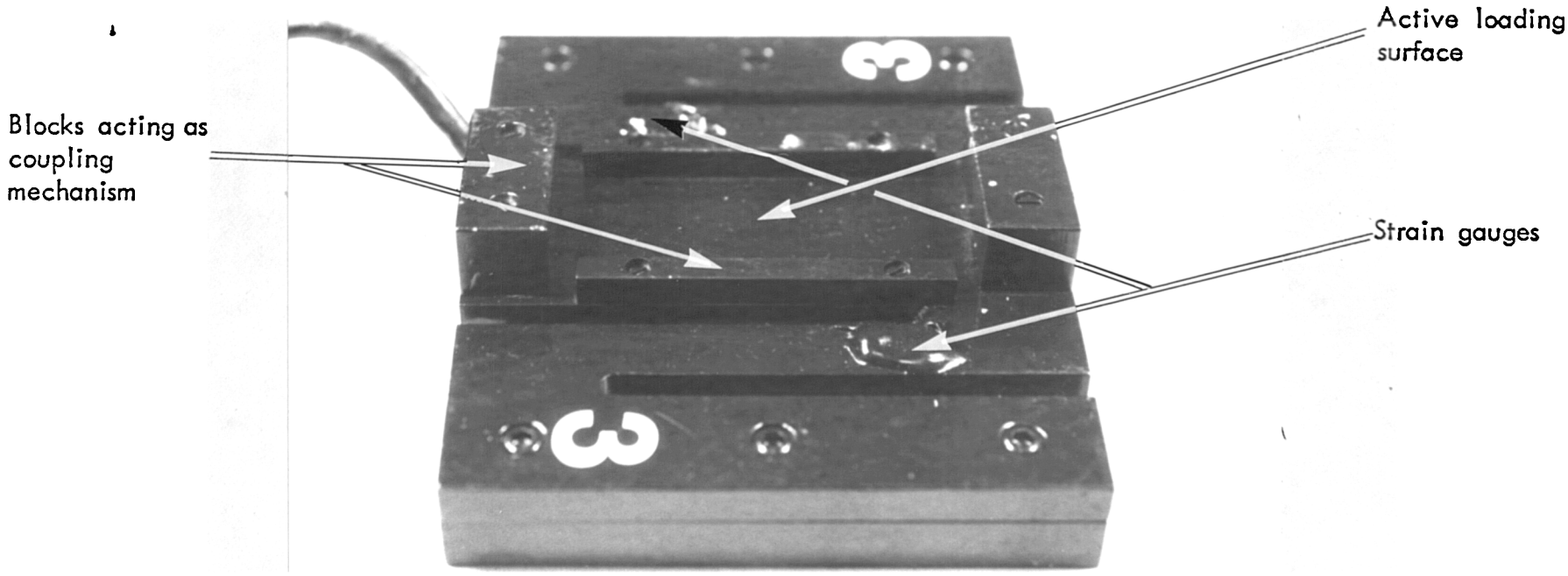


Figure 5.9 An assembled force transducer.

available for mounting the strain gauges, and their arrangement enabled cross-effect strains to be balanced out, leaving the transducer sensitive only to loads applied along a specific axis. Connection to each strain gauge was made by polyurethane lacquer insulated, copper wire, attached to solder tabs on the gauge by means of micro soldering. Care was taken to ensure that the insulation around the wire was maintained for its entire length, since any break led to a short circuit and damage to the strain gauge amplifier. To optimise thermal compensation, the leads between the strain gauges were all of approximately the same length. A four-way solder tab was mounted on the uppermost surface of the load cell to provide a connection between the strain gauge bridge and the external energisation and amplification.

Environmental protection: To protect the strain gauges and inter-connecting wiring from damage, they were coated in a hard setting plastic (Gage coat G, Micro-Measurements, Basingstoke, U.K.). After the application and setting of which, repositioning or modification of the strain gauges was not possible. To prevent rusting and corrosion, the complete load cell was painted with "Wilmar" air drying hammer finish paint. After connection of the energisation and signal lines, silicone rubber was applied over the solder tab, providing a watertight seal (figure 5.10). However, unlike the Gage coat G, it was possible to remove the silicone rubber without damage to the solder tab and connections beneath it. Figure 5.11 shows a completed load cell in position underneath a hospital bed.

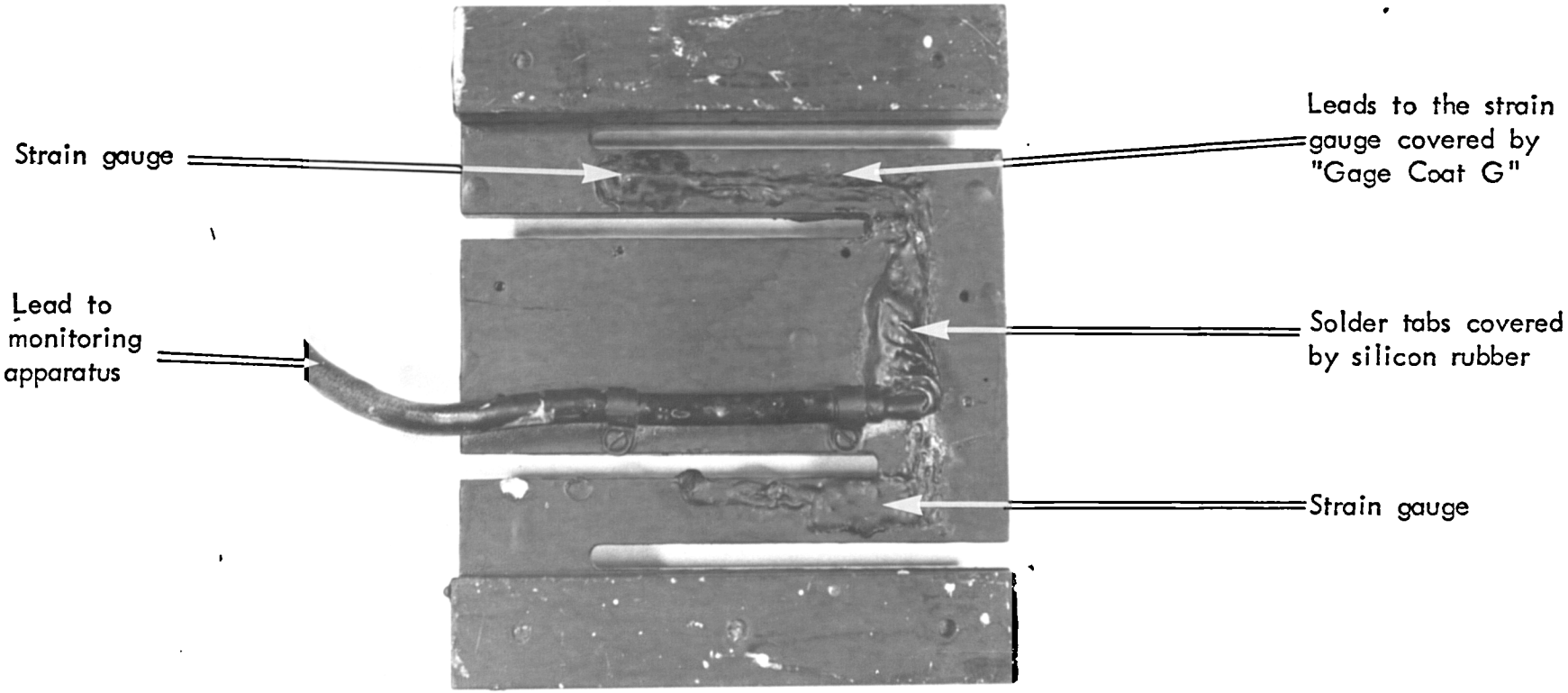


Figure 5.10 Underside of the force transducer indicating the position of the strain gauges and the wiring to them.

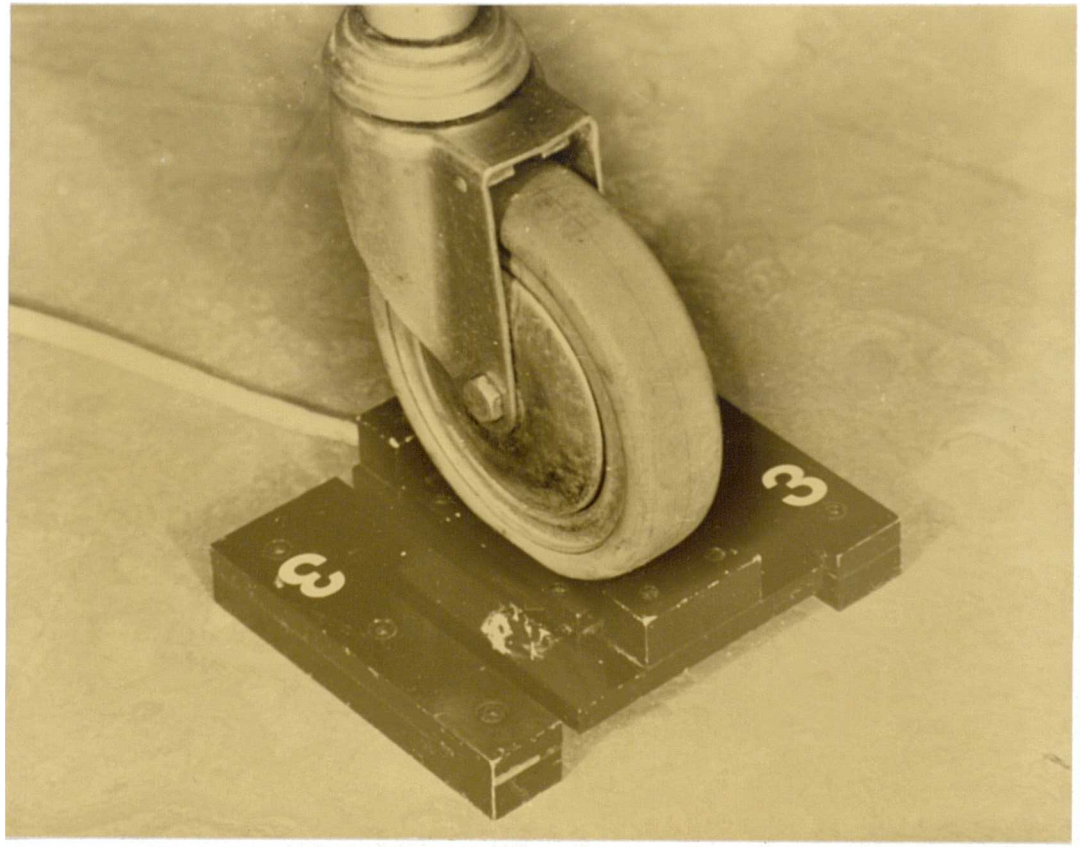


Figure 5.11 Force transducer in position underneath a bedleg.
(after Wheatley et al, 1980)

5.5 CALIBRATION OF THE FORCE TRANSDUCER

A series of tests was carried out to determine the characteristics of the force transducer with regard to: uniaxial loading, the effect of non-axial loading and the change of sensitivity with position on the loading surface.

5.5.1 Transducer characteristics under uniaxial vertical loading

Each transducer was positioned upon a support platform attached to the crosshead of an Instron TTCM mechanical testing machine (figure 5.12). A hardened steel indentor was fixed to the accurately calibrated measuring load cell of the Instron, the indentor having a cross-sectional diameter of 5 mm. Load was applied to the force transducer by means of raising the crosshead so that the indentor was in contact with the centre of the loading surface. The load was applied in a continuous ramp from 0 to 1.0 kN, and back down to 0 N. Movement of the crosshead was constant at 0.017 mm/s. The output of the force transducer and the force applied to it was recorded by means of a calibrated X-Y pen recorder.

The strain gauge bridge on the force transducer was energised by a four volt power supply and the output of the Wheatstone bridge was amplified by a CIL 307 strain gauge amplifier (CIL Electronics Ltd., Worthing, U.K). The test was repeated at other points on the loading surface, in order to determine the effect of off-centre loading.

5.5.2 Transducer characteristics under non-axial loading

Ideally the output of a uniaxial force transducer is dependent upon the force applied along one axis only. This axis is specified by the design of the transducer. However, in practice this is never the case and "off-axis" loading will give rise to artefactual outputs. This characteristic is commonly termed "cross-effect" and arises from imperfect coupling, limitations in design, accuracy of machining and errors in the placement of the strain gauges. A set of experiments was designed to evaluate the response to cross-effects of this force transducer. The loading and recording techniques were similar to those used in the previous calibration tests. However, the support platform was

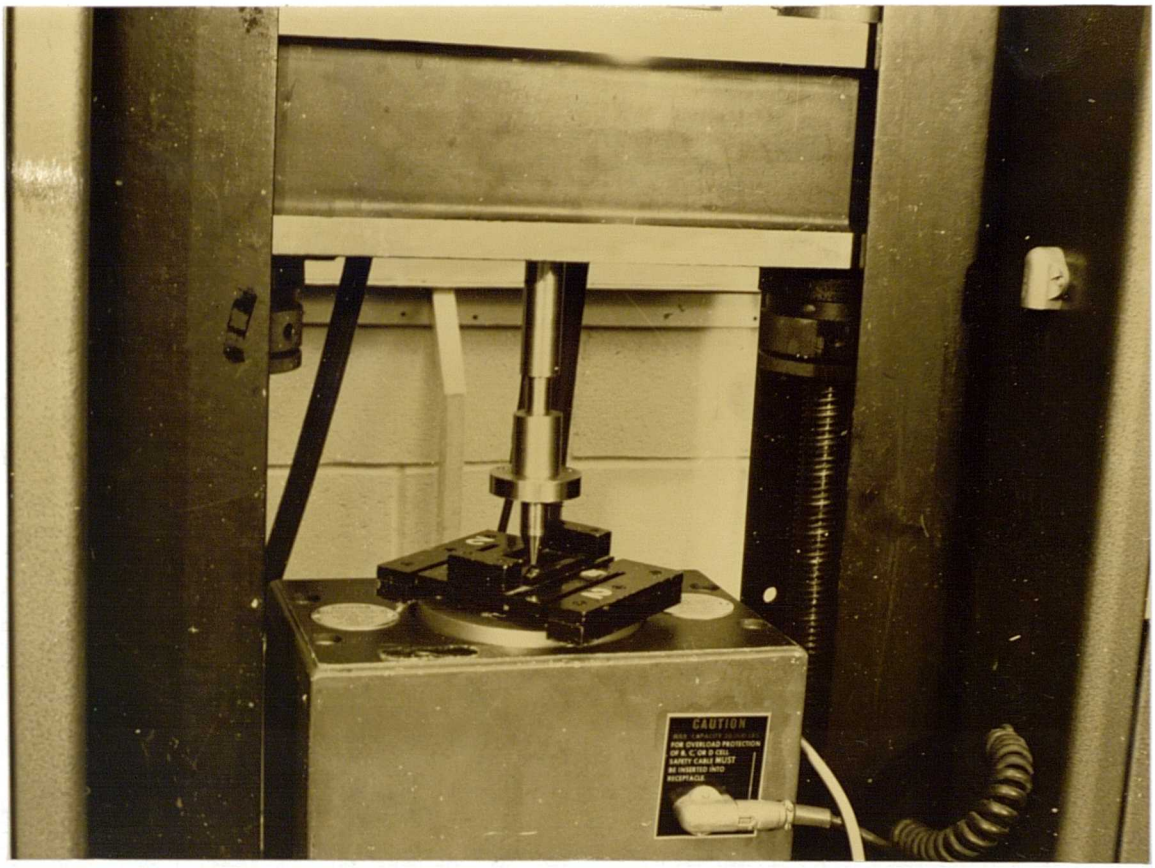


Figure 5.12 Calibration of the force transducer in an Instron TTCM mechanical testing machine.

modified, so that the force transducer was presented to the indentor at an angle of 12°. The theory of design and positioning of the strain gauges, meant that for the ideal case, the transducer measured only the normal component of the load.

5.5.3 Results

The results of the static loading tests indicated a force transducer senisitivity of 0.425 μV/V N. A linear regression of the data, established that within the range 0 to 1.0 kN the response of the force transducers was completely linear (s = 0.9999) and free from hysteresis (figure 5.13).

Results of the outputs derived from varying loading positions on the loading surface are summarised in figure 5.14 a and b. Analysis of this data showed that the sensitivity of the transducer varied by approximately 2% or less, depending upon the position of the load.

Results from the series of experiments to establish the response of the load cell to off-axis loads, were compared with values derived from the previous set of tests. To analyse the data, allowing comparison with earlier results, it was decided to consider the applied load in component form, these components being normal (Lx) and horizontal (Ly) with reference to the loading surface of the transducer and were calculated as,

$$Lx = L \sin 12^{\circ}$$

$$Ly = L \cos 12^{\circ}$$

where L is the applied load

In this manner the response due to cross effects was determined to be 2% - 6% of the applied load, depending upon where the load was applied on the loading surface.

5.5.4 Summary

Characteristics of the force transducer were established as,

Sensitivity	0.425 μV/V N
Nonlinearity	Better than 0.1%

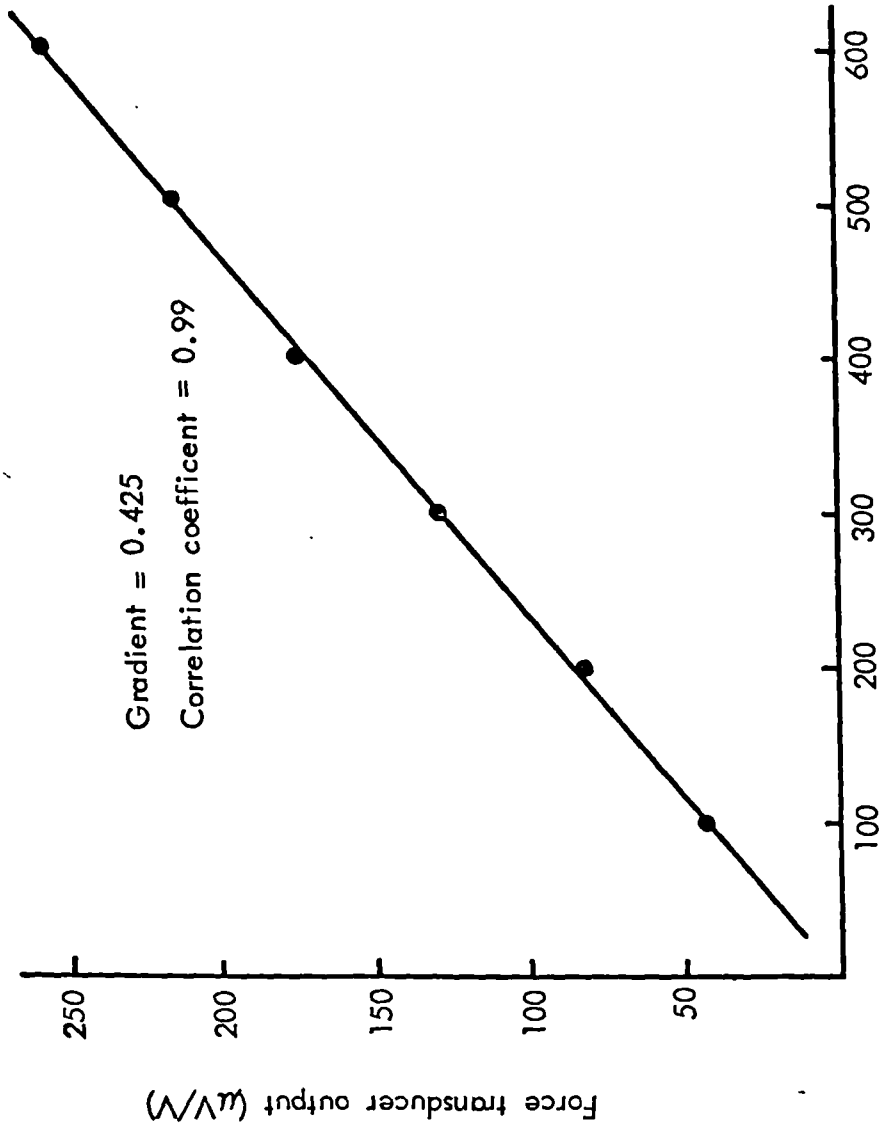


Figure 5.13 Force transducer calibration - output versus applied load.

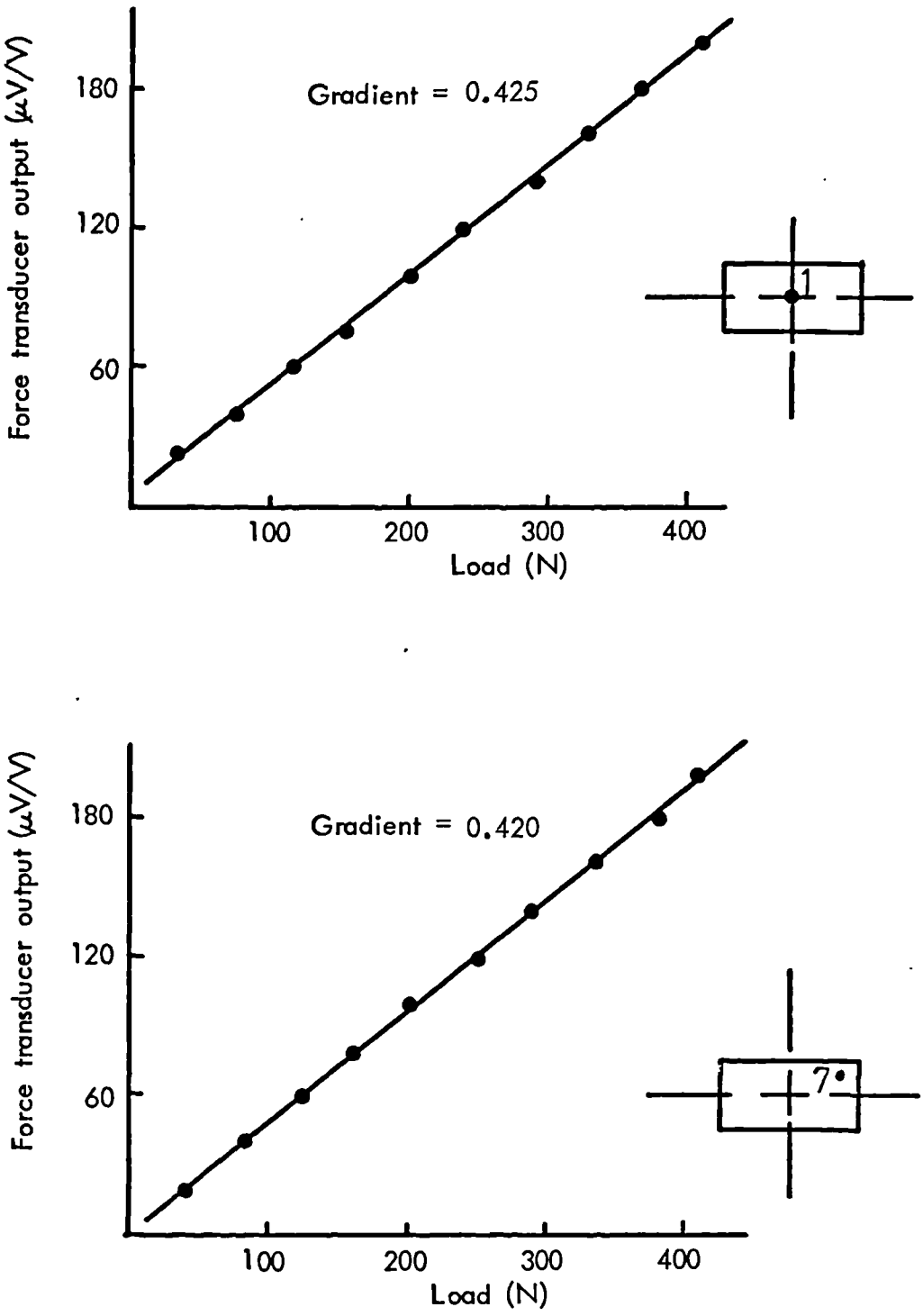


Figure 5.14a Test for artefacts due to off - centre loading of the force transducers.

Loading positions 1 and 7

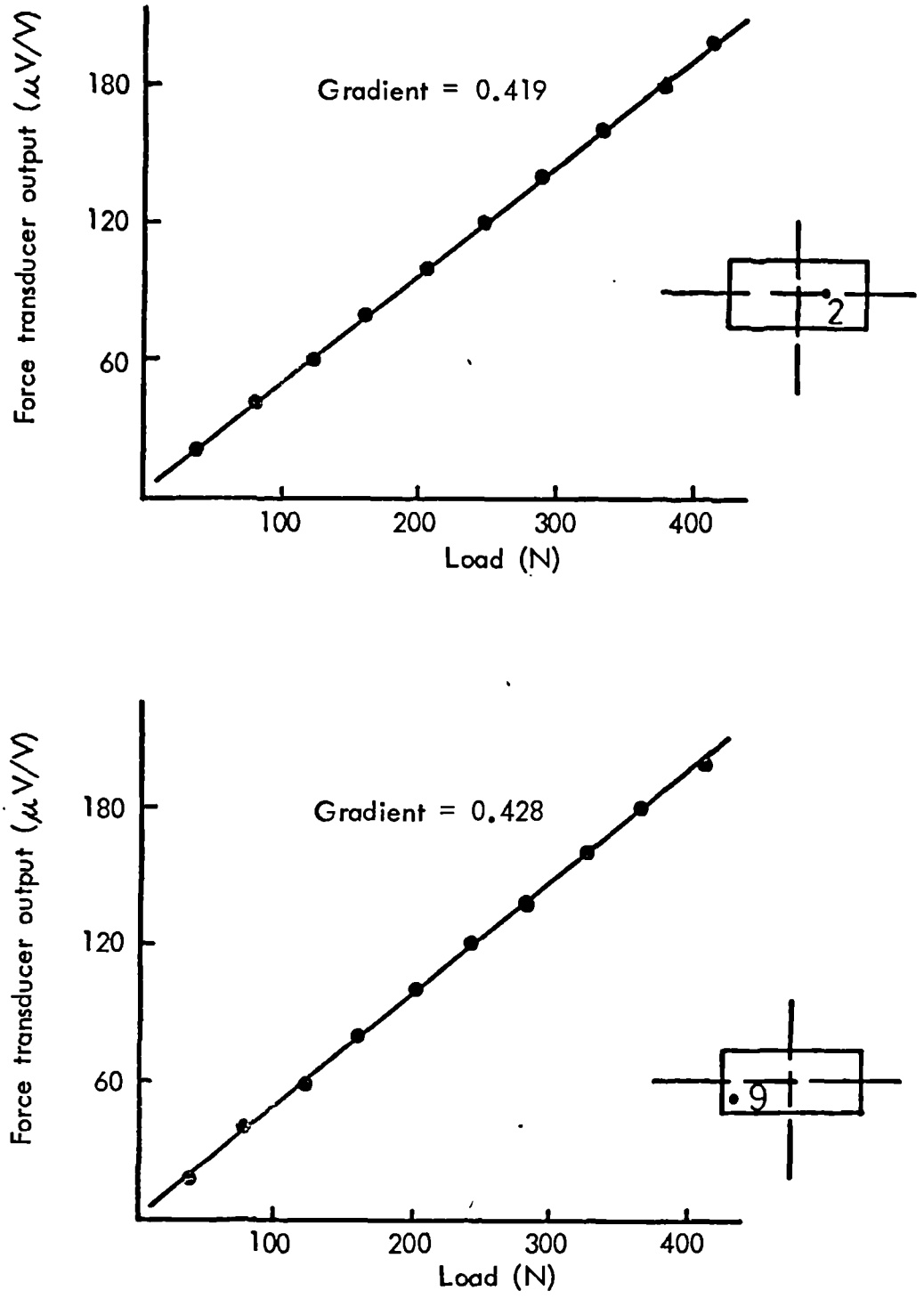


Figure 5.14b Test for artefacts due to off - centre loading of the force transducers.

Loading positions 2 and 9

Changes in sensitivity with varying position on the loading surface	Better than 2.0% of applied load
Cross-effects	2% - 6% of the applied load

The force transducer produced, met all the demands of the design criteria (vide 5.4.1). It was of low profile (21.5 mm) (excluding coupling), compact (130 mm x 154 mm x 21.5mm), the transducer had a large "active loading surface", was sensitive to uniaxial loads only (within 2% - 6%), was capable of a quick, simple and efficient means of coupling to a bedleg wheel and could sustain up to 3.0 kN before permanent damage.

Although the sensitivity of the force transducer was limited, suitable, low noise, high gain, drift free amplifiers provided signals with adequate resolution.

5.6 OBJECTIVES FOR ELECTRONIC SIGNAL DETECTION AND PROCESSING

There are two basic approaches which could be used to provide processing of the signal - analogue and digital. For this study, the conceptually simpler, lower cost analogue system was selected as the method of choice. However, with recent developments in the capabilities of micro-computers, a second generation digital system has been designed (vide chapter 8) to monitor two beds simultaneously and is being developed to extend the research programme described in this thesis.

5.6.1 Theory of operation

The analogue processing electronics was required to produce an electrical output representative of the magnitude of a movement by a subject in bed. To achieve this, use was made of equation 5-8 (vide 5.3.1) namely,

$$\underline{M} = \sqrt{\Delta x_s^2 + \Delta y_s^2} = \frac{\sqrt{\left((\Delta(S_1+S_2)) \cdot X_D\right)^2 + \left((\Delta(S_2+S_3)) \cdot Y_D\right)^2}}{S_1 + S_2 + S_3 + S_4}$$

where, x_s and y_s represent the position of the subject's centre of gravity in bed.

S_1, S_2, S_3 and S_4 are the reaction forces at each of the bedlegs, attributable to the weight of the subject.

Thus, if the reaction forces and the dimensions of the bed are known, then the magnitude of the movement may be calculated.

By placing load cells (vide 5.4) beneath the bedleg wheels, it is possible to obtain an electrical representation of forces S_1, S_2, S_3 and S_4. The bed dimensions X_D and Y_D are constants, related to the size of the bed and as such, may also be easily represented in electrical form.

On this basis an analogue processing system was developed to calculate the spatial coordinates x_s and y_s of the centre of gravity of a subject in bed. Vector format addition led to an output representative of the magnitude of a move by the subject in the bed.

Previous systems had taken no direct account of the weight of the

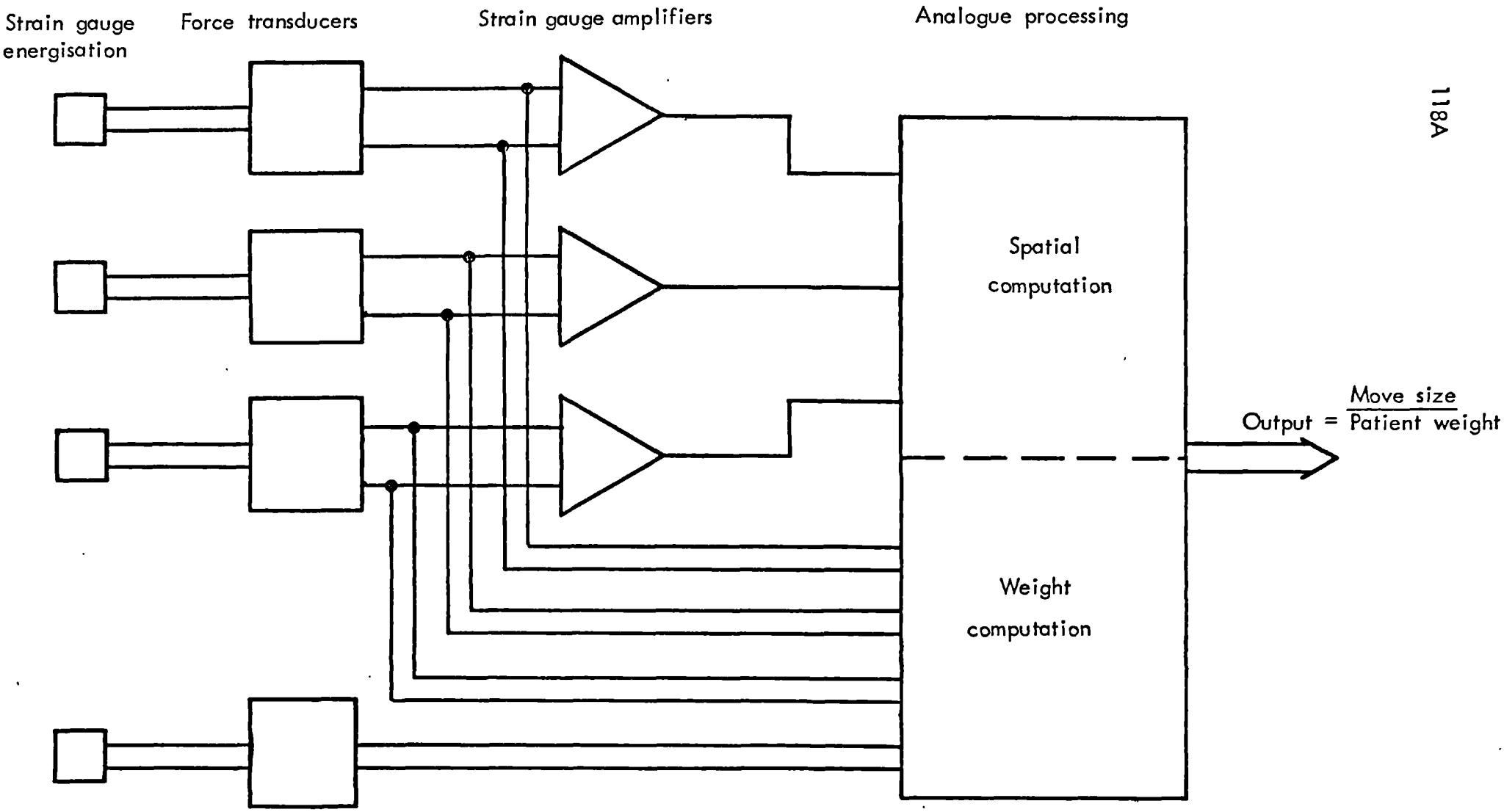


Figure 5.15 A schematic diagram of the elements in the transducing and data processing system.

subject. However, Crisp et al (1970) calculated an equation, based upon careful calibration of his system which allowed him to compensate for changes in weight between subjects. Bardsley (1977) was able to provide adjustment by the use of a calibrated, externally adjustable, potentiometer, the output of which was representative of the subject's weight. Both of these methods of compensation served to introduce further inaccuracies into the mobility monitoring system. In addition, they require the exact weight of the subject to be known, before each test. This requirement can present difficulties in the clinical situation, since the physical condition of the patient may preclude their removal from bed to be weighed, or substantial weight changes may occur during their stay in hospital. Thus, to improve the accuracy of the system and to facilitate clinical usage the subject weight, S was calculated automatically.

Analogue voltages representative of the physical parameters have been denoted as Vs_1, Vs_2, Vs_3, Vs_4, Vx and Vy related to S_1, S_2, S_3, S_4, X_D and Y_D respectively.

In these terms the equation developed to calculate the magnitude of the centre of gravity change is represented as;

$$\sqrt{\Delta x_s^2 + \Delta y_s^2} = \frac{\sqrt{\left(\left(\Delta(Vs_1 + Vs_2)\right).Vx\right)^2 + \left(\left(\Delta(Vs_2 + Vs_3)\right).Vy\right)^2}}{Vs_1 + Vs_2 + Vs_3 + Vs_4} \qquad (5\text{-}9)$$

5.6.2 System overview

A schematic representation of the processing system is presented in figure 5.15. The design may be considered to be composed of three elements, these being strain gauge bridges, strain gauge power supply and amplification and analogue computation.

Strain gauge bridges: These formed the measuring element of the force transducer (vide 5.4.3).

Strain gauge power supply and amplification: To provide an electrical output it was necessary to apply an energisation voltage to the strain gauge bridge. The Wheatstonebridge formation of strain gauges

produced a change in output dependent upon a variation in any one or combination of the strain sensitive resistance devices. The magnitude of this output being dependent upon the size of change in resistance and the energisation voltage. A strain gauge bridge amplifier was employed to convert this signal to a size suitable for further processing.

Analogue computation: After strain gauge bridge amplification, the voltage output of three of the four transducer bridges was analysed using analogue circuitry in order to represent the numerator of equation 5-8. The output of all four transducers served as inputs to a separate circuit, designed to provide an electrical analogue of the weight of the subject, this being the denominator of equation 5-8. Finally, the two analogue processing circuits were combined by means of a divider, to give an electrical analogue representation of the magnitude of a move of a subject in bed.

5.7 SIGNAL ACQUISITION

The strain gauges attached to the force transducer were connected in the form of a Wheatstone bridge. This output was then amplified to a level suitable for further analogue processing.

5.7.1 Strain gauge bridges

The magnitude of the output from a Wheatstone bridge may be represented as

$$\frac{R_2R_3 - R_4R_1}{(R_1 + R_2)(R_3 + R_4)} \cdot V_e$$

where, R_1, R_2, R_3 and R_4 are strain gauge resistances (figure 5-8)

Ve is the energisation voltage

For this application Ve was selected as 5V, this being within the specified optimum working range of the strain gauges (Micro-Measurements Ltd, Basingstoke, U.K). Energisation voltages outwith this causing a degradation in the characteristics of the device.

5.7.2 Strain gauge amplifiers

Amplification of the strain gauge output signal required the use of a high quality electronic amplifier. The general characteristics of such a device have been outlined by Trappitt (1979). These include high input impedance, low output impedance, high common mode rejection ratio, low zero drift and a dynamic range comparable with that of the signal being detected.

The amplifier input impedance was required to be high, relative to the output impedance of the strain gauge bridge in order to reduce the current drain on the transducer (Doebelin, 1966). In a similar manner an output impedance was required which was low in relation to the input impedance of the following component, preventing excessive current drain from the amplifier.

The common mode rejection ratio (C.M.R.R.) is defined as the ratio of the output voltages produced by a unit difference voltage compared to that of a common mode signal of equal amplitude, ie.

$$C.M.R.R. = \frac{\text{output voltage/unit difference voltage}}{\text{common mode signal}} \quad (5\text{-}10)$$

The C.M.R.R. is particularly important if an amplifier is operating in differential mode and when the inputs are approximately equal. To minimise errors in this situation, amplifiers exhibiting large values of C.M.R.R. are selected.

Zero drift is a low frequency change in the mean value of the output voltage, related in part, to fluctuations in ambient temperature. Thus, as the amplifier was to be used for long periods (overnight) in an environment where the room temperature was likely to change (hospital), it was necessary to select an amplifier with a low thermal drift characteristic.

Low noise in an amplifier is a desirable feature, since the maximum level of noise is representative of the resolution of minimum signal magnitude. This is particularly important in the case of strain gauge bridge amplification where the input signals are likely to be in the order of microvolts.

Dynamic range is the ratio of expected maximum to minimum amplitudes of the input signal. The amplifier must have a dynamic range at least equal to that of the signal, so that it can recognise the lowest level of signal without it being obscured by noise, and the highest level of signal without introducing distortion.

Trappitt (1979) outlines four different designs of amplifiers capable of amplifying the signals from a strain gauge bridge.

- the carrier amplifier, which has the advantage of extremely low zero offset drift, but requires a carrier frequency of approximately ten times that of the most rapidly changing signal frequency to be detected.
- the chopper amplifier, which converts the difference between the d.c. or low frequency input voltage, at high input impedance, and the feedback voltage to a high-frequency square wave. This is then amplified with no drift, demodulated and filtered, producing an output waveform that is an amplified analogue of the input.
- the d.c. amplifier maintains d.c. coupling throughout its amplification process, providing a large signal bandwidth. However, drift

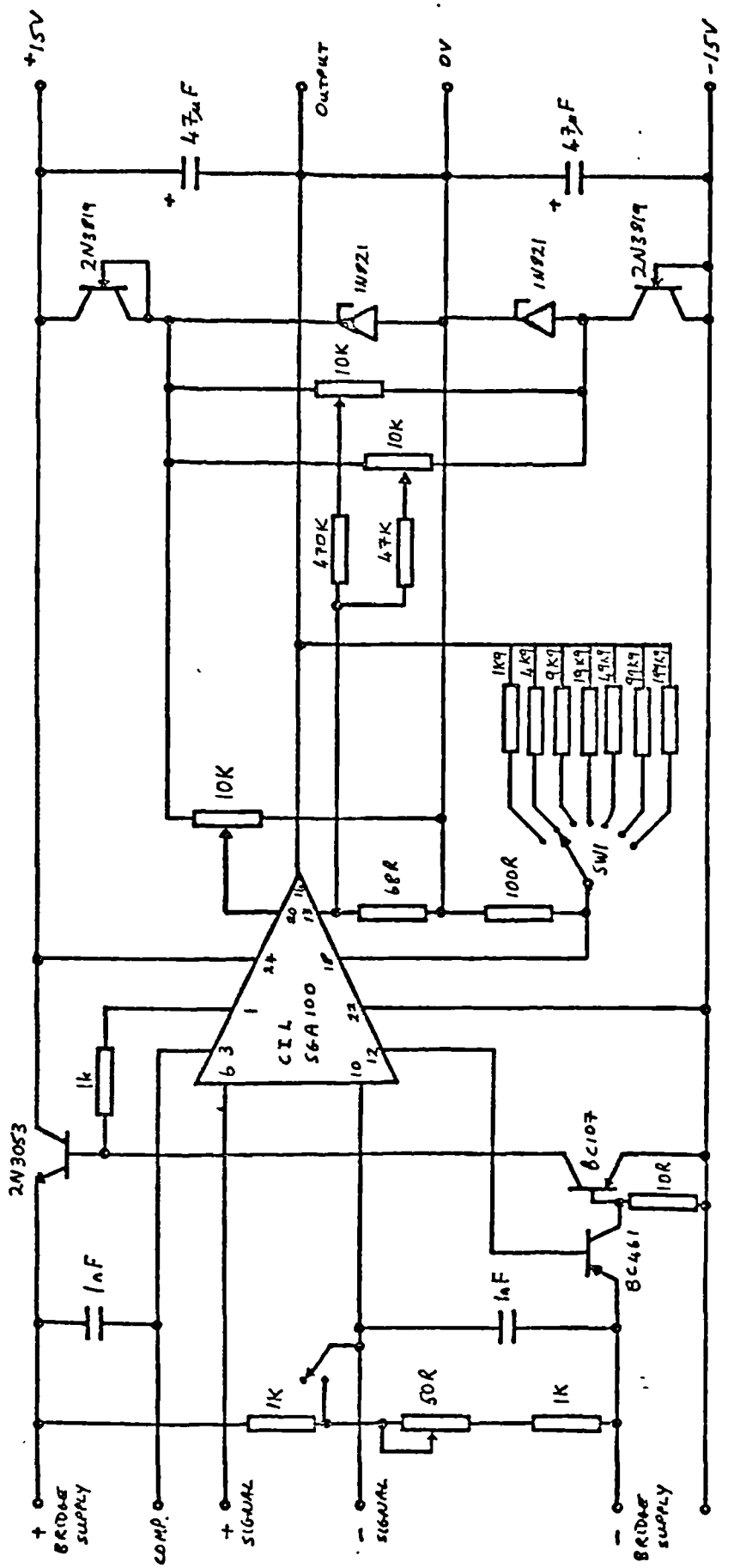


Figure 5.16 Strain gauge bridge amplifier employing the SGA 100 amplifier unit.
(after Trappitt, 1979)

in these amplifiers is greater than in those utilising signal modulation.

The choice of amplifier was based on the design criteria for this particular application, which may be summarised as,

Bandwidth of 0 - 10 Hz: it has been demonstrated that only the low frequency component of the signal, produced by the force transducers as a result of a move was required (vide 4.6).

Input impedance of at least 100 kΩ: The strain gauge bridge provides a source impedance of 120 Ω, thus a100kΩ input impedance alters the sensitivity of the system by less than 0.1%.

Noise level of 5μV (referred to the input): The signal resolution is equivalent to the minimum input magnitude and was 50μV. To provide a 10 : 1 signal to noise ratio, the corresponding level of noise was considered to be 20 dB less than this. Similarly a thermal drift characteristic of 1μV/°C was required.

The amplifier initially selected was based on an SGA 100, precision strain gauge amplifier (C.I.L. Electronics Ltd., Worthing, U.K), which was capable of both energising the strain gauge bridge and amplifying its output (figure 5.16). The amplifier and surrounding components provided a positive and negative regulated bridge voltage of up to 12V at 60 mA. Offset zeroing could be obtained by the use of "coarse" and "fine" potentiometers, affording adjustment up to 16.6 mV and 1.75 mV (referred to the input) respectively. Gain was adjustable between 20 and 2000 in seven steps.

The design of the strain gauge amplifier proved to be suitable for this application, satisfying all of the specifications. Unfortunately when used in the hospital environment, the SGA 100 unit was prone to failure. After extensive investigation and consultation with the manufacturers, no definite cause for failure could be identified. As a result of this the strain gauge amplifier was redesigned.

An alternative (figure 5.17), employed an ICL 7650 (Intersil Inc., U.S.A.) chopper stabilised amplifier which had particularly good specifications with regard to zero drift (100 nV/mnth) and common mode rejection ratio (120 dB). A pair of chopper amplifiers operating as voltage

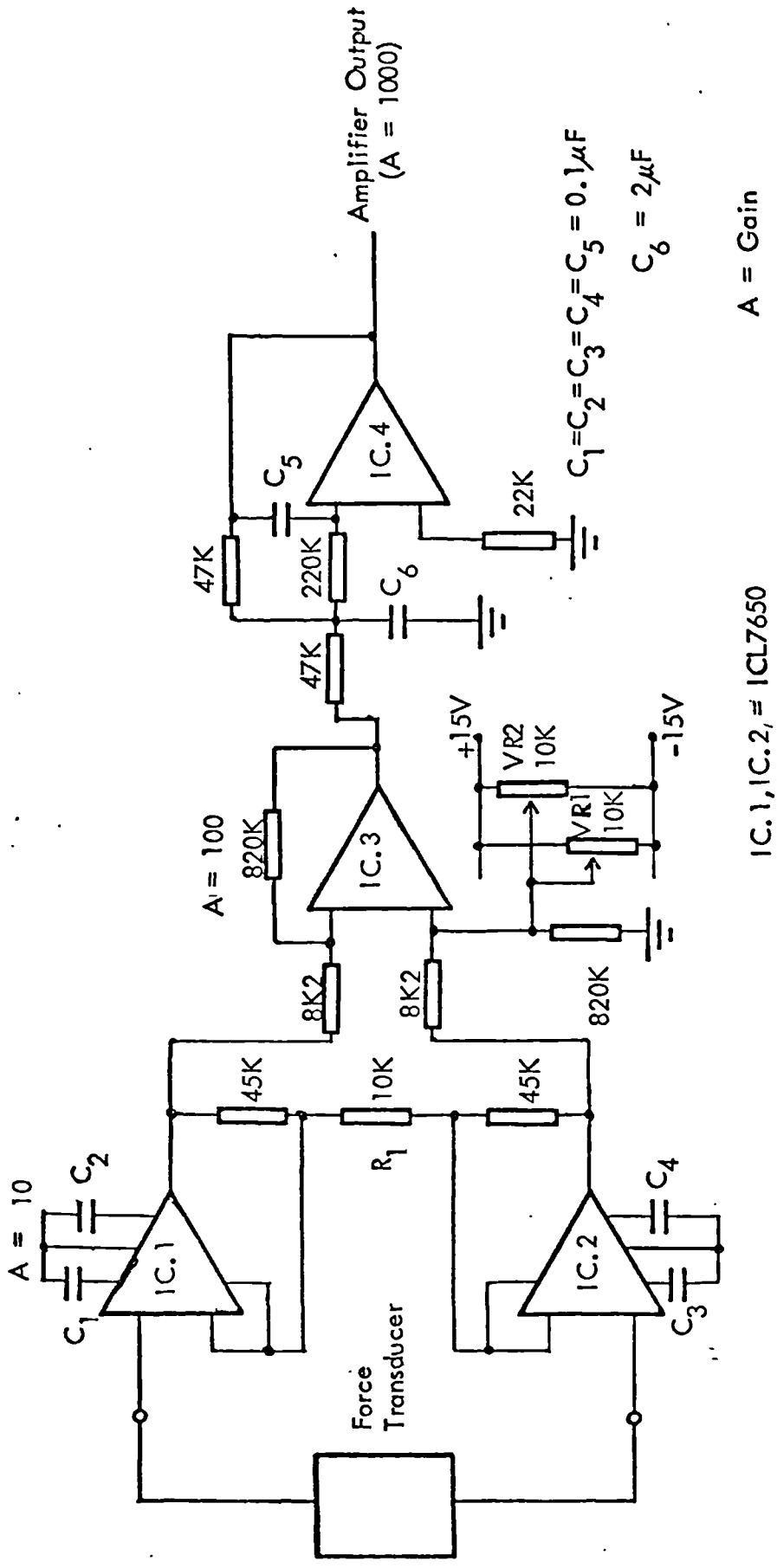


Figure 5.17 Strain gauge bridge amplifier.

followers with gain, were used as the first of a two stage amplification process. The signal output from the strain gauge bridge provided the noninverting inputs to each of the amplifiers.

The two amplifiers were coupled together (R_1), reducing the common mode gain to unity, while amplifying the differential signal. The common mode rejection ratio was then a direct function of the gain of the amplifiers. The second stage consisted of a conventional operational amplifier operating in differential mode, with gain. Using this configuration of amplifiers, the strain gauge bridge output was amplified to a magnitude suitable for further analogue manipulation.

One of the features inherent in chopper amplifiers is the presence in the output of rapid voltage pulses related to the internal chopping frequency of the device. As a result, their bandwidth is limited and is a function of the amplifiers's chopping frequency, (typically 10 Hz). However, this satisfied the design criteria for the strain gauge amplifier of 0 - 10 Hz. To eliminate the higher frequency components of the output from the chopper amplifiers, a single stage Sallen and Keye active low pass filter with a cut off frequency of 10 Hz formed the third and final stage of the amplification circuit.

The chopper amplifier achieves its low offset drift by comparing the inverting and non-inverting input voltages in a nulling amplifier. Using an internal clock pulse this zeroes the main amplifier on the positive going edge of the pulse and is itself zeroed on the negative going edge. Two external capacitors were required to store the correcting potentials on the two amplifier nulling inputs. These have optimum values depending on the clock frequency. For the present internal clock, the recommended value was 0.1μF.

To provide zeroing of the amplified load cell output signal, an offset control was provided in the second stage which, as previously with the SGA 100 design, facilitated "coarse" and "fine" balance.

Results of tests using the SGA 100 strain gauge amplifier, which allowed switchable gain between 20 and 2000 showed that a gain of 1000 was the optimum level. As a result the chopper stabilised amplifier was designed to have a fixed gain of 1000. This was achieved in two stages of 10 and 100

respectively.

Energisation for the strain gauge bridges was afforded by a 5V positive and negative regulated supply. An appropriate fixed potential divider providing a $\pm$ 2.5 volt energisation voltage. To allow fine adjustment of this voltage, a multiturn potentiometer was incorporated into the positive bridge voltage line.

The strain gauge amplifier utilising the chopper amplifiers proved capable of fulfilling the design specifications. However, the problem of failure of components following protracted usage in the hospital had not been eliminated. Extensive bench testing failed to repeat the situation. As a result, the quality of the mains supply in the hospital was questioned. To avoid damage by fluctuations in this supply, diodes (IN 4148) were connected between the inputs. The amplifiers then proved to work more reliably, although occasionally amplifiers appear to fail due to rapid voltage spikes damaging their input stages.

Bridge amplification was provided for three of the four force transducers. The fourth was required for calculation of the subject's weight only, and no bridge amplifier was provided for it, although an RS7805 voltage regulator (R.S.Components Ltd, London, U.K), with an output of 5V was used to provide energisation for the strain gauge bridge.

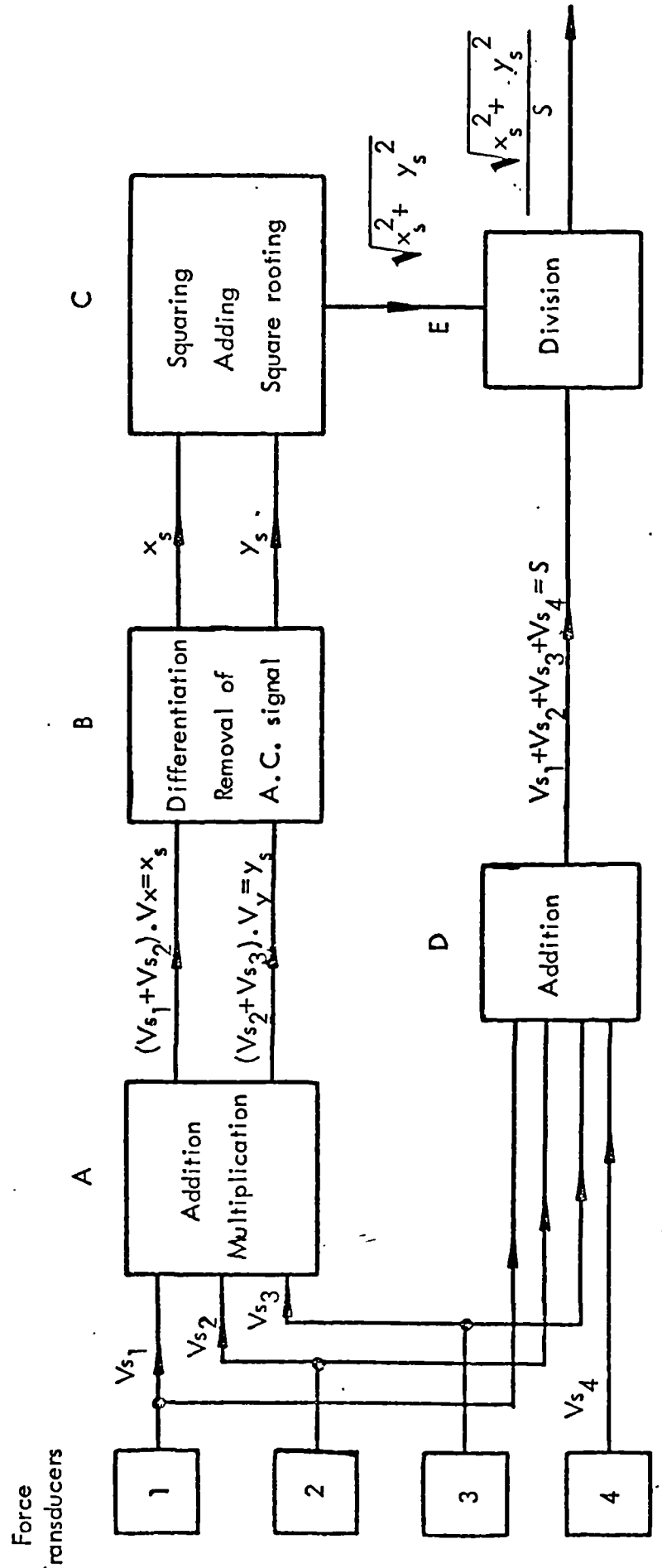


Figure 5.18 Schematic diagram of the analogue processing circuitry.

5.8 SIGNAL PROCESSING

Processing of the amplified outputs from the force transducers placed under the bedlegs was accomplished by means of an analogue system of electronics (vide 5.6.2).

Figure 5.18 is a schematic representation of the circuitry which will be considered in five sections A, B, C, D and E.

A and C representing algebraic analogue manipulation while B was used to specify the low frequency component of the signal, disregarding any associated transient input. D provided a representation of the patient's weight and E combined the outputs of C and D by means of analogue division.

5.8.1 Circuit A

The amplified voltages produced by three of the force transducers (Vs_1, Vs_2 and Vs_3) provided the input to this section of the circuitry. These were then processed to give an output of $(Vs_1 + Vs_2).Vx$ and $(Vs_2 + Vs_3).Vy$, which are the analogue equivalents of x_s and y_s per unit subject weight respectively. A second amplification stage provided scaling of the signal. Figure 5.19 is a detailed circuit diagram of A.

Circuit principle: The functions $(Vs_1 + Vs_2)$ and $(Vs_2 + Vs_3)$ were determined by operational amplifiers in summing mode (figure 5.19). The multiplicative constants Vx and Vy which described the dimensions of the bed, were represented by the gain of the summing circuit. To facilitate the use of beds with varying geometries, the gain was adjustable.

The second stage consisted of an operational amplifier connected as a follower with gain. A gain of 4.5 was selected based upon limitations of the signal magnitude required by the dynamic range of analogue components used in further circuitry (figure 5.19). The output of A could now be represented as $(Vs_1 + Vs_2).Vx.K$ and $(Vs_2 + Vs_3).Vy.K$, where K is a constant equal to the gain of this stage. The total gain of A, dependent upon the value of the first stage gain, ranged from 0 to 5 for Vx and from 4.5 to 7 for Vy.

All operational amplifiers were corrected for zero offset.

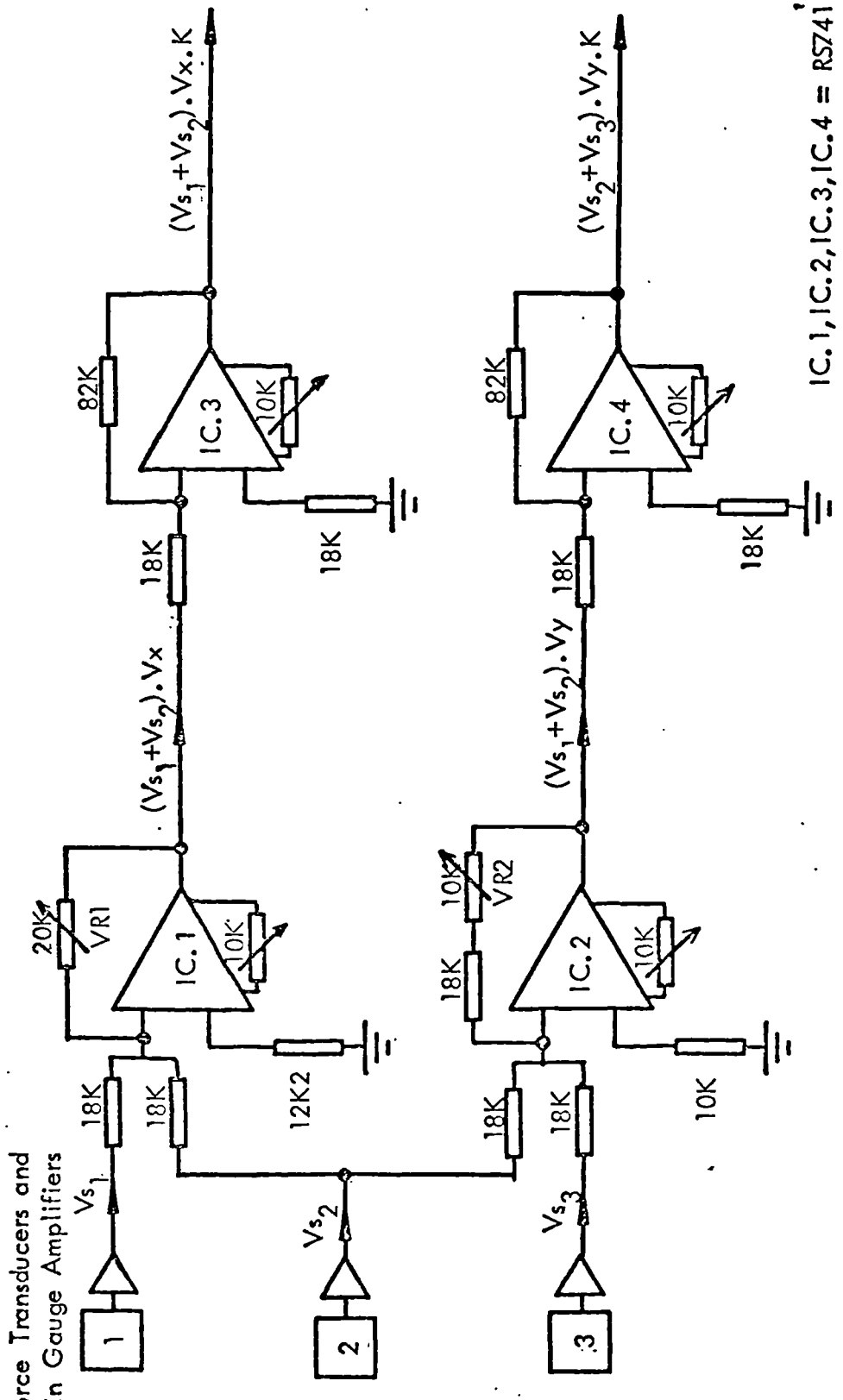


Figure 5.19 Circuit diagram of A.

5.8.2 Circuit B

The inputs to this section were $(Vs_1 + Vs_2)$. Vx and $(Vs_2 + Vs_3)$. Vy. The function of the circuit was to calculate the d.c. changes in signal level, disregarding transient components.

Circuit principle: Identical circuits were built to process each of the inputs separately. Figure 5.20 provides a simplified schematic outline of the circuit, which includes two pairs of sample and hold devices (I and II). Pair I outputs to a comparator (C_1) while the output of pair II formed the differential input to a comparator in section C.

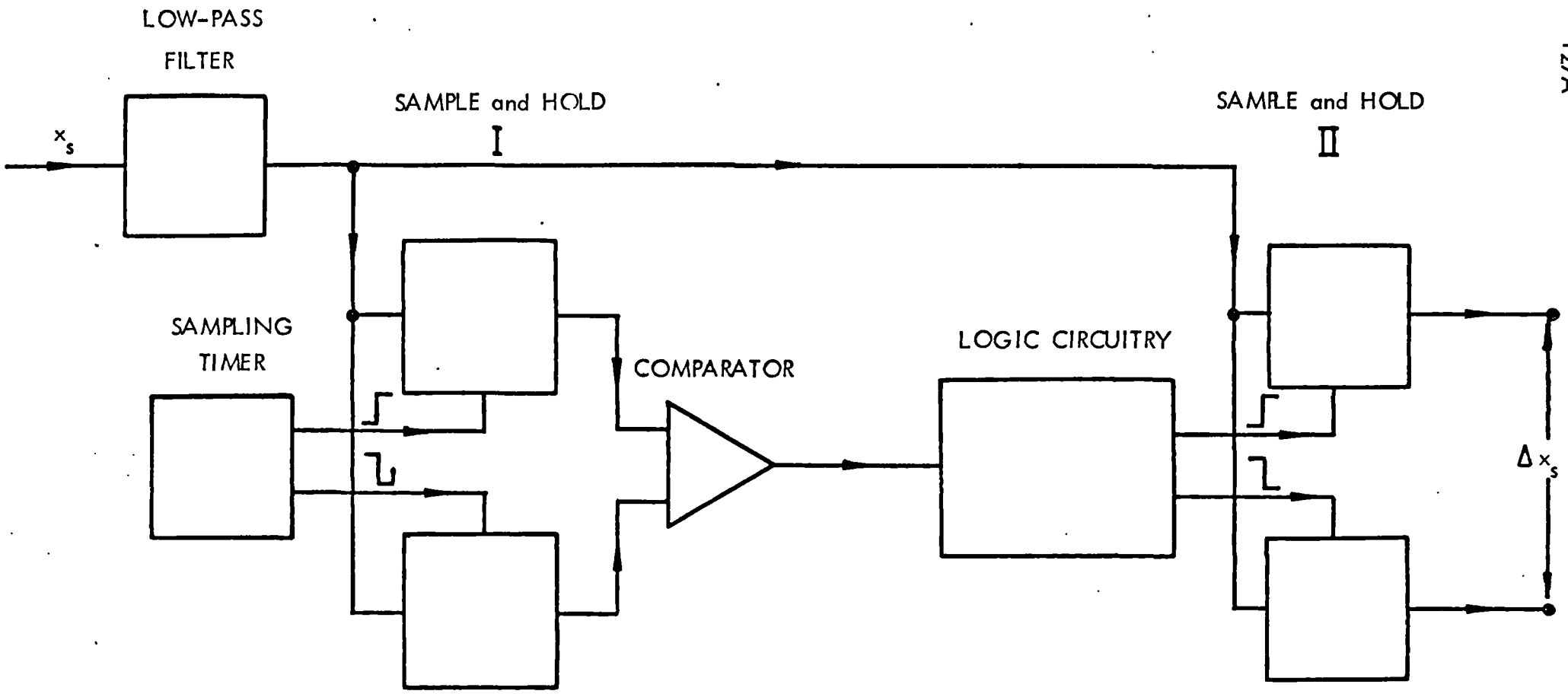
To reduce input signal noise, a low-pass filter with a cut-off frequency of approximately 15 Hz, formed the first stage of the circuit. Both pairs of sample and hold device sampled the output of this filter. However, each pair of devices were triggered to sample asynchronously.

Considering pair I; if the input signal was steady, with no changes in d.c. level, then the output from comparator (C_1) was low. However, if a move occurred, the d.c. level changed, and the comparator (C_1) output was high. By means of logic circuitry this led to the disabling of the second pair (II), of sample and hold devices. When the signal returned to a steady state, the comparator output (C_1) became low and sample and hold units II were re-enabled. However, the devices were storing the signal level as it was prior to the movement. Thus, the first sample "post-move" was compared to the last sample "pre-move", the difference in voltage representing the shift in signal level. In this way, the circuit served to remove the transient component of the signal, the output being exclusively d.c. in nature, and referred to the size of the change in position of the centre of gravity of the patient. Identical circuits were constructed for both X and Y channels.

Details of circuit B are described in Appendix 1.

5.8.3 Circuit C

The inputs to this portion of the circuitry, consisted of two channels which represented the change in magnitude of the vectors $\underline{x}_s$ and $\underline{y}_s$, that is Δx_s and Δy_s per unit subject weight. The function of this section of



A similar circuit was employed to process y_s.

Figure 5.20 Simplified schematic diagram of circuit B.

electronics was to calculate the vector sum of the two, resulting in an output of $\sqrt{\Delta x_s^2 + \Delta y_s^2}$ or, in terms of the signal voltages,

$$\sqrt{\left(\left(\Delta (Vs_1 + Vs_2)\right) .Vx\right)^2 + \left(\left(\Delta (Vs_2 + Vs_3)\right) .Vy\right)^2}$$

(vide 5.6.1, equation 5-9)

Circuit principle: A method was developed which allowed solution of the vector sum function in three stages, namely; squaring, summing, and square rooting (figure 5.21). It was necessary to choose analogue elements capable of fulfilling these functions.

The first stage made use of an analogue multiplier operating in squaring mode. For this particular application an AD534K (Analog Devices, U.S.A.) was selected, with specifications including an untrimmed accuracy of $\pm$ 0.5% max and $\pm$ 0.3% max in multiplier and squarer modes respectively over a signal range of $\pm$ 10 V. However, as was evident from an error analysis (Appendix 1), by means of suitable offset controls, the error may be reduced significantly. The device had fully differential, high impedance inputs which were consistent with the differential type of input provided by B. The output of the squarers was representative of Δx_s^2 and Δy_s^2.

The second stage consisted of an operational amplifier in summing mode, the output of which was an analogue representation of $\Delta x_s^2 + \Delta y_s^2$.

The third stage computed the square root of its input. A multiplier connected in such a manner that the product of its output multiplied by itself is made equal to the input, produced an output which was the square root of the input (figure 5.22a). In this mode a multiplier was subject to a reduction in accuracy and dynamic range, the AD534K was specified in square root mode at $\pm$ 0.5% of full scale, over a signal range of 100 mV to 10V. Any input voltages less than 100mV being subject to increased error. However, a multifunction device, such as the AD433B, connected in square root mode (figure 5.22b) had a specified accuracy of $\pm$ 0.15% of full scale with an input signal of + 10mV to + 10V. When compared with the multiplier this represented a considerable increase in accuracy with a 20 dB improvement in dynamic range. Due to the superior characteristics of the multifunction

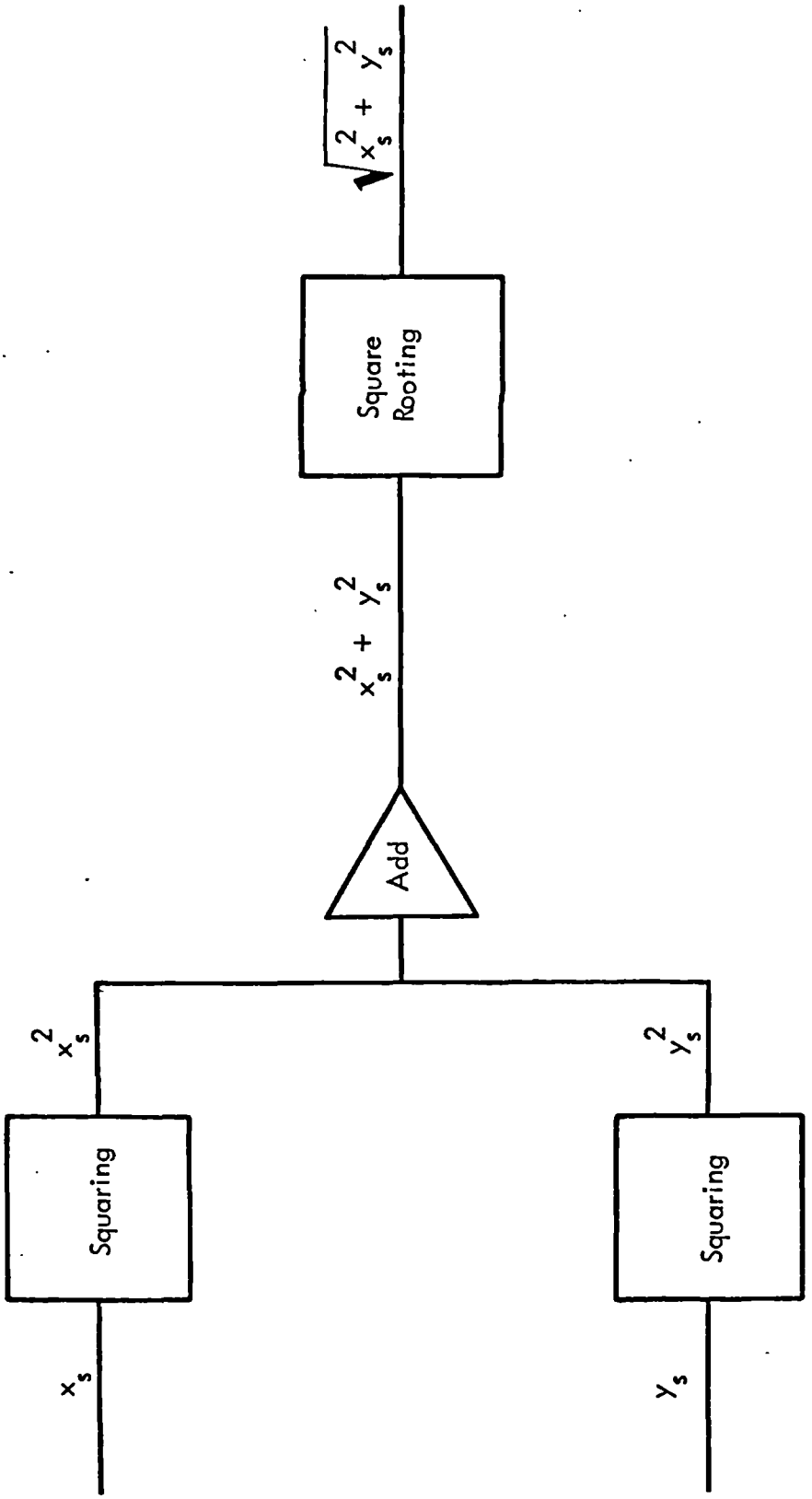
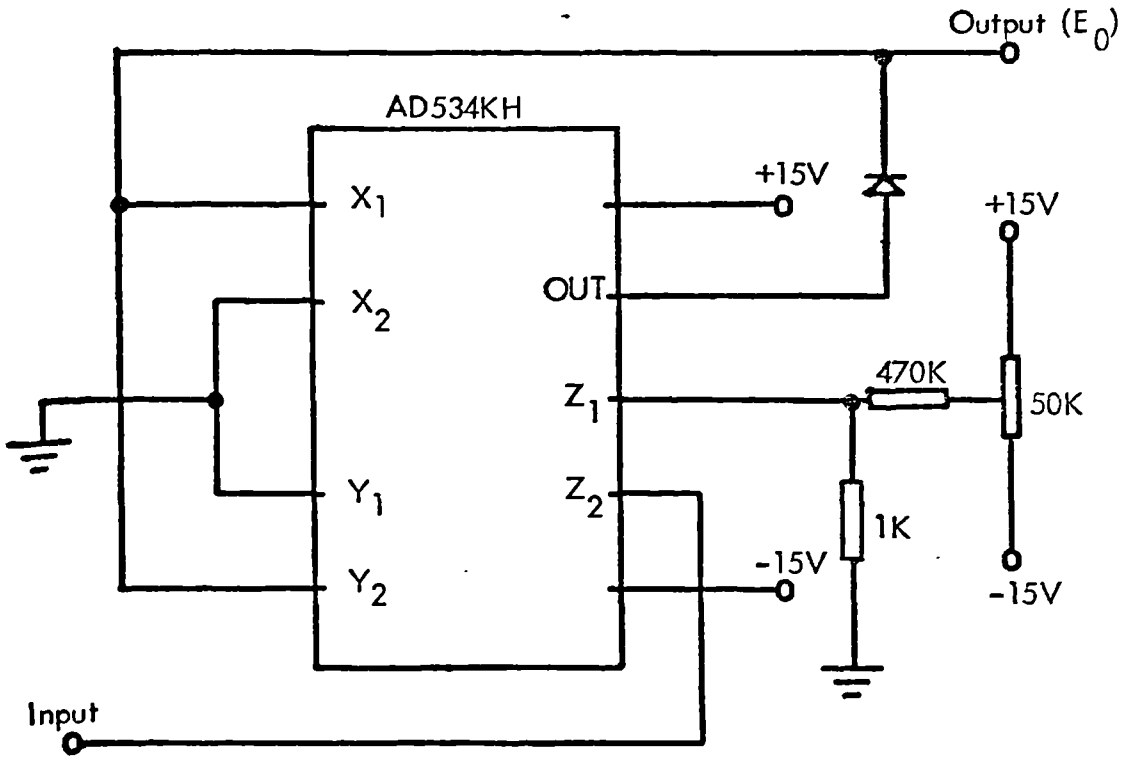


Figure 5.21 Analogue elements involved in computation of the vector sum function.



General transfer function for the AD534KH multiplier is described as;

$$\frac{(X_1-X_2)\,(Y_1-Y_2)}{S.F.} - (Z_1-Z_2)$$

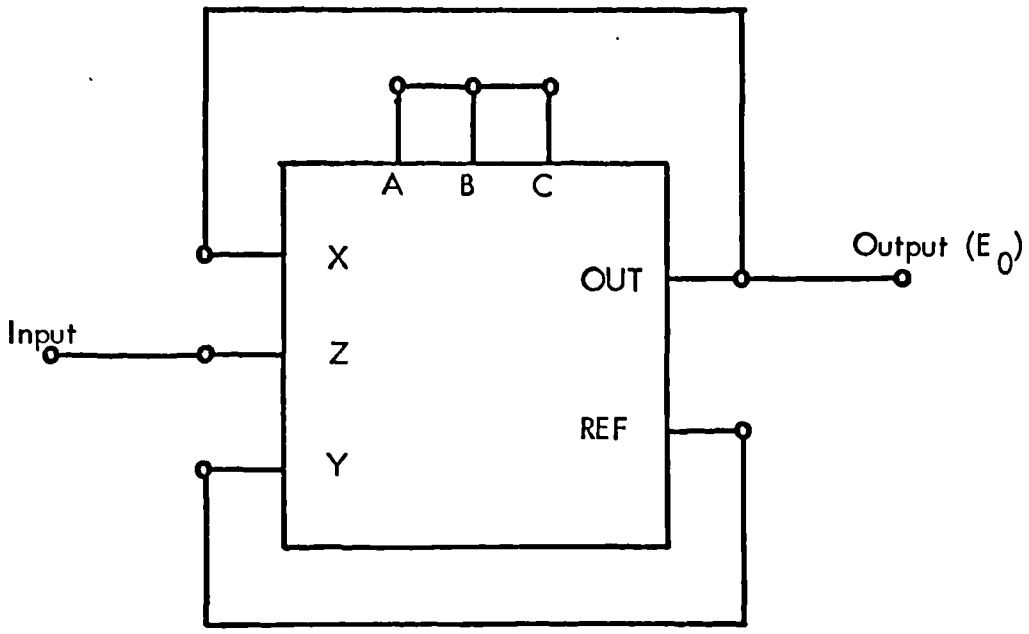
where S.F. = scale factor (untrimmed = 10)

When connected as above, $E_0 = \sqrt{10(Z_2-Z_1)} + X_2$

However, $X_2 = 0$ Therefore $E_0 = \sqrt{10(Z_2-Z_1)}$

Z_1 provides a zero offset trimming control, with Z_2 as input.

Figure 5.22a Computation of a square root function by an analogue multiplier (AD534KH).



General transfer function = $\frac{+10}{9} \cdot V_y \cdot \left(\frac{V_z}{V_x}\right)^m$

By introducing resistances between A, B and C it is possible to program the value of m. However, with a short circuit connection, m = 1

Also, in this configuration $V_y = V_{ref}$ and $V_x = E_0$

$$\text{Thus, } E_0 = 10.\frac{V_z}{E_0}$$

$$\text{or, } E_0 = \sqrt{10V_z}$$

Figure 5.22b Use of a multifunction module (AD433B) to compute a square root function.

module it was selected to perform the square root function.

The output voltage from this final stage represented the vector sum $\sqrt{\Delta x_s^2 + \Delta y_s^2}$, the numerator of equation 5-8.

5.8.4 Circuit D

This circuitry provided an analogue representation of the denominator of equation 5-8. Input consisted of voltages derived from all four load cells, that is Vr_1, Vr_2, Vr_3 and Vr_4, which were summed and offset to produce an output characteristic of the subject's weight S.

Circuit principle: The output from each load cell may be represented as a combination of forces due to the weight of the bed and the weight of the subject in the bed (vide 5.3.1).

Thus,
$$\begin{aligned} Vr_1 &= Vb_1 + Vs_1 \\ Vr_2 &= Vb_2 + Vs_2 \\ Vr_3 &= Vb_3 + Vs_3 \\ Vr_4 &= Vb_4 + Vs_4 \end{aligned} \qquad (5\text{-}13)$$

where Vb_1, Vb_2, Vb_3 and Vb_4 are voltages descriptive of the load in each bedleg associated with the weight of the bed itself.

The required output of the circuit was S which may be represented as $Vs_1 + Vs_2 + Vs_3 + Vs_4$ (vide 5.3.1).

Rearranging equations 5-13 gives,

$$Vs_1 + Vs_2 + Vs_3 + Vs_4 = (Vr_1 + Vr_2 + Vr_3 + Vr_4) - (Vb_1 + Vb_2 + Vb_3 + Vb_4)$$

or
$$\sum_{i=1}^{4} Vsi = \sum_{i=1}^{4} Vri - \sum_{i=1}^{4} Vbi \qquad (5\text{-}14)$$

Thus the circuit summed the outputs from the load cells (Vri) and by means of an offset facility subtracted the weight of the bed ($\sum_{i=1}^{4} Vbi$), leaving the required $\sum_{i=1}^{4} Vsi$.

Circuit D consisted of three stages (figure 5.23). The first stage consisted of four operational amplifiers in differential mode with gain. These were used to directly process the output signals from the load cells. Due to

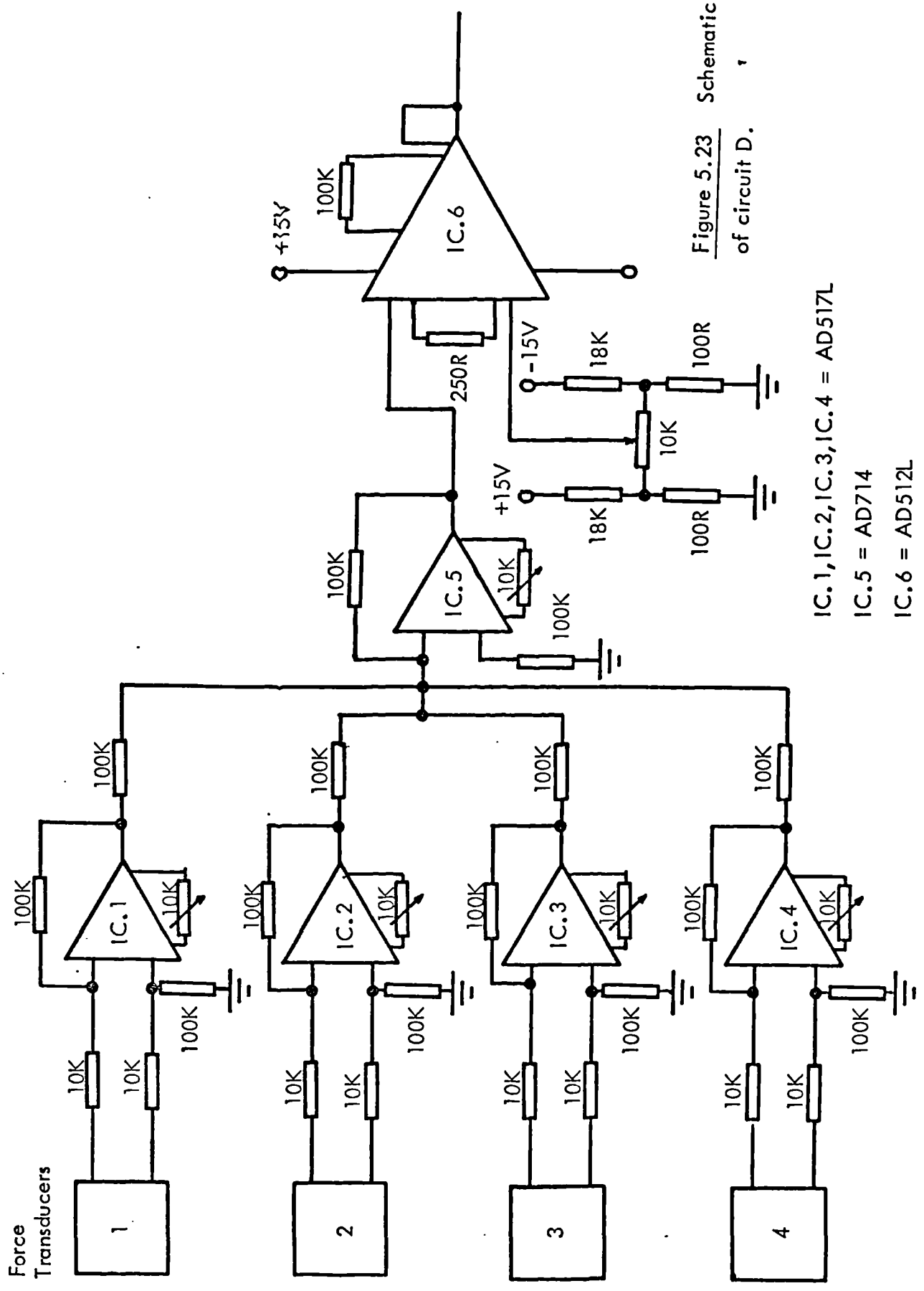


Figure 5.23 Schematic diagram of circuit D.

the small signal size it was necessary to choose amplifiers with low noise and low thermal drift characteristics. AD517L operational amplifiers were selected, with specifications of 2μVp-p noise and 0.5μV/^{o}C thermal drift. A gain of 10 on the input signal was produced in this stage and input offset zeroing was provided.

The second stage employed a conventional operational amplifier in summing mode with unity gain.

The third stage was in the form of an AD521K instrumentation amplifier (Analog Devices, U.S.A.) capable of providing gains in the range of 0.1 to 1000. The non-inverting input of the amplifier was connected to a pair of potential dividers providing a voltage swing of $\pm$ 100 mV. Adjustment of this voltage enabled the output of the amplifier to be offset. The gain of the amplifier was selected empirically as 390, producing output signals of a suitable magnitude for further analysis. In combination with the first stage gain of 10, the total gain of the circuit was 3900.

Zeroing the output from the final stage when the patient was out of the bed,reduced $\sum_{i=1}^{4}$ Vbi to zero (equation 5-14). When the subject was returned to the bed the output from D was representative of their body weight, S (equation 5-8).

5.8.5 Circuit E

The last section of the processing electronics combined the outputs of C and D by the use of an analogue divider, to produce the ratio $\frac{C}{D}$ (figure 5.18). The device chosen was a 4291H dedicated divider (Burr Brown, U.S.A.) which utilised a log - antilogue principle. Detailed description of the selection procedure for the divider is presented in Appendix 1. This device was capable of an accuracy to within $\pm$ 0.5% over a dynamic range of 40 dB signal range of the numerator, the transfer function descriptive of the output of the divider in relation to its input was $10\frac{N}{D}$, when N = numerator, D = denominator.

The input voltage range was specified as,

$$-10V \leq N \leq +10V$$

and $+100\ mV \leqslant D \leqslant +10V$

Thus the numerator, represented by the vector sum of the coordinates defining the position of the subject's centre of gravity in bed per unit subject weight, could be of dual polarity within a 10V range. The denominator, representing the subject weight S, was of single polarity and required to be within the + 100 mV to + 10V range.

Knowing the dynamic range of the numerator, the gain of D, the force transducer sensitivity and the strain gauge bridge energisation voltage, it was possible to derive an associated range of subject weight.

Force transducer sensitivity (F.T.S.) = 0.425μV/V/N (vide 5.5.2)

With an energisation voltage of 5V (vide 5.7.1)

$$F.T.S. = 0.425 \times 5\mu V/N$$
$$= 2.13\mu V/N$$

Gain of D = 3900

Allowable output range of D (as defined by the divider specifications)

= 100mV to + 10V

Thus, the combined output of all four force transducers could range from

$$\frac{100 \times 10^{-3}}{3900} \text{ V to } \frac{+10}{3900} \text{ V}$$

or

$$2.6 \times 6 \times 10^{-5} \text{ V to } 2.6 \times 10^{-3} \text{ V}$$

In terms of the load applied to the transducers this is

$$\frac{2.6 \times 10^{-5}}{2.13 \times 10^{-6}} \text{ N to } \frac{2.6 \times 10^{-3}}{2.13 \times 10^{-6}} \text{ N}$$

or

12.2 N to 1221 N

Thus, the "on scale" range of subject "weight" was 12.2 N to 1221 N or 1.22 kg to 122 kg. This compared favourably with earlier specifications for the overall system (vide 5.2).

5.9 POWER SUPPLY

The critical criteria for selection of a power supply were considered to be:

Voltage output: The majority of the components in the analogue circuitry had a wide operating range of power supply input voltages. However, there were two exceptions, these being the 4291H divider and the ICL 7650 chopper amplifier. The divider had a specified power supply voltage range of $\pm$ 14V d.c. to $\pm$ 16V d.c. and the chopper amplifier one of $\pm$ 8V d.c. maximum. The selection of a power supply capable of providing a $\pm$ 15V output satisfied the demands of the divider. By the use of voltage regulators, or another form of voltage divider, the supply voltage to the ICL 7650 chopper amplifier was adjusted to within the $\pm$ 8V maximum.

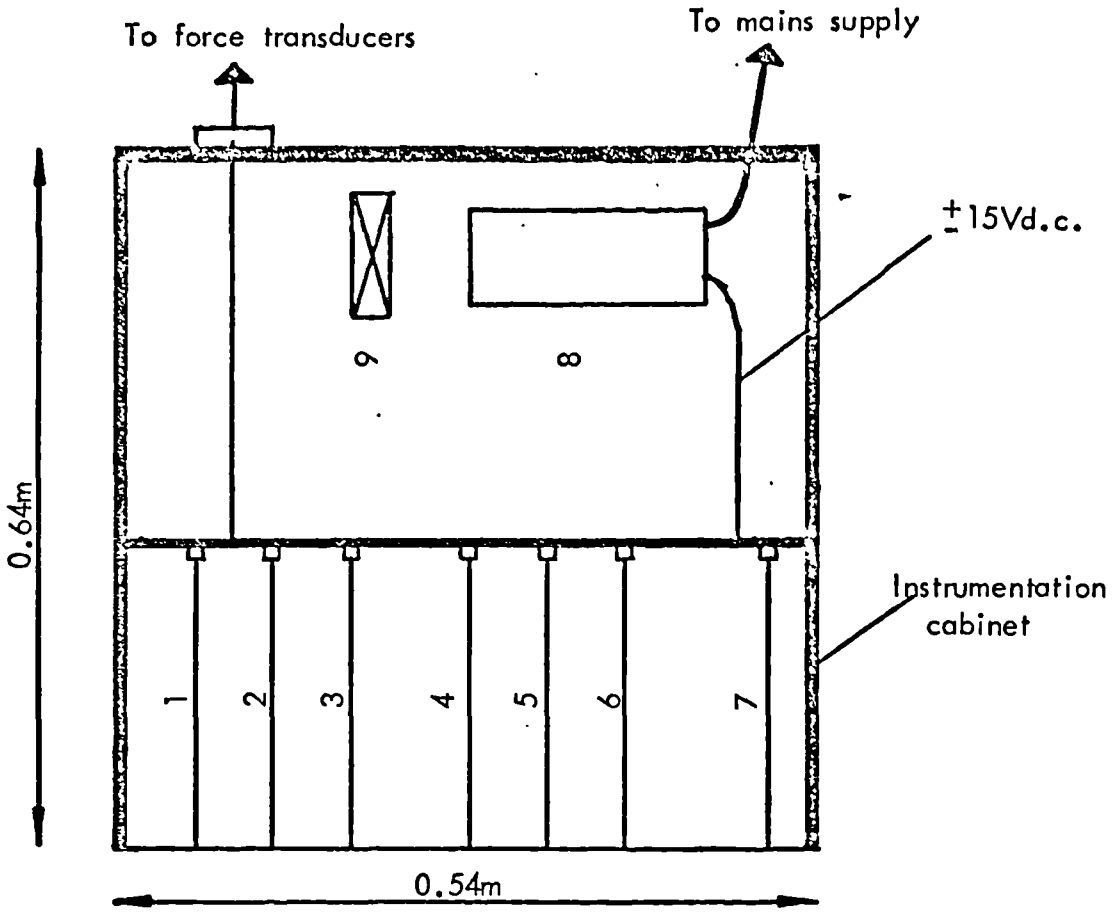
Current capability: The power supply had to be capable of yielding sufficient current to energise all of the active components. The overall current requirement for the system was estimated by summing the individual current requisites of each of the components in the analogue circuitry. The total current requirement of the power supply was thus estimated to be approximately 200mA.

Noise: Any perturbations in the power supply voltage may lead to a degradation of the characteristics of the components it is supplying. Determination of the critical level of noise in the power supply signal was dependent upon both the common mode rejection ratio (CMRR) (vide 5.7.2) and input signal level of the components it was powering.

From equation 5-10

$$C.M.R.R. = \frac{\text{output voltage / unit difference voltage}}{\text{common mode signal}}$$

For this particular case, the power supply output noise was considered to represent the numerator, with the denominator being representational of the required signal resolution of a component, energised by the power supply. The input signal resolution will be related to the minimum input signal of any component, experienced during normal use.



1 = Strain gauge amplifier for force transducer 1

2 = Strain gauge amplifier for force transducer 2

3 = Strain gauge amplifier for force transducer 3

4 = Circuits A, C and E

5 =
6 =] Circuit B

7 = Circuit D

8 = Power supply

9 = Fan

Figure 5.24 Layout of circuit boards and power supply, within the instrumentation cabinet.

Consider the differential amplifiers which formed the first stage of circuit D. Their input was provided by the strain gauge bridges on the load cells and ranged from approximately 20μV to 2000μV, depending upon the size of the movement of the subject in the bed. Thus the minimum signal resolution of the amplifiers was required to be better than 20μV. A resolution of 5μV ensures accuracy. The AD517L amplifiers employed at this stage of the circuit had a specified CMRR of over 90 dB for a frequency range of 0 to 1.5 kHz.

The magnitude of noise in the power supply output to produce a 5μV input in the differential amplifier may be represented as ;

Magnitude of noise = Required minimum signal resolution x C.M.R.R.

$$= 5 \times 10^{-6} \times \text{antilog } \frac{90}{20}$$

$$= 0.158\text{V or } 158\text{mV}$$

Thus the noise associated with power supply output must be less than 150mV.

Using these criteria a PC500C15/15 (ITT, Harlow, U.K.) power supply unit was chosen. This provided an output voltage, adjustable in the range $\pm$ 12V to $\pm$ 15V. To ensure that the "common" (zero) voltage was not offset and lay equally between the dual polarity outputs, a balance control was provided. For this application the device supplied + 15.00V and - 15.00V, with 500mA per channel. The noise, and ripple, on the output lines were specified as 10mVp-p (Appendix 1).

The printed circuit board (P.C.B.), on which the power supply was mounted, was protected by an aluminium cover, which also served as a heat sink. To reduce thermal effects, resulting from the power supply, it was mounted separately in the rear of the instrument cabinet. Additionally, an extractor fan was provided to promote a rapid thermal equilibrium (figure 5.24).

5.10 DATA RECORDING

The output from the processing system was recorded on magnetic tape and replayed into a PDP12 computer.

The major requirements for the data recording device were:

Long duration, continuous recording: To conform with the overall specifications for the mobility monitoring system (vide 5.2.1), the tape recorder was required to have the capability of at least 10 hours continuous recording.

Bandwidth of d.c. - 25Hz: The output signal from the processing electronics could be considered to be composed of two components. The first represented the output when the subject was immobile and was unchanging. The second was a result of a move by the subject in the bed and was in the form of a 40ms voltage pulse, the magnitude of which was indicative of the size of the move.

Signal to noise ratio of 35 dB: The dynamic range of the output signal from the analogue processing system was approximately 26 dB (for a minimum system resolution of ±50 mm move size). The range of the differential source signal was 3 dB. Thus, the required dynamic range of the tape recorder was 26 dB + 3 dB = 29 dB. To permit signal recognition at the lower end of the amplitude range of the signal, the magnitude of the noise was required to be at least 6 dB lower than that of the signal (ie 1% of full scale). Thus the necessary signal-to-noise ratio was approximately 29 dB + 6 dB = 35 dB.

Accelerated replay: Considering the duration of the experiments to be 10 hours, it was essential that the tape recorder be capable of an accelerated replay speed of at least 30 times that of the record speed. With this facility a 10 hour sleep record could be replayed into the computer in 20minutes.

Linearity: The overall acceptable inaccuracy of amplitude measurement of both the processing electronics and the tape recorder errors combined, was nominally $\pm$ 2%. The inaccuracy in the signal output from the processing electronics was of the order of $\pm$ 1% (vide 5.11). By selecting a signal to noise ratio of 6 dB the maximum error attributable to noise, was $\pm$ 0.5%.

Thus, the acceptable inaccuracy from the non-linear amplitude transfer function of the tape recorder was ($\pm$ 2%) - ($\pm$ 1%) - ($\pm$ 0.5%) = $\pm$ 0.5%.

A Racal Store 4 FM magnetic tape recorder (Racal-Thermionic Ltd., Southampton, U.K.) was selected on the basis of the design requirements. This device allowed continuous recording for up to 18 hours on 4 channels or up to 72 hours on just one channel. At the slowest recording speed (24 mm/s) the frequency response of the recorder was d.c. to 313 Hz with a signal to noise ratio of 45 dB. The system non-linearity was specified as $\pm$ 0.3%. The tape recorder had a range of 7 speeds from 0.152 m/s to 24 mm/s providing a possible accelerated replay of 63 times real time.

5.11 CALIBRATION

Details of the calibration procedure are presented in Appendix 2. Results of the experiments are summarised in the following section.

The response of the processing electronics was determined to be linear (s = 0.99), although an error was apparent when measuring moves in different directions, this variation was within 2%.

For the ideal case, the system was independent of subject weight. At low loads (less than 20 kg) the output error increased significantly up to $\pm$ 8% for a 10 kg load. This additional error was attributed to the impaired response, at low signal input levels, of the analogue components within the system, particularly the divider. However, for loads greater than the minimum design load of 30 kg the system's output was consistent.

The sensitivity of the apparatus, that is the ratio of system output to the magnitude of a centre of gravity shift by the patient, was calculated to be 14mV/mm (with 5V energisation of the strain gauge bridges). The maximum move size that could be measured was 0.27 m.

Performance assessment of the analogue processing system may be summarised as:

Highly linear input/output characteristic	(s = 0.99)
Variation in output for different loading direction	within 2%
System output dependency, related to subject weight	
a) Greater than 30 kg	independent
b) less than 30 kg	dependent, increasing with reduction in weight ($\pm$ 8% for a 10 kg load)
Sensitivity	14mV/mm
Maximum move size	0.27m
Bandwidth	d.c. only
Error attributable to magnetic tape recorder	$\pm$ 0.3%
Overall system error	2.6%

The results of the calibration indicated that the mobility monitoring system was capable of accurately measuring the magnitude of a move of the centre of gravity of a subject in the bed, independent of the body weight of the subject.

5.12 SUMMARY

A review of the literature, describing methods that have previously been developed to measure the mobility of a subject in bed, established that no technique was available that would be suitable for this application. As a result a system was designed and constructed which was capable of fulfilling the design criteria established for monitoring the mobility of elderly patients in the clinical environment.

The design principle selected required the measurement of the force transmitted to the floor by each of the bedlegs. Thus, a force transducer was designed which was capable of sustaining the combined load of the bed and the patient and was suitable for use in the clinical situation. The measuring element of the transducer was in the form of a pair of strain gauged cantilever beams. Calibration of the transducer indicated a sensitivity of 0.425μV/V/N.

The load cells provided the input to a computational, analogue electronic circuit which processed the input signals from the force transducers, to produce an output representative of the magnitude of a shift of centre of gravity of a subject in bed.

Data recording was effected by the use of an analogue tape recorder, which was capable of continuous recording for the duration of the experiment. Using an accelerated replay facility, the stored data was input to a PDP12 computer.

Calibration of the complete mobility monitoring apparatus indicated an accuracy within 3% and a system independent of subject weight.

Figure 5.25 shows the instrumentation in position beside a patient's bed in a hospital ward.

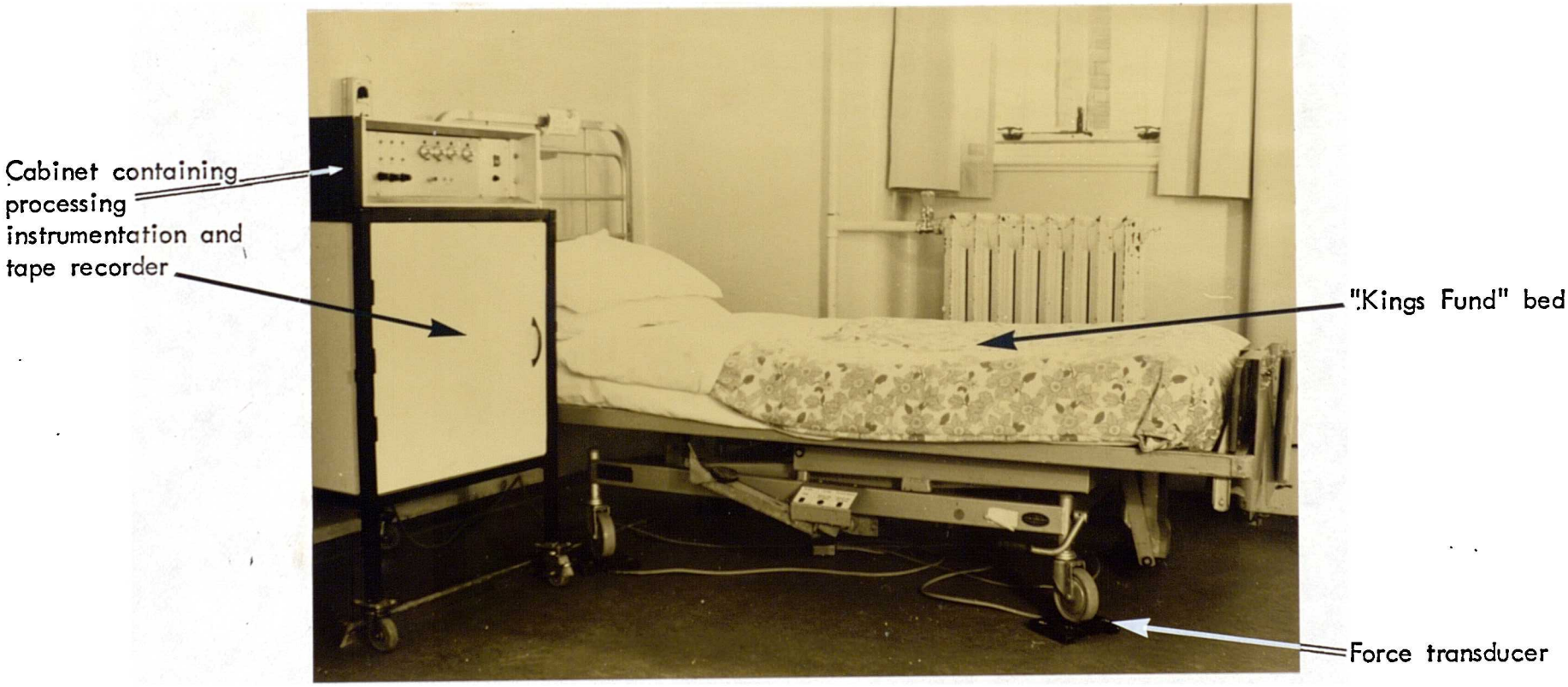


Figure 5.25 The mobility monitoring system in position in the hospital.

CHAPTER 6

DEVELOPMENT OF PATIENT ASSESSMENT AND EXPERIMENTAL PROTOCOL

6.1 INTRODUCTION

In Chapter 2 some of the factors which have been proposed as being of importance in the aetiology of pressure sores were described. To provide background information on the patients participating in this study, a form was developed to record these factors. Additional information was recorded relating to the nurse's assessment of the patient's susceptibility to pressure sores.

Chapter 5 described the design and development of an instrumentation system, for measuring the mobility of patients in bed, overnight. As this system was to be utilised in the clinical environment, it was particularly necessary to develop an experimental protocol which caused the least possible disruption to the patients, the nursing staff and the day to day routine of the hospital.

Techniques were developed to provide analysis of the recorded information, these included computer programs and graphical displays.

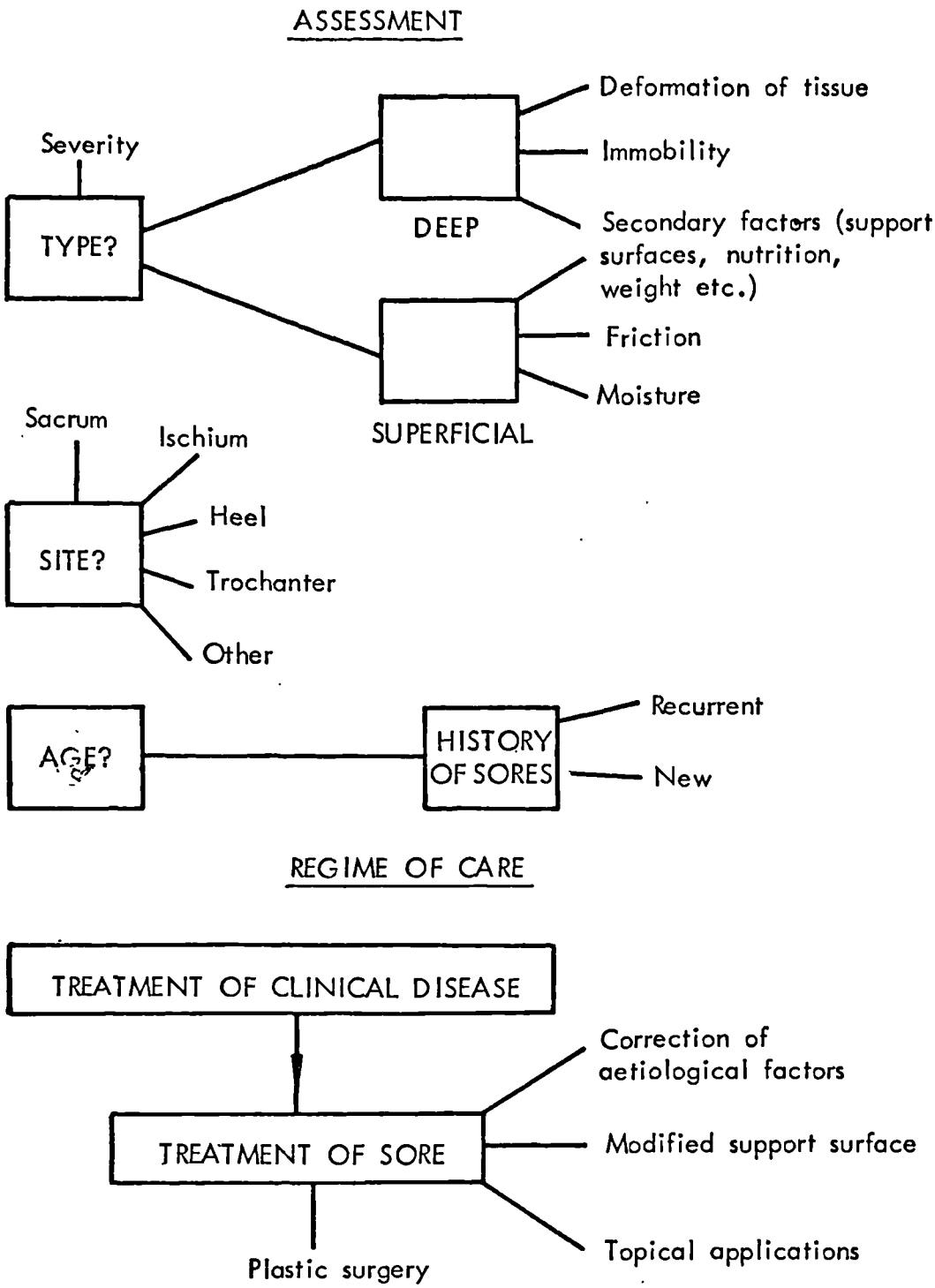


Figure 6.1 Assessment and care of patients already suffering from a pressure sore.

6.2 PRESENT METHODS OF PATIENT ASSESSMENT IN RELATION TO PRESSURE SORES

Assessment of patients with regard to pressure sores is related to their clinical condition. Those patients with a sore, are assessed in relation to the reasons for the sore developing and the optimum method of treatment. Patients exhibiting a recent tissue injury, but no sore, are assessed in an effort to determine the site and severity of the injury. Patients with normal soft tissues are assessed in order to distinguish those most likely to be susceptible to pressure sores.

6.2.1 Assessment of patients with pressure sores

Figure 6.1 illustrates the two stage procedure in the assessment of patients who have already developed sores.

Firstly the sore is evaluated with regard to its type. That is, whether it is deep, involving lower layers of soft tissue such as the muscle, or superficial involving only the epidermis and dermis of the skin. The site of the sore is established and any previous history of pressure sores, particularly in the area of the present injury, is noted. Aetiological factors associated with its formation are determined. Typically a deep sore will be related to tissue deformation associated with a reduction in mobility and a superficial sore from the affects of friction and moisture. In addition, these "primary" factors may be supplemented by a wide range of other "secondary" factors such as incontinence and poor nutrition, (vide 2.1.1 and 2.1.2).

Secondly, assessment of the patient promotes a particular regime of care. This will involve an attempt to correct those aetiological factors responsible for the development of the sore allied with clinical treatment including, topical applications, plastic surgery, modified support surface etc.

6.2.2 Assessment of patients with recent tissue injury

Such patients have no fully developed pressure sore lesion but have soft tissue damage which may be a centre for the development of a sore. This may manifest itself as a patch of persistent red skin, noticeable to an

observant nurse, or as an injury to deeper layers of the soft tissue such as the muscle, which produces no visual surface change and thus cannot be identified by eye. Such deep soft tissue injury has been assessed using techniques involving thermography. Davies and Newman (1981) used a thermal camera to "screen" patients newly admitted to a geriatric ward,for recent deep tissue injury. Video tape recordings of a thermogram of the buttock region of these patients were made immediately after admission and regularly during the following 12 weeks. Using this technique Davis and Newman (1981) were able to predict which patients were liable to pressure sores. Nilsson (1975) showed that thermography measures surface skin temperature disributions, which may be abnormal due to disruption of deep tissues.

6.2.3 Assessment of patients with no pressure sores and no recent tissue injury

It is this group of patients who are of most interest to this study. In their case, assessment serves to aid prophylaxis and allows identification of those patients "at risk", who can then be assigned pressure sore preventive care.

Torrance (1981) suggests the use of a "team approach" in the care and prevention of pressure sores. Although the nurses look after the patients on a day to day basis, the clinical staff should also play an important part in the identification of those at risk. The experience of an established member of the medical staff (doctor, nurse, physiotherapist etc.) provides probably the best form of patient assessment, and is the most widely used method of determining those who are "at risk".

However, identification of patients particularly vulnerable to developing pressure sores is possible, even by the inexperienced, if their condition is extreme. For example, an undernourished, wasted, incontinent old person is an obvious risk. A gradual change in the condition of a patient, particularly if they are elderly, previously determined not to be "at risk" may be less obvious. As their condition changes, the patient may become gradually more and more susceptible to pressure sores and by the time

The 'risk' of developing a pressure sore based on the nurses decision using the NORTON SCORE.

This score is based on the use of five categories, all relevant to the development of pressure sores. Each category has four grades. The grade relevant for the patient on day of assessment should be put into the appropriate box.

(N.B. 'Physical Condition, means general state at the time and not the degree of disease or prognosis).

DATE OF ASSESSMENT :

A.	PHYSICAL CONDITION :	Good	= 4	
		Fair	= 3	☐
		Poor	= 2	
		V. Bad	= 1	
B.	MENTAL CONDITION :	Alert	= 4	
		Apathetic	= 3	☐
		Confused	= 2	
		Stuporous	= 1	
C.	ACTIVITY :	Ambulant	= 4	
		Walk/Help	= 3	☐
		Chairbound	= 2	
		Bedfast	= 1	
D.	MOBILITY :	Full	= 4	
		Sl. limited	= 3	☐
		V. limited	= 2	
		Immobile	= 1	
E.	INCONTINENT :	Not	= 4	
		Occasionally	= 3	☐
		Usually	= 2	
		Doubly	= 1	
	TOTAL SCORE		=	☐

A score of 14 or below is classed as 'at risk'

Figure 6.2 The Norton score assessment form.

it is realised that a change has ensued, damage may already have occurred to the tissues.

To assist in the identification of potentially susceptible patients, Norton et al (1962) suggested the use of a risk assessment form, which was simple to use and easily interpreted. The form consists of five sections, each describing an aspect of the patient's condition associated with the aetiology of pressure sores (figure 6.2). The patient is assessed in relation to each of the sections and the record form is completed, grading each of the parameters with a score ranging from 1 (very poor) to 4 (normal). Thus, the maximum total is 20 and the minimum is 5. Norton et al (1962) correlated the incidence of pressure sores with a low score on the assessment scale. A graph of incidence of sores against initial score (figure 6.3) showed a primarily linear relationship illustrating the predictive power of the method. Clinical studies showed that a score of around 12 should be considered as the threshold below which patients are "at risk" of developing pressure sores. Norton suggested that patients with a total of 14 or less be considered for extra nursing care and the application of prophylactic aids. She also recommended that patients be assessed on the first two days after admission and thereafter at regular intervals, provided that they have a high enough score and that there is no deterioration in their condition, or in their score.

Any assessment of a patient's condition using the Norton scoring technique is an individual judgement and is necessarily subjective, although different observers, scoring the same patient, have been found to obtain similar results. The limited number of parameters considered on the assessment form suggests that while being of great use in the majority of cases, it cannot be considered as a rigorous assessment technique, and should be used only as a first step in the care of a patient.

Other variations of the Norton score have been developed (Bliss and McLaren, 1966; Gosnell, 1973; Lowthian, 1975; Bliss, 1981). However, all of them employ the same general format as the Norton scale, including assessment of a patient's susceptibility to pressure sores in relation to a number of variables, and culminate in a numerical score.

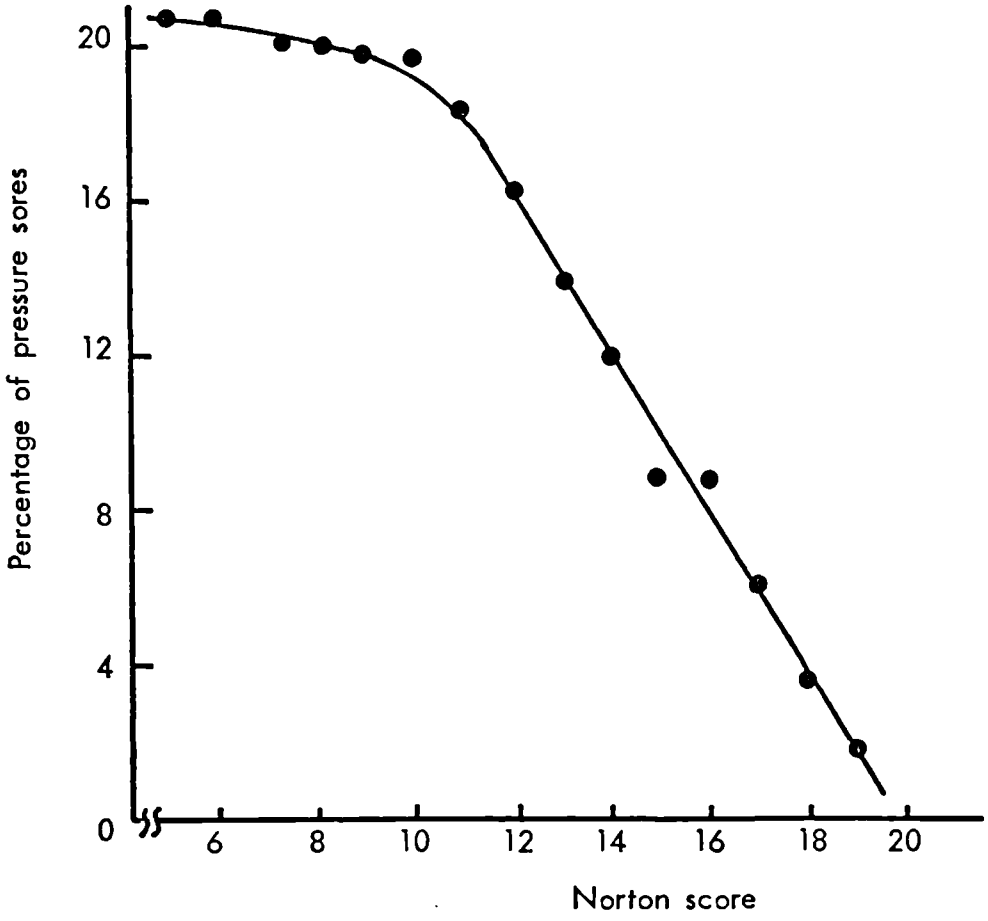


Figure 6.3 The percentage of patients with pressure sores in relation to their Norton score (300 patients).
(after Norton et al, 1962)

6.3 PATIENT DATA RECORDING

Documentation was developed for this study to provide a simple format with which it was possible to characterise the general condition of the patient, to record how this varied during the period of the investigation, and to obtain detailed information on specific factors which may predispose to pressure sores(vide 2.1.3). This included details of the patient's clinical condition and proposed treatment. A typical patient record form used in this study is presented in Appendix 3. A section of the record was dedicated to the status of the patient with regard to pressure sores. If the patient being monitored had a pressure sore, note was made of its position on the body. In addition the severity of each sore (or sores) was described using the grading system established by Barbenel et al (1977).

Limitations of this system as suggested in section 2.2 were understood. However, to provide continuity with earlier work, particularly that of the epidemiological study of Barbenel et al (1977) a similar grading system was used.

Any treatment specific to the pressure sore was noted. This included the use of special support surfaces such as the ripple mattress, the application of any topical agent, such as Debrisan, or aserbine lotion or simply regular relief of pressure by turning. The history of any previous pressure sores was noted.

Diagnosis of the patient was recorded as one of a list of diseases taken from the International Classification of Diseases (1978). Similarly any drugs used in the treatment of the patient were registered against a list, in this case reproduced from MIMS (1980). A further section of the documentation was concerned with the support surface and included details of the mattress type, cover and thickness, and of the bed clothing.

The patient record booklet was completed by three groups of people. Clinicians, provided information on the clinical condition, diagnosis and proposed treatment including drug therapy, the experimenter made note of the test number and details of the support surface and the nursing staff completed the sections relating to the presence of pressure sores, pressure

sore treatment, continence and posture.

In addition to the patient data booklet a daily record was kept of the patient throughout the period of monitoring. This supplied information on the activity of the patient, drugs administered in the evening, prior to the sleep period and of body temperature. The nursing staff were also asked to make a daily assessment of the patients susceptibility to pressure sores using the Norton score (vide 6.2).

6.4 SELECTION OF SUBJECTS

Subjects for this study were drawn from a geriatric assessment ward at Stobhill Hospital, Springburn, Glasgow. To avoid disruption to another part of the hospital and to facilitate communication with the medical staff, all of the experiments were performed in the same ward and as a result, all of the patients studied were female.

The average duration of stay in the ward was two weeks. Patients were monitored on their first night after admission and for the six nights following this. Suitable patients were usually mentally stable and not prone to dementia, seizures or persistent spasm. Selection of patients with a particular diagnosis was difficult, since in many cases the patient was suffering from more than one of the conditions defined in the International Classification of Diseases (1978). However, in general the patients selected were suffering from either a disease of the circulatory system or a disease of the nervous system. The patient's bed was required to be of a conventional design, in particular water beds were unsuitable due to their excessive weight.

6.5 EXPERIMENTAL PROTOCOL

On the afternoon previous to the first night's recording the four force transducers were placed underneath the bedlegs. A scissor jack was used to gently lift each leg in turn, off the floor by about 70 mm, facilitating the positioning of the transducer. Care was taken to ensure that the castor wheel fitted on to the loading surface of the transducer. Each device was numbered and was related to a particular leg of the bed (vide 5.3, figure 5.1). The cables leading from each of the four transducers were brought to a junction box, placed underneath the bed and from there a single lead connected to the processing apparatus. Brakes, if fitted, were applied to the bed wheels.

The analogue F.M. tape recorder (vide 5.10) was placed inside the instrument cabinet, which also housed the mobility monitoring instrumentation, and was rewound to the start of the tape. The output cable from the processing electronics was connected to one of the recorders input channels, the input voltage range was adjusted to 2 volts full scale deflection and the speed to 24 mm/s (15/16 i.p.s.). The cabinet, containing processing instrumentation and the tape recorder was then wheeled into position near the patient's bed, and the load cells were connected to the instrumentation.

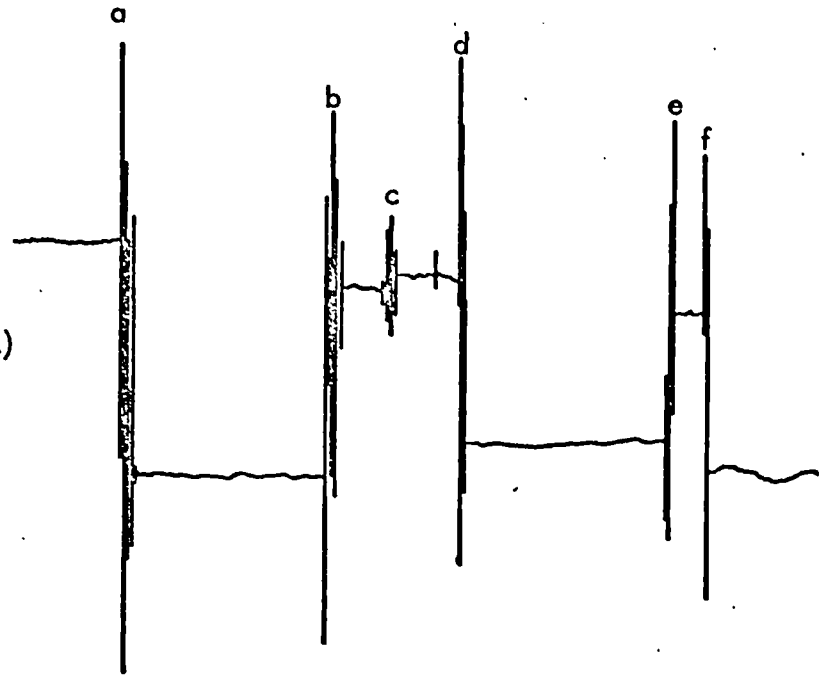
The processing electronics was then switched on and allowed to warm up for a period of 30 to 40 minutes. To provide cooling for the components of the processing system, a small fan was mounted in the instrumentation case (vide 5.9). The level of noise produced by this device was generally found to be inaudible above the background level in the ward. With the patient out of bed, the output of the weight calibration circuitry (vide 5.8.4) was zeroed by means of a potentiometer mounted on the front panel. The position of the potentiometer was then locked and provided that no changes were made to the bed (addition or removal of bed clothes, ripple mattress etc.) this remained unaltered during the series of tests. The patient was then returned to bed, and when positioned approximately in the centre of the bed, the outputs from the strain gauge amplifiers were separately zeroed. At this stage the equipment was ready to start the test. The system was left until approximately 10 o'clock in the evening, when the patient had settled down.

to sleep. At this time the tape recorder was adjusted to record mode and the experiment was started. Some of the later tests incorporated a timing device which automatically energised the tape recorder and therefore relieved the experimenter of the need to travel to the hospital each evening to begin the experiment. Additionally, when used, the device stopped the recording in the morning. The ward sister was provided with a patient data booklet at the start of the series of tests, to be completed by the patient's clinician and by the nursing staff on a daily basis.

The apparatus was switched off by the nurses when the patient was woken up the following morning, usually around 6 am. The tape recorder was then removed and taken to the Bioengineering Unit, where the recorded data was replayed into a P.D.P. 12 computer (vide 6.6.1).

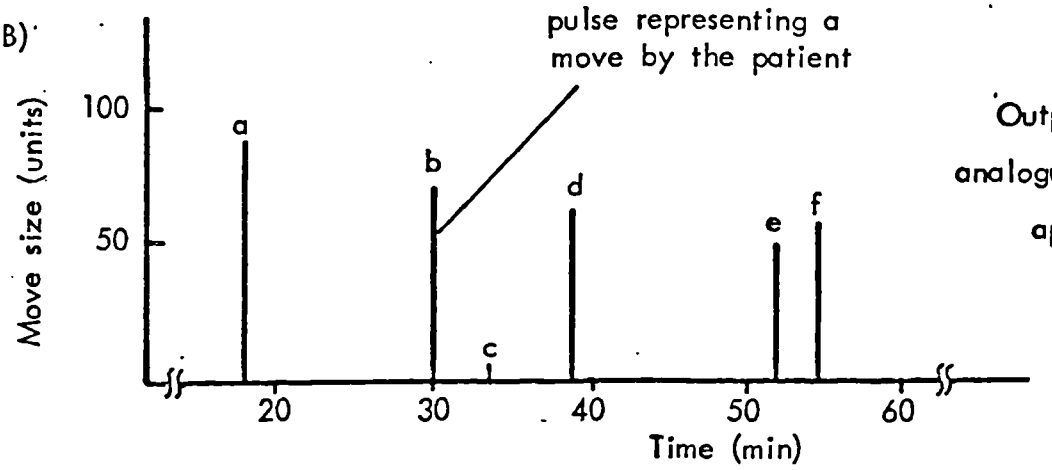
The following afternoon the tape recorder was returned to the hospital, replaced in the cabinet and the equipment adjusted as previously described. However, no alteration was made to the position of the potentiometer associated with the weight circuit. The nursing record, relating to the patient being studied, was read and relevant comments (description of the night's sleep, episodes of incontinence etc.) were added to the patient data booklet.

(A)



Output from load cells

(B)



Output from the analogue processing apparatus

(C)

	Time of move (min)	Size of move (units)
a	18	86
b	30	72
c	33	9
d	39	63
e	52	49
f	55	57

Final output format after editing and digitising of the data

Figure 6.4 Conversion of raw data including d.c. shifts and transients, to tabular format, specifying the magnitude of centre of gravity movements and their time of occurence.

6.6. ANALYSIS OF DATA

The analogue processing unit, described in Chapter 5, served to manipulate the signal collected from the load cells, consisting of d.c. shifts and transients (figure 6.4.(A)) to a form consisting of discrete pulses, the magnitude of which was related to the size of centre of gravity movement (figure 6.4 (B)).This "processed" signal was then analysed in two stages.

6.6.1 Editing of the overnight record

The output from the mobility monitoring system, recorded on magnetic tape, was replayed into a P.D.P. 12 computer which served to digitise and store it. A sampling frequency of 5 milliseconds was found to produce sufficient resolution, while minimising the volume of data. However, even with optimum selection of the sampling interval, the long duration of the experiment gave rise to a large quantity of data, requiring a complete "LINC" tape (250 Kilobytes), for each night's recording. Of this, the greater majority was related to "quiet" periods during the night, when the patient was lying still without moving. For practical and economic reasons it was necessary to reduce this data, facilitating storage and further analysis. To achieve this an editing procedure was employed. The data was displayed on the V.D.U. of the P.D.P. 12 computer (the information displayed over the width of the screen was approximately $1\frac{1}{2}$ minutes, real time). Data format consisted of a baseline (quiescent) with movements in the form of voltage pulses, occurring throughout the night. The editing procedure was contained within the program EDITOR. Each move was visually identified, a cursor was positioned on the baseline, immediately preceeding the pulse and a second on the top of the pulse. The position of each cursor was defined in terms of a co-ordinate system related to the computer and scaled in arbitrary units. The co-ordinates of the two cursors and their temporal position was stored on a further "LINC" tape. This process was repeated for every move, recorded. Thus, the "storage" tape contained a series of pairs of co-ordinates, associated with each move that occurred during the night. In this manner all of the data collected when the patient was lying still was discarded.

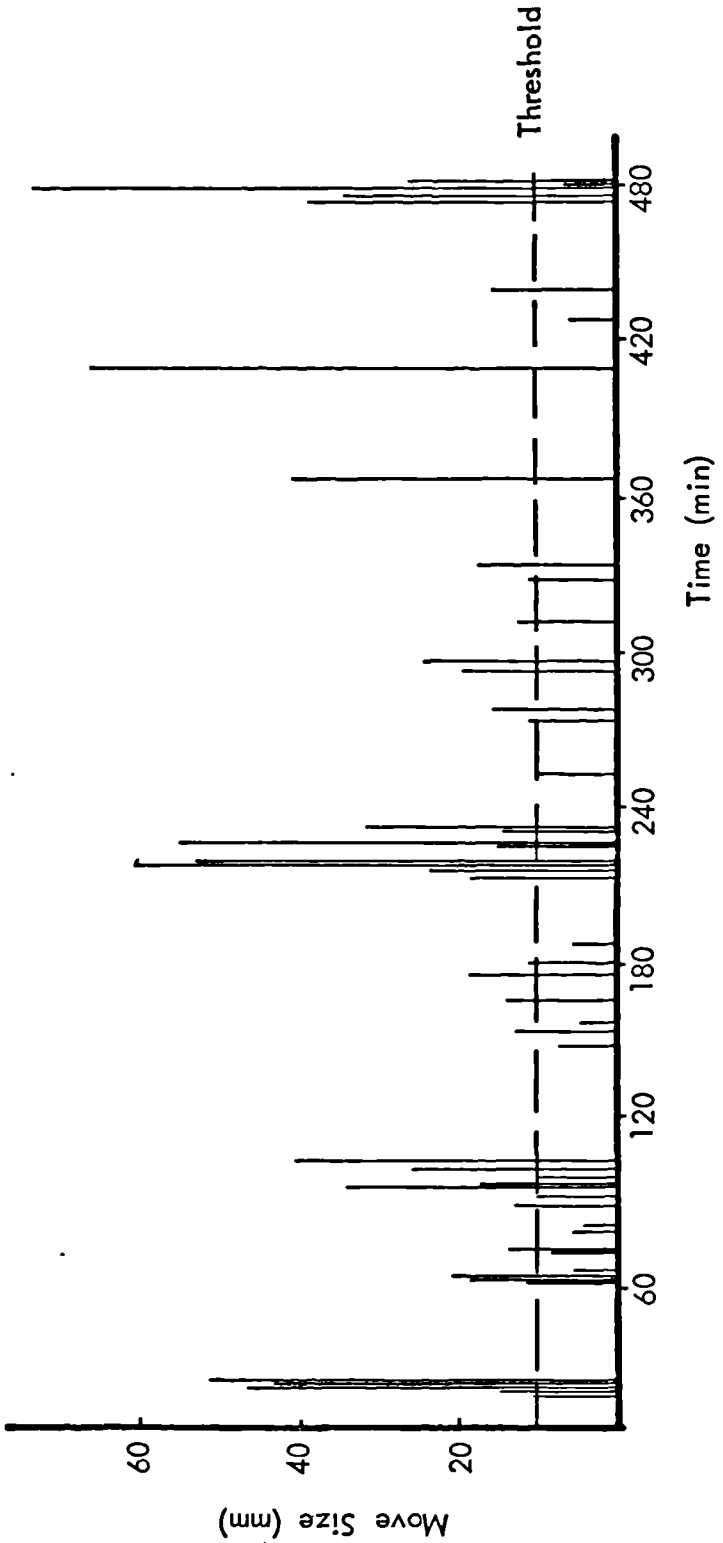


Figure 6.5 Example of the output of the monitoring system, after data refinement.

The editing process required visual assessment, firstly in identification of a move and secondly in placement of the cursors. This suggested an element of subjectivity, of particular importance in the selection and measurement of small moves. However, movements of this magnitude are unlikely to be relevant to the relief of tissue pressure and were discarded in later stages of the analysis.

The edited data, stored on the P.D.P. 12, was transferred to punched tape and was input to an ICL 1904 S digital computer. This information was used to calculate a magnitude and time of occurrence for each of the moves occurring during the night (figure 6.4 (C)).The data was described in graphical form, with plots of move size (figure 6.5); number of moves in 30 minute intervals; moves larger than a threshold; and the number of moves in 30 minute intervals, larger than a threshold, versus time. These graphs provided a rapid evaluation of a night's recording indicating the number of moves and the size of moves.

6.6.2 Data refinement

The first hour of the sleep record was discarded to allow time for the patient to fall asleep and was a period in the data demonstrating wide variability from night to night. Similarly the final hour was discarded to avoid artefacts arising from the patient waking up. In this way, assuming that the tests began at a regular time, the length of the sleep record was standardised to six hours. The total number of movements within this period were calculated. To permit analysis of the data with regard to move size a threshold (figure 6.5) was selected, related to changes in interface pressure (vide 4.6). The number of moves larger and smaller than the threshold were calculated. The record was then divided into 30 minute intervals and the number of moves occurring in each interval was calculated.

Movements occurring within one minute of each other were assumed to be a movement complex and the average move size and average time of occurrence of each complex was calculated.

On some occasions information in the patient data booklet indicated that the patient had suffered a disturbed night, usually as a result of her

clinical condition or the effect of external influences. The data for these nights were not included in the generalised analysis.

CHAPTER 7

RESULTS AND DISCUSSION

7.1 INTRODUCTION

Studies to establish the relative importance of specific aetiological factors are perhaps most easily accomplished by analysis of patient records, or even by undertaking a pressure sore incidence survey. The duration of such a study can be calculated to give "order of magnitude" estimates using data such as that provided in the prevalence survey of Barbenel et al (1977). An estimate for the duration of a survey to determine the relative importance of, for example, incontinence in a particular group of patients vulnerable to pressure sores, is given below:

i Assume that the percentage of patients that are incontinent is similar to the mean value in Barbenel et al (1977) = 38%

ii Assume that the percentage of incontinent patients that have already, or will develop a sore during their stay in hospital is similar to prevalence values in Barbenel et al (1977) = 17%

iii Assume that the number of admissions per week = N

For a statistically valid result a minimum of 5 incontinent patients with sores would have to be recorded, if the χ^2 test is to be used.

Number of sores developed in incontinent patients in the group being studied $= N \times \frac{38}{100} \times \frac{17}{100}$.

Thus, the number of weeks for 5 sores to be observed

$$= 5 \; / \; N \times \frac{38}{100} \times \frac{17}{100}$$

For a typical geriatric assessment ward N might be ≈ 10

Thus, the duration of the survey $\approx$ 8 weeks.

In practice the duration of the survey may be longer or shorter than this dependent upon variations in patient admission rates and the validity of assumptions i and ii, for the group being studied. Although it is clear that useful clinical data can be collected to describe the pressure sore incidence of a ward in a relatively short time, this approach has limitations. Firstly, the results are confined to sampling the patients as a group and hence only generalisations can be made for subgroups that are apparently at risk. No information is obtained for the individual, although if he exhibits the characteristic of the subgroup, it might be assumed that his vulnerability to

pressure sores is similar to that of the subgroup. Secondly, it is necessary for patients to actually develop sores before changes in the pattern of incidence can be detected.

Measurements of patient mobility would appear to overcome some of the limitations of maintaining simple incidence records for a ward. By obtaining a measure of the baseline mobility characteristics of a general group of geriatric patients, variations in individuals can be assessed with relative ease. However, an assessment of the significance of such variations in relation to the potential the patient has for developing sores, requires a measure of the variance in the general population and comparisons of sub-groups of patients known to be "at risk" within the general group. The objectives of the analysis of results for this study were therefore directed towards establishing these characteristics of mobility in an elderly population of patients in a geriatric assessment unit. In summary the following characteristics were sought.

- Parameters suitable for characterising the mobility records (vide 7.4)
- Variation in mobility during sleep, on each night, between patients (vide 7.5)
- Night to night variation in mobility for each individual (vide 7.6)
- Variation in mobility related to factors associated with the treatment or clinical condition of the patient (vide 7.9)

7.2 GRAPHICAL REPRESENTATION OF THE INITIAL OUTPUT DATA

The output data following initial computer processing, provided for the first evaluation of a patient's overnight mobility. Such an appraisal was necessarily unrefined but served to indicate the general pattern of mobility, number of moves, size of moves etc.

7.2.1 General observations based on data collected for individual patients

There is always a risk when the collection of clinical data is highly organised that important details which help to provide a coherent summary of the individual patient's condition may be lost. Such details are perhaps of less importance in a research study than they are for working clinical records. In order to clarify the background to the discrete data collected for this study, two case sketches are provided.

Patient 24: This elderly undernourished woman was admitted to hospital suffering from diverticulitis. She had a small pressure sore (grade 4) in the sacral region, which was treated with topical agents (aserbine solution, barrier cream etc.), a ripple mattress and regular turning. On arrival in the ward her susceptibility to pressure sores was determined using the Norton score (vide 6.2.3). Her record is reproduced below, indicating a total score of 13.

Physical Condition	Mental Condition	Activity	Mobility	Incontinent	Total
3	4	1	1	4	13

By the sixth day after admission her Norton score had risen to 17,

Physical Condition	Mental Condition	Activity	Mobility	Incontinent	Total
3	4	3	3	4	17

and the condition of her sore had improved. Upon examination of the individual factors associated with the Norton score, it was evident that her improvement was related to an increase in mobility - as assessed by the nursing staff. Figures 7.1 and 7.2 describe her mobility at the beginning and end of her first week's stay in hospital. From these it is apparent that her mobility had increased dramatically. This difference was further illustrated when comparing the number of large moves for the two nights (figures 7.3 and 7.4). Thus, for this patient the changing clinical condition of a pressure sore appeared to be associated with a corresponding change in mobility.

Patient 15: An 87 year old lady, admitted with chronic bronchitis and a history of cardiovascular complaints. Her general condition was stable, she had no pressure sores and a high Norton score (18), suggesting a low susceptibility to pressure sores. However, despite this the patient was nursed on a ripple mattress and was being turned at four hourly intervals. This stable clinical condition was reflected in her overnight mobility. Figures 7.5 a, b and c, describe the number of movements, in 30 minute intervals, made by the patient during each of the first three nights after admission. There was no marked change between them. However, by examining the graphs of move size for each of the nights (figures 7.6 a, b and c) it was apparent that the size of movements increased. The percentage number of moves greater than 100 units, for the three nights, were calculated to be 16%, 35% and 48% respectively. This result was not attributable to any obvious clinical characteristic and may be related to a change in environment, followed by a period of adjustment.

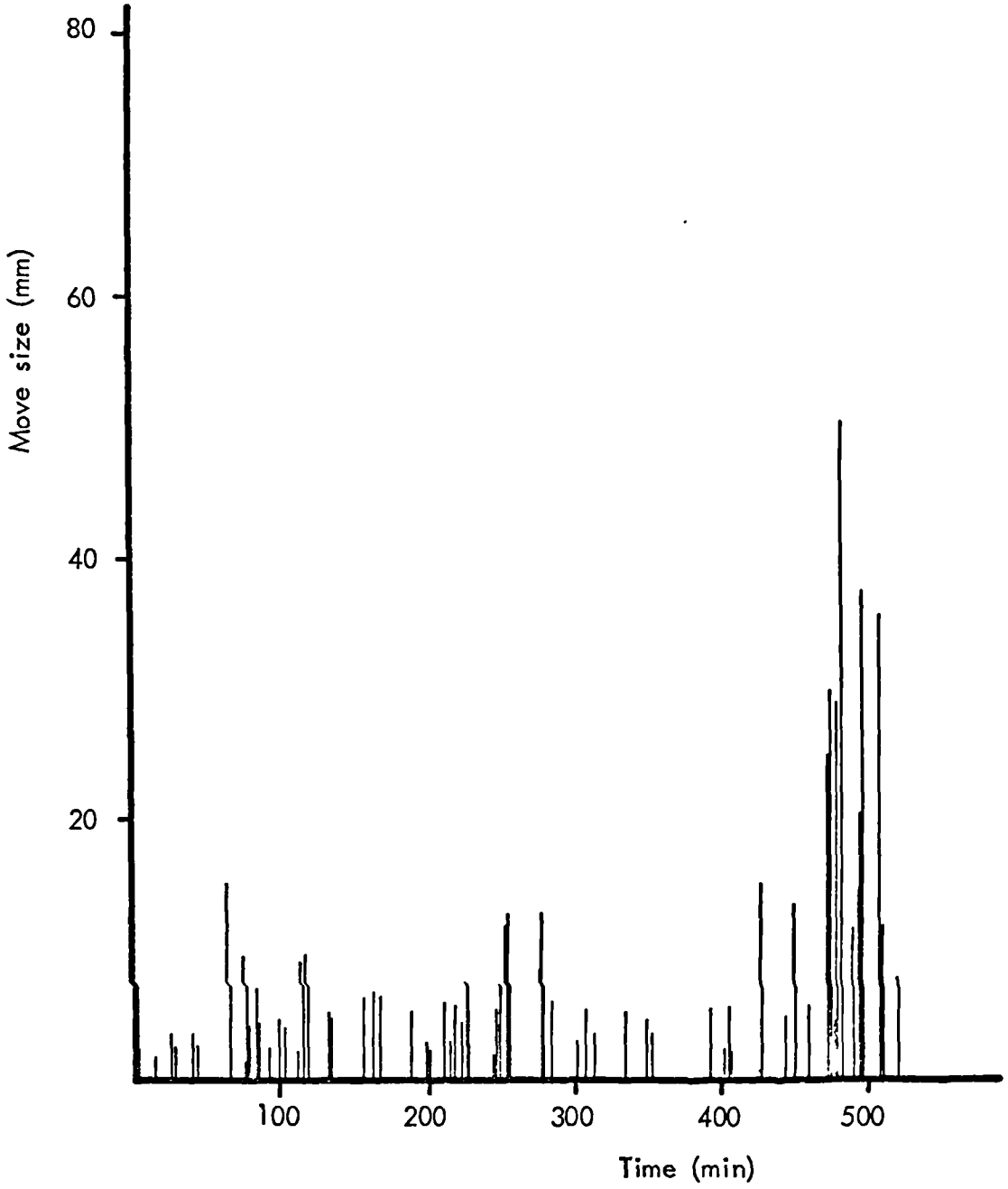


Figure 7.1 Distribution of moves throughout the night.
Patient 24, 3rd night.

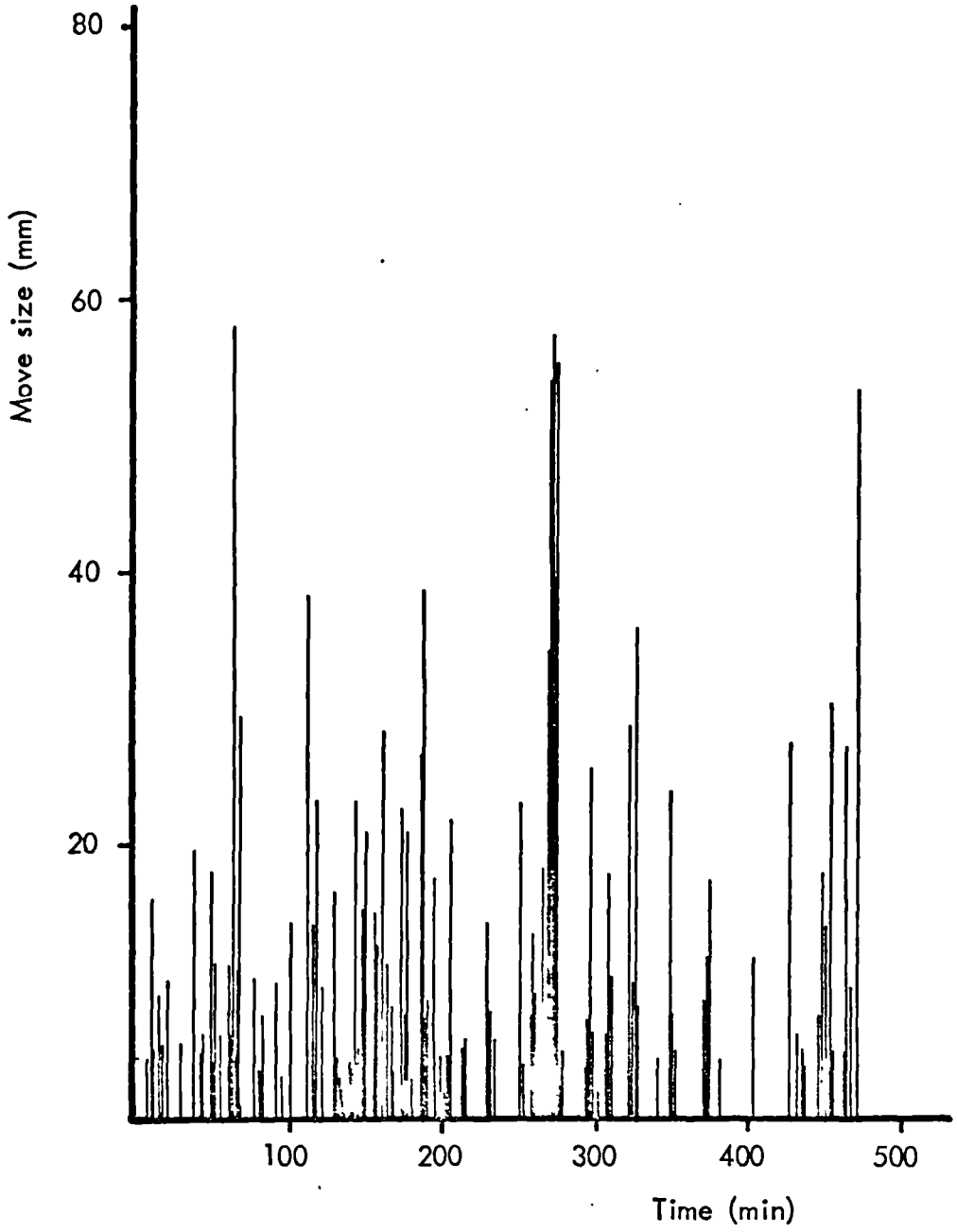


Figure 7.2 Distribution of moves throughout the night.
Patient 24, 6th night.

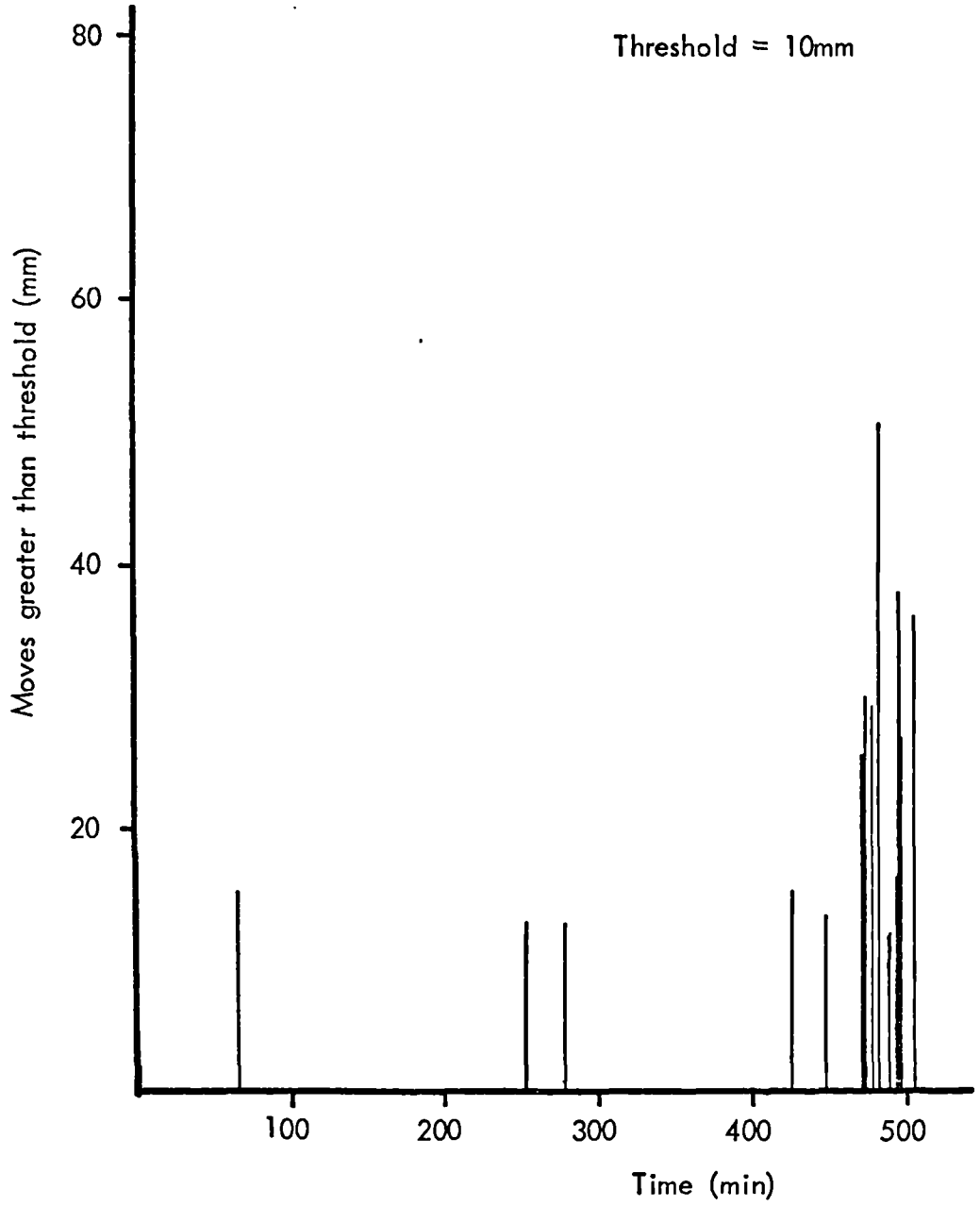


Figure 7.3 Distribution of moves larger than a threshold throughout the night. Patient 24, 3rd night.

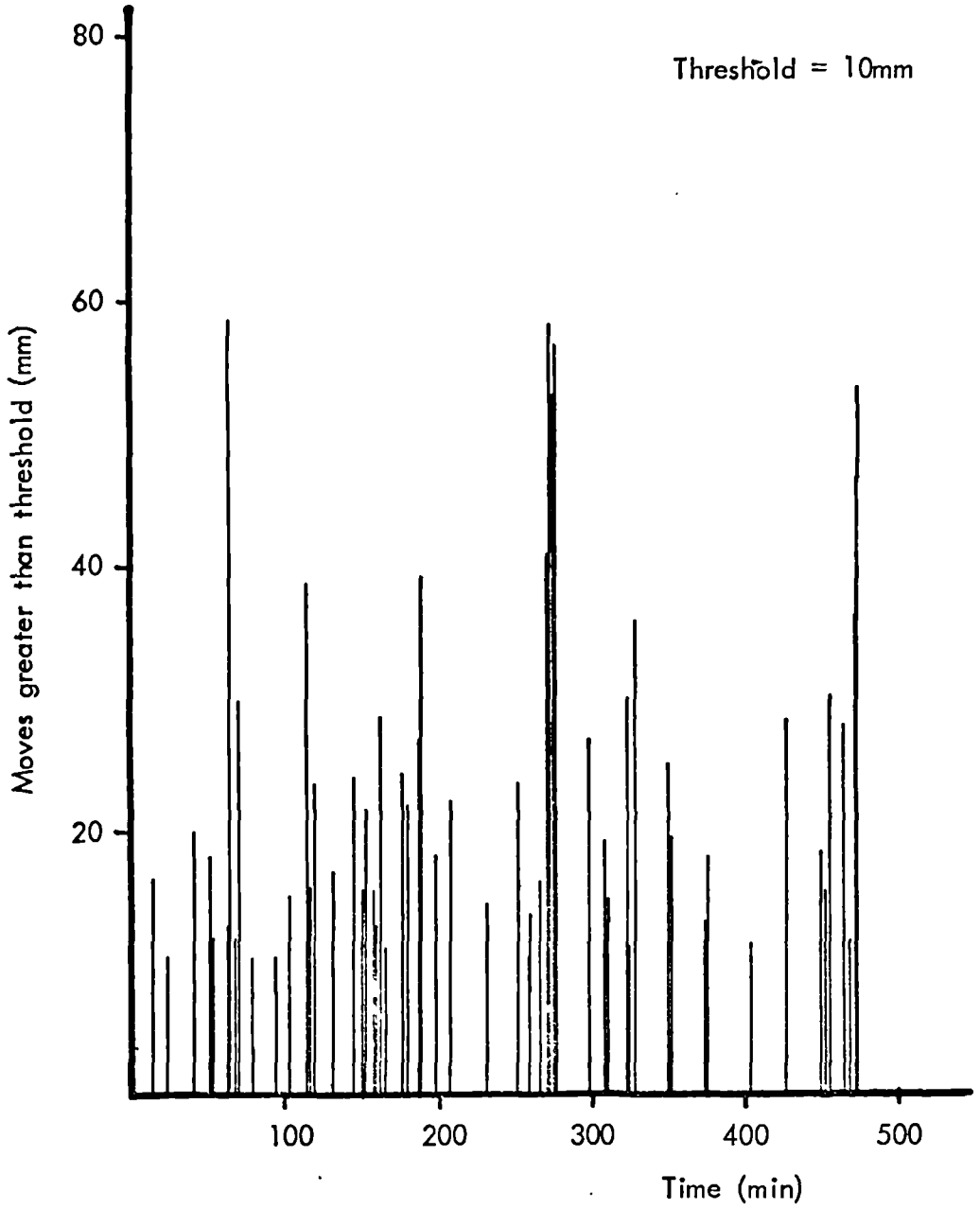


Figure 7.4 Distribution of moves larger than a threshold throughout the night. Patient 24, 6th night.

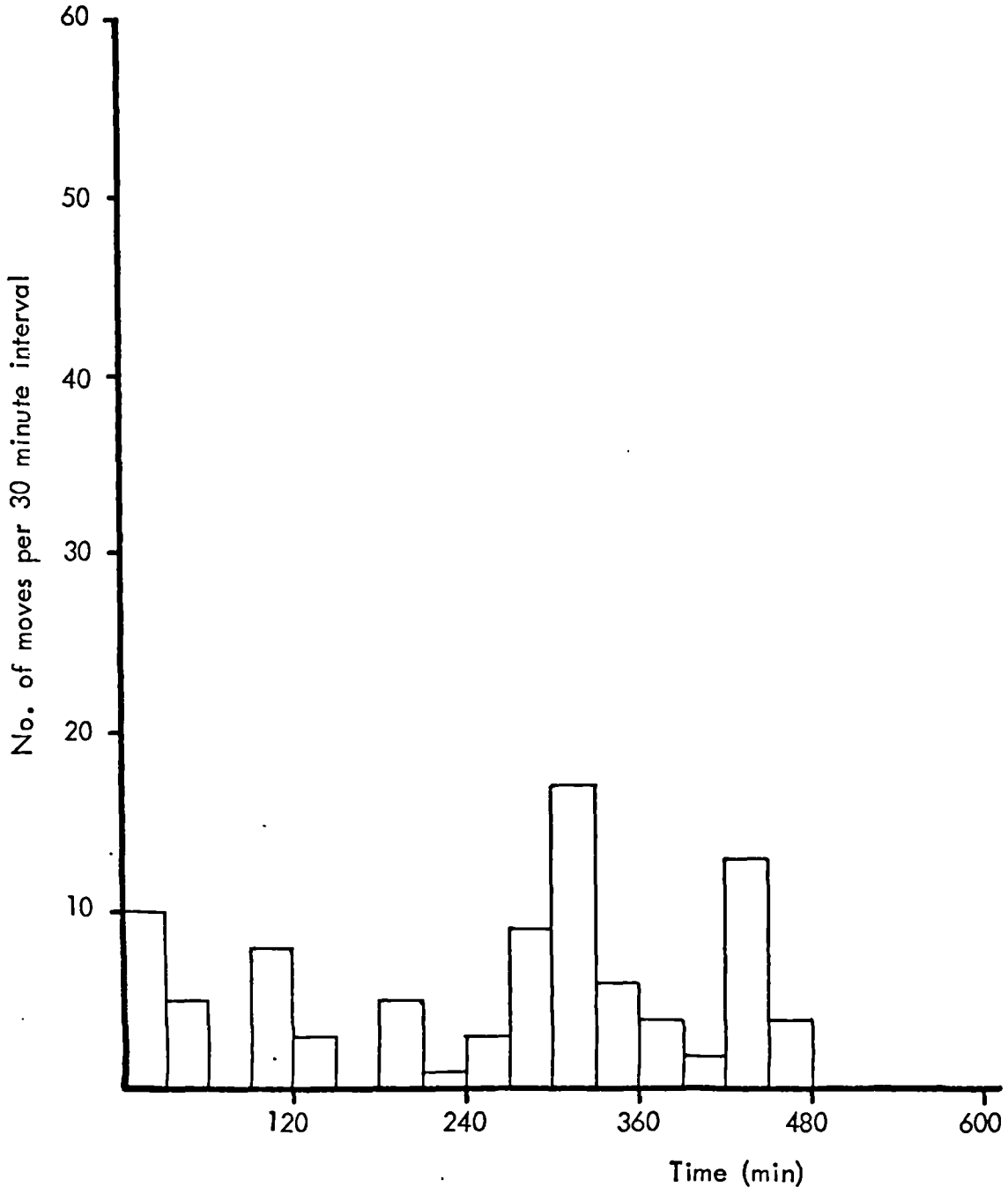


Figure 7.5a Number of moves in 30 minute sample intervals. Patient 15, 1st night.

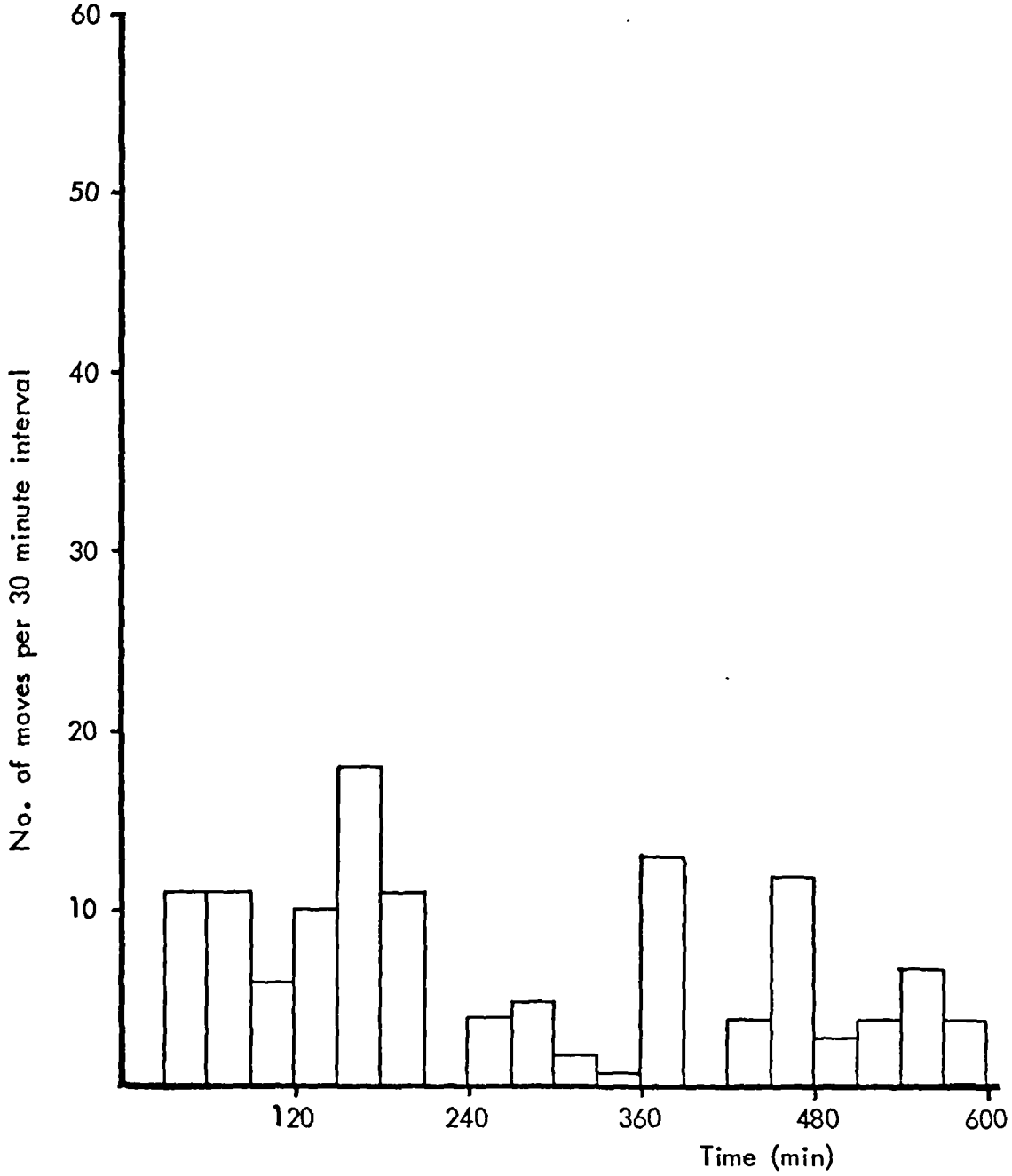


Figure 7.5b Number of moves in 30 minute sample intervals. Patient 15, 2nd night.

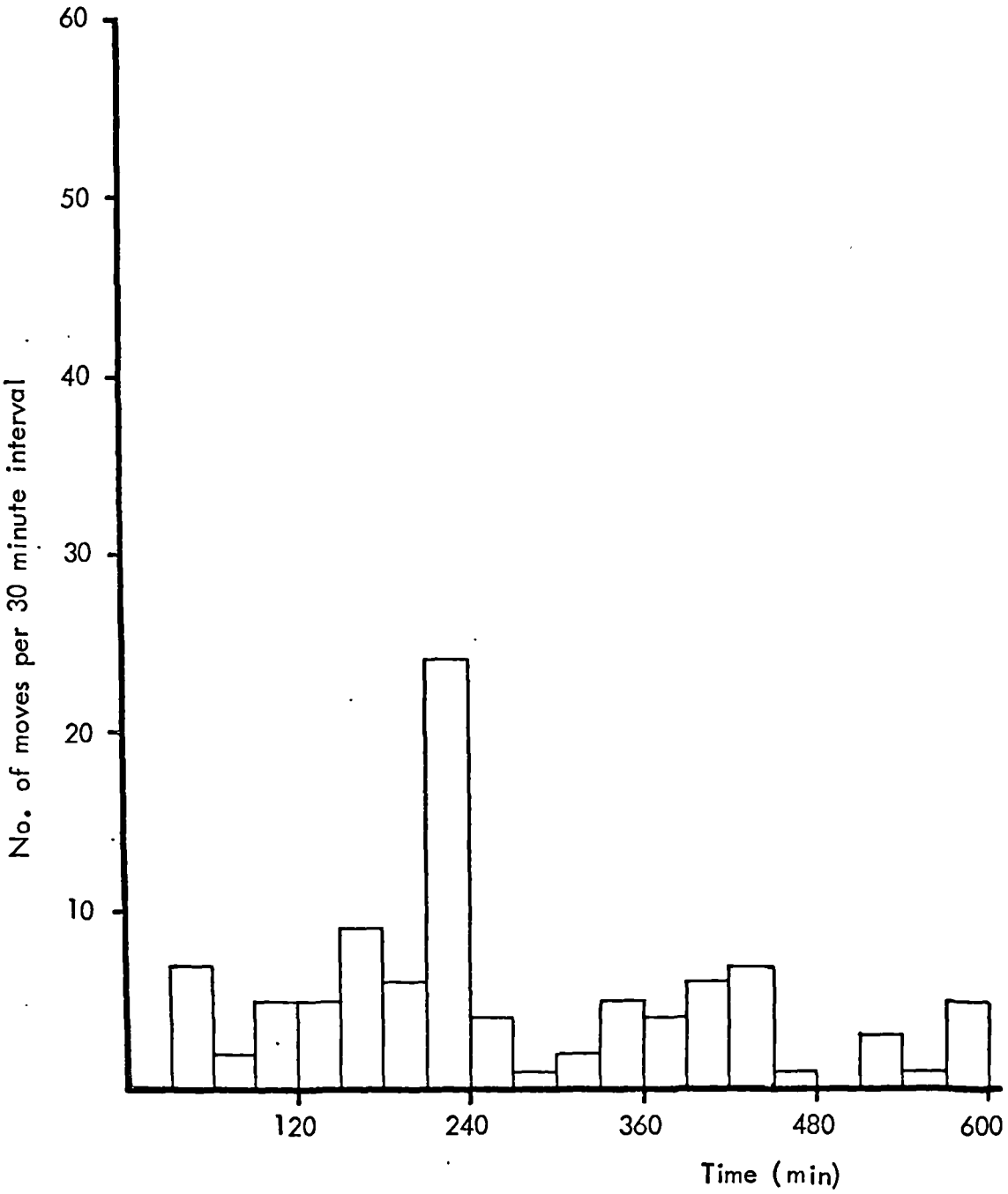


Figure 7.5c Number of moves in 30 minute sample intervals. Patient 15, 3rd night.

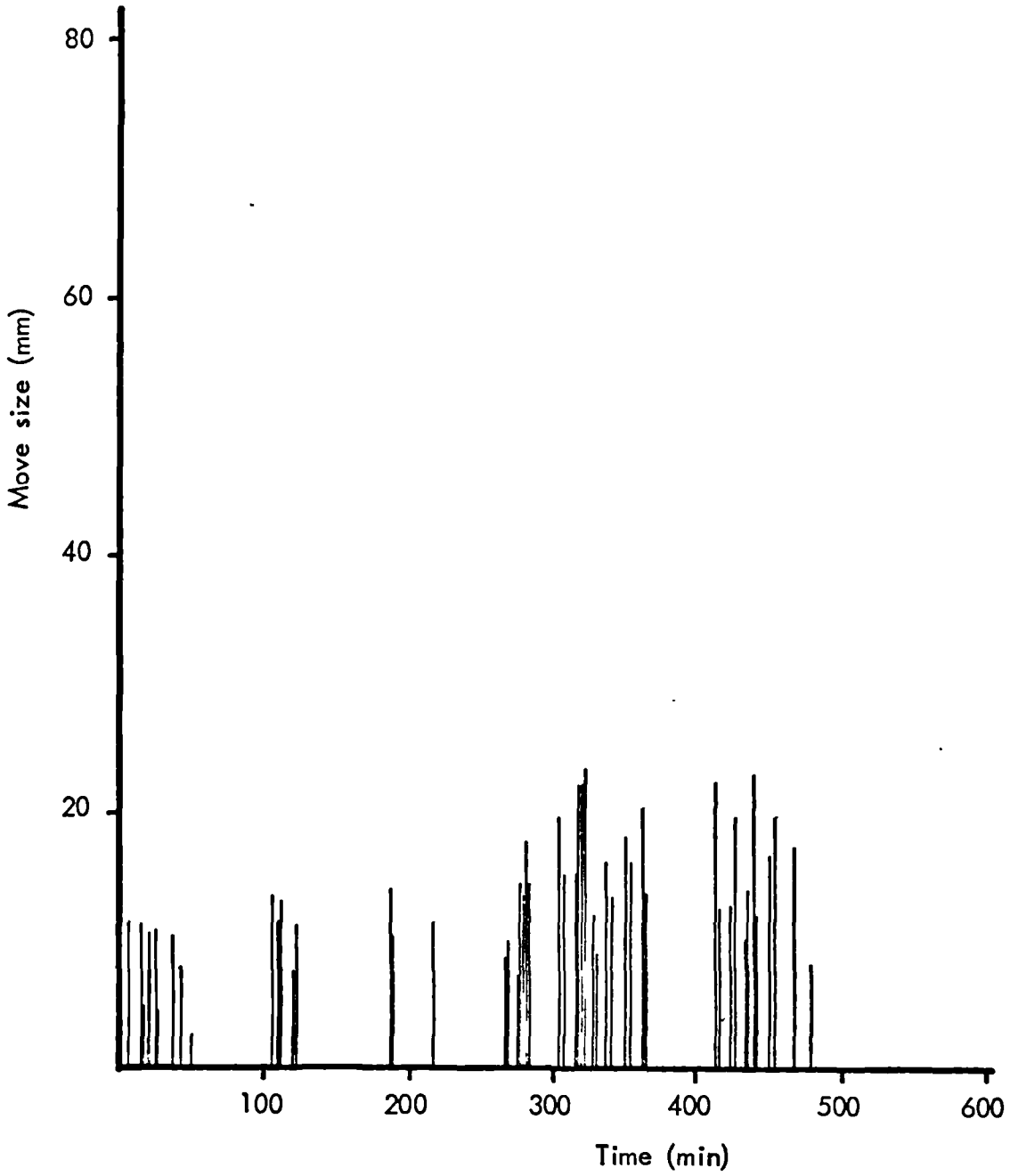


Figure 7.6a Distribution of moves throughout the night.
Patient 15, 1st night.

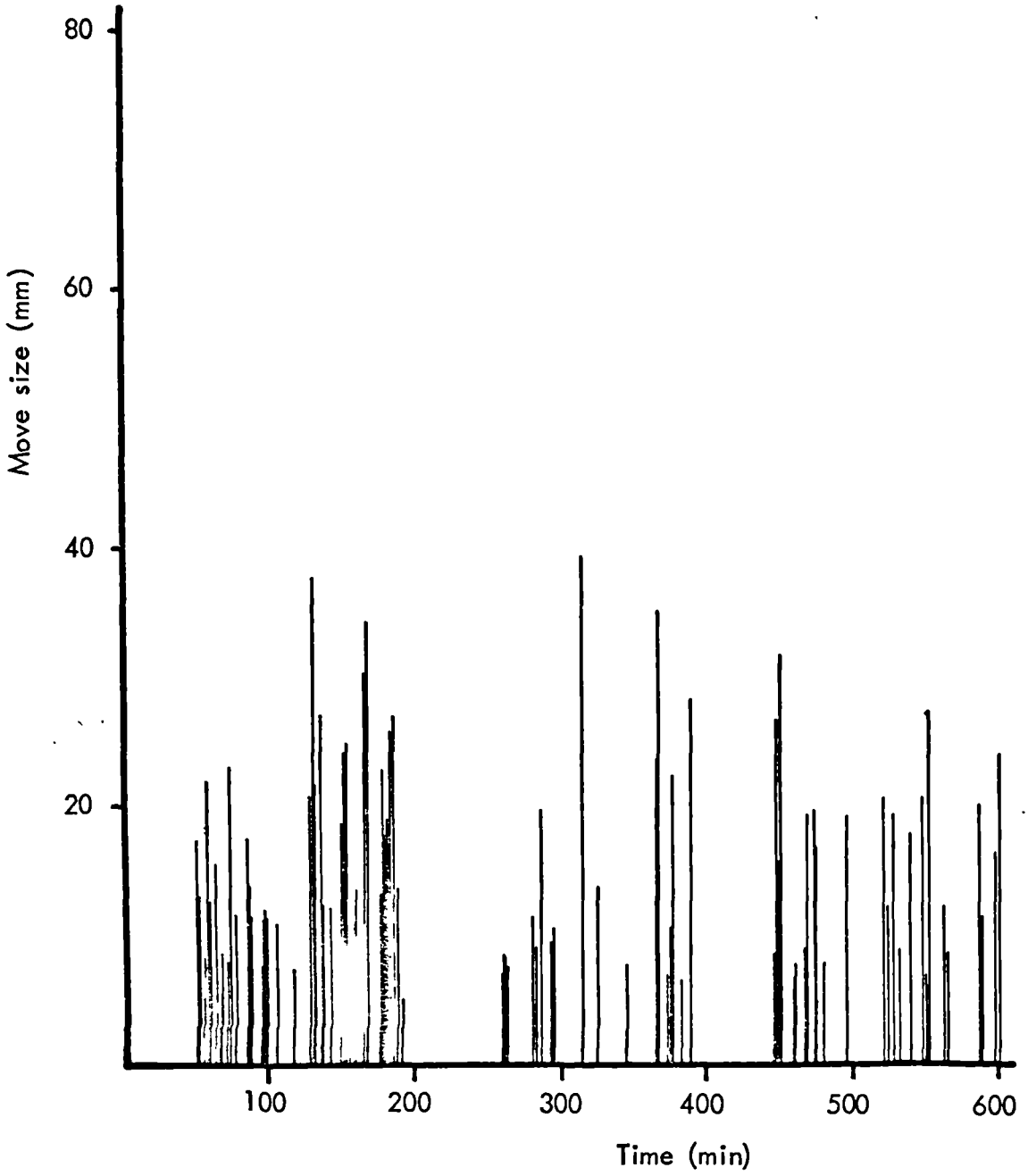


Figure 7.6b Distribution of moves throughout the night.
Patient 15, 2nd night.

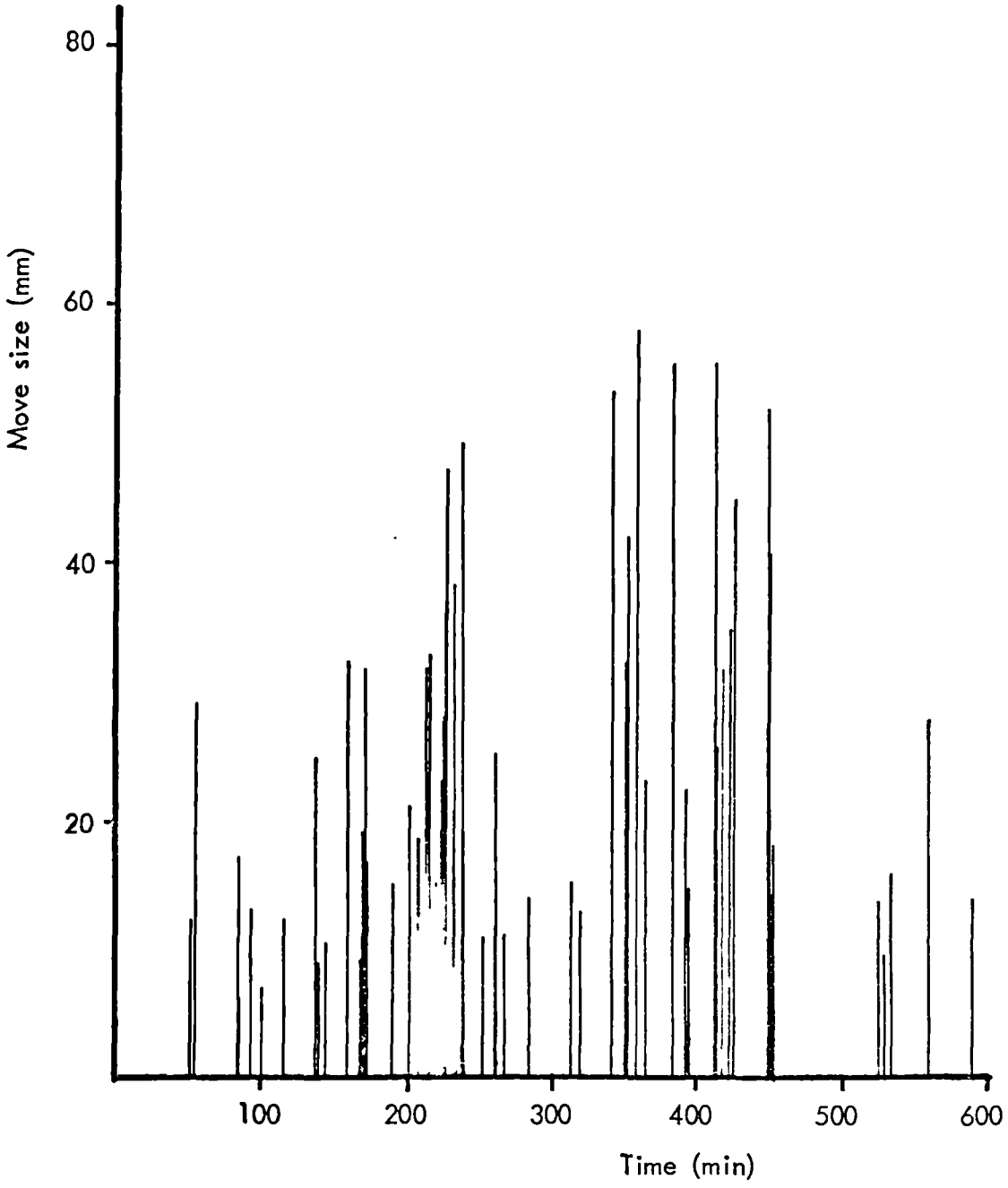
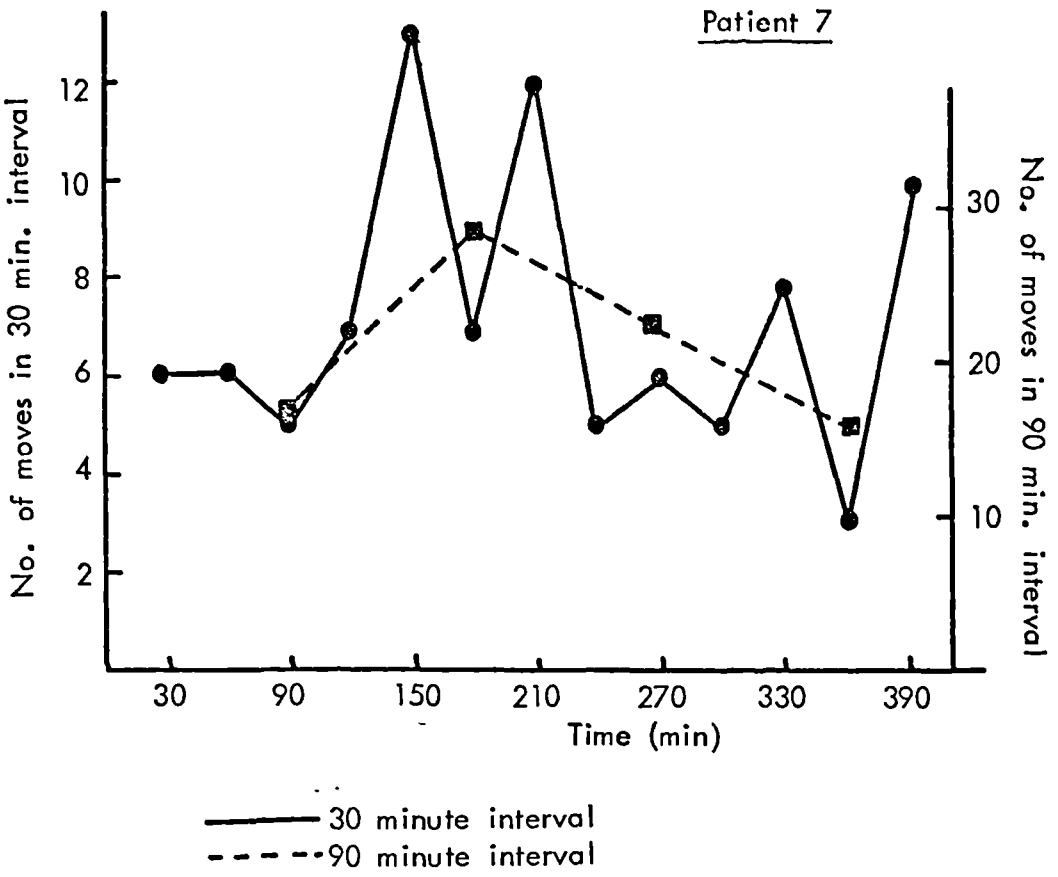


Figure 7.6c Distribution of moves throughout the night.
Patient 15, 3rd night.

a)



b)

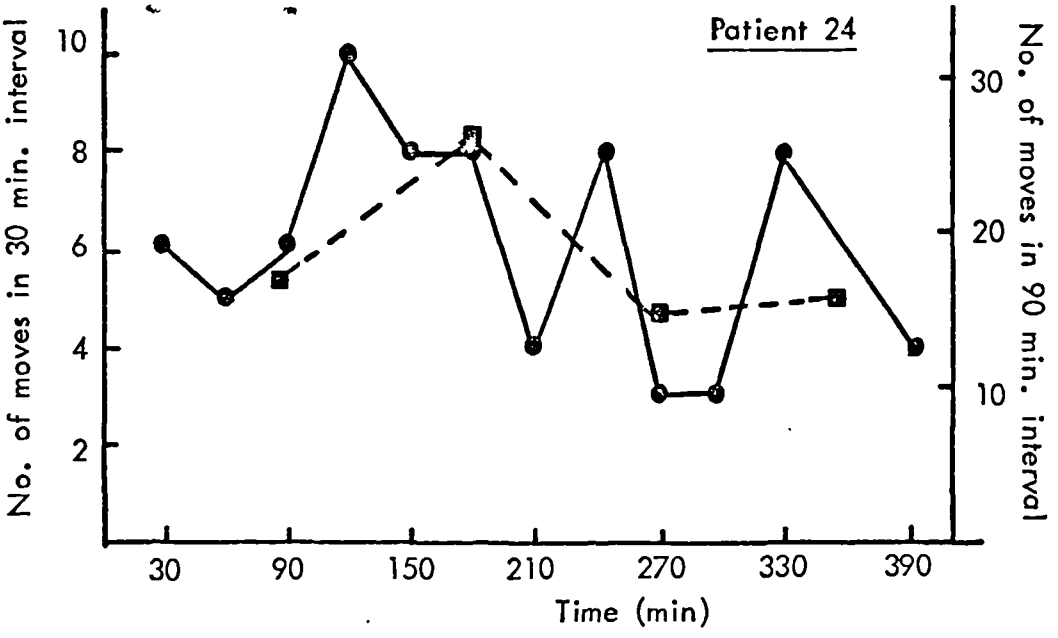


Figure 7.7a,b

Movement frequency - 30 and 90 minute intervals.
a) Patient 7, 3rd night suggesting a 60 minute periodicity
b) Patient 24, 1st night exhibiting a lack of periodicity.

7.3 FREQUENCY DISTRIBUTION OF MOVEMENTS DURING SLEEP

7.3.1 Results

Each of the sleep records was divided up into 30 and 90 minute intervals and the number of movements occurring in each interval was recorded and displayed in graphical form. Some of the recordings exhibited periodicity (figure 7.7a), although the majority did not (figures 7.7 b, c and d). The number of moves occurring in each 90 minute interval also showed no regular characteristics (figures 7.7 a, b, c and d).

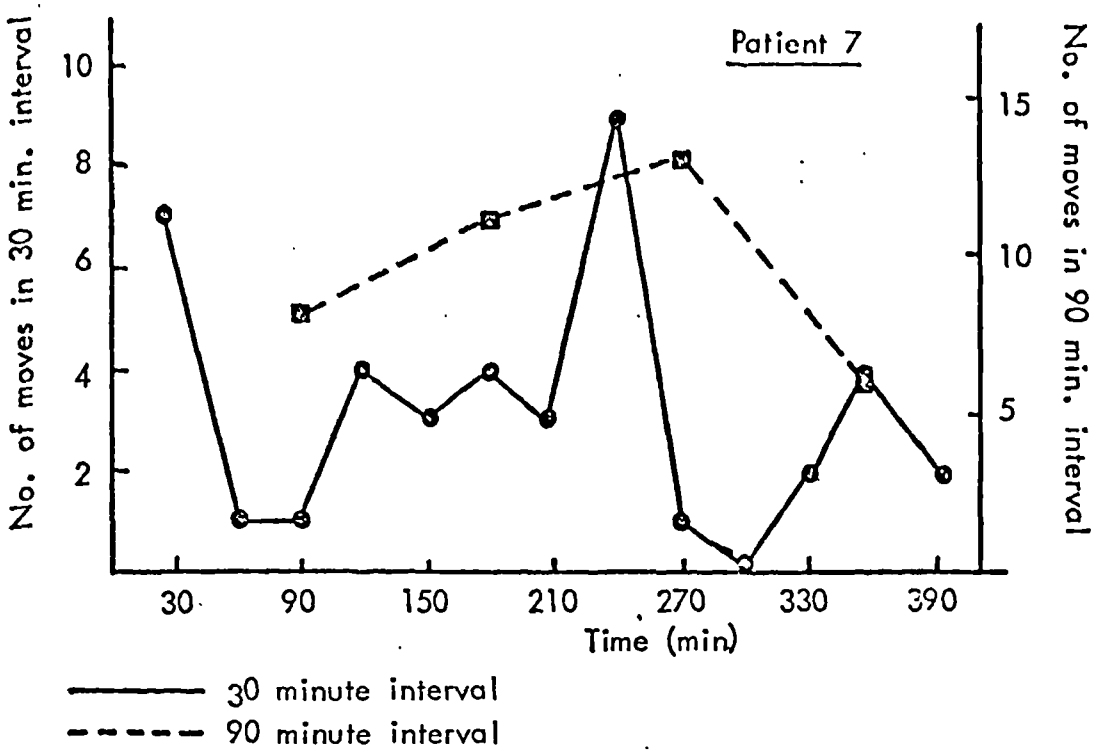
The frequency distribution of moves performed in different halves of the night for each of the recordings, was also considered. The number of moves occurring in each half of the night was approximately the same, 51% of the recordings exhibited a greater mobility in the first half of the night, 47% in the second half, with 2% having equal frequencies.

7.3.2 Discussion

The lack of periodicity and no consistent relationship in the frequency of movements made during the night is in marked contrast to Bardsley (1977) who, in a study of young normal adults, discovered that all of his subjects exhibited periodicity in their sleep patterns. The movement frequency of his subjects tended to increase during the course of the night and there was a marked reduction in the number of moves occurring in the first half of the night, in relation to the second. Such a discrepancy may be related to one or the combination of two causes.

Firstly, the hospital environment is almost certainly disruptive to normal sleep patterns. Typically, the ward in which the study was conducted, contained approximately 20 - 25 patients. A number of these would have undergone an impairment and deterioration in their physical and mental condition and were prone to vocal outbursts such as shouting for attention, weeping, or simply continual moaning. In addition, the medical staff were moving about the ward. As a result, the background noise level was not only high, but was subject to fluctuation. This is in direct contrast to Bardsley (1977) who ensured that his subjects slept with the minimum background

c)



d)

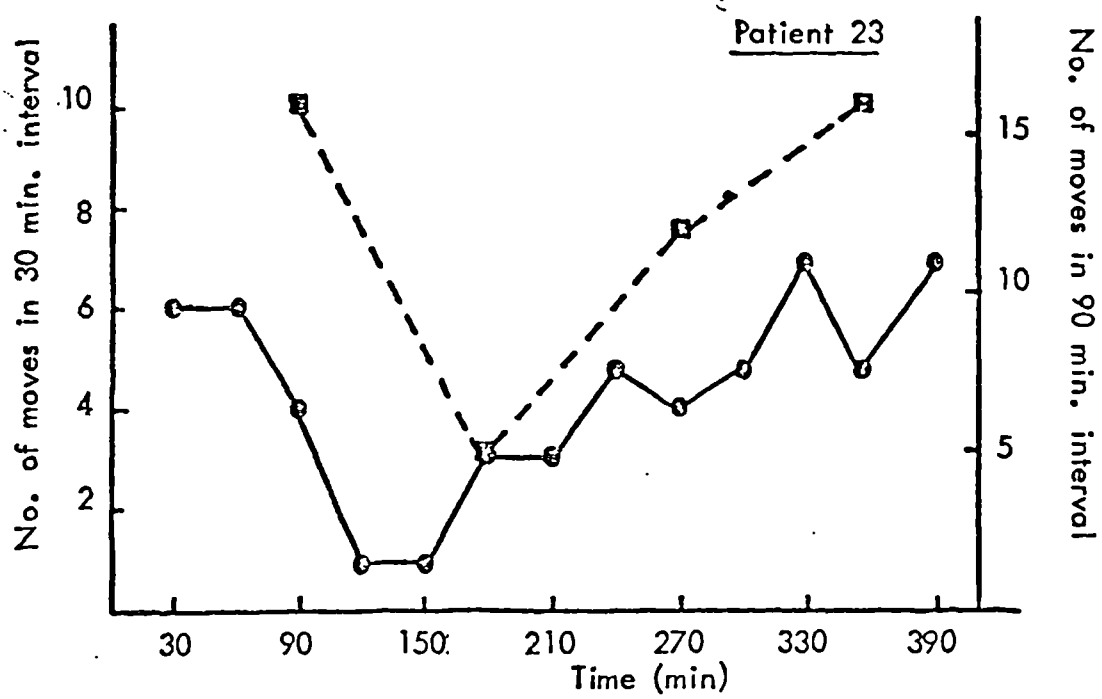


Figure 7.7c,d Movement frequency - 30 and 90 minute intervals c) Patient 7, 2nd night, and d) Patient 23, 4th night. Exhibiting a lack of periodicity.

noise level and to prevent fluctuations of level, he even employed a white noise generator.

Secondly, the age group of Bardsley's population was under 30 and therefore characteristic movement patterns were established for a young age group only. No equivalent studies have been conducted on the healthy elderly patient and so there is insufficient evidence to attribute the loss of periodicity solely to a change in the environment. Increasing age has been shown to alter other characteristics during sleep (duration, distribution of depth of sleep etc.) (vide 3.3.3), and it is possible that in a similar manner, periodicity during sleep is related to age.

Before any firm conclusions can be drawn as to the reason for the contrast in frequency of movement between the young normals and the elderly sick, it is first necessary to investigate the elderly normal and the young sick populations in similar environments.

7.4 PARAMETERS FOR CHARACTERISING THE SLEEP RECORDS

After initial data processing, additional analysis was conducted (vide 6.6.2) to regulate the length of the sleep record, to pool those moves occurring close together (move complex) and to determine the value of a set of parameters, chosen to characterise the data. Selected parameters related to the total number of movements, made by a patient during the night and the time interval between them. In addition the magnitude of each move was compared to a threshold value (vide 4.6) discriminating "large" movements from "small" movements.

7.4.1 Number of moves

The most obvious parameter available to characterise mobility during sleep, and one of the easiest to assess, is the number of movements performed by the patient. This describes a measure of the "restlessness" of the patient, although it provides no information as to the distribution of movements during the night or of their magnitude. In this analysis, the total number of moves occurring during a sleeping period (six hours) was denoted as N_m and was calculated for every night's recording.

To incorporate a measure of size, each move was grouped according to its magnitude as "large" ($>T$) or "small" ($<T$) (vide 6.6.2, figure 6.5), where T was a threshold value, established in chapter 4, as being the size of centre of gravity shift, producing the greatest correspondence with simultaneously measured changes in interface pressure. The number of moves in each group was then recorded. This total was denoted as $N_m > T$ and $N_m < T$. Additionally the ratio of the number of "large" moves to "small" moves was calculated and denoted as $N_m > T / N_m < T$. Thus, parameters for characterising the mobility data, associated with the number of moves and their size were N_m, $N_m > T$, $N_m < T$ and $N_m > T / N_m < T$.

Normalisation of the data: On some occasions the sleep record was shorter than the six hours chosen as the duration for analysis (vide 6.6.2). This was due to a number of factors, including failure of the equipment, accidental unplugging of the mains supply, premature awakening of the patient

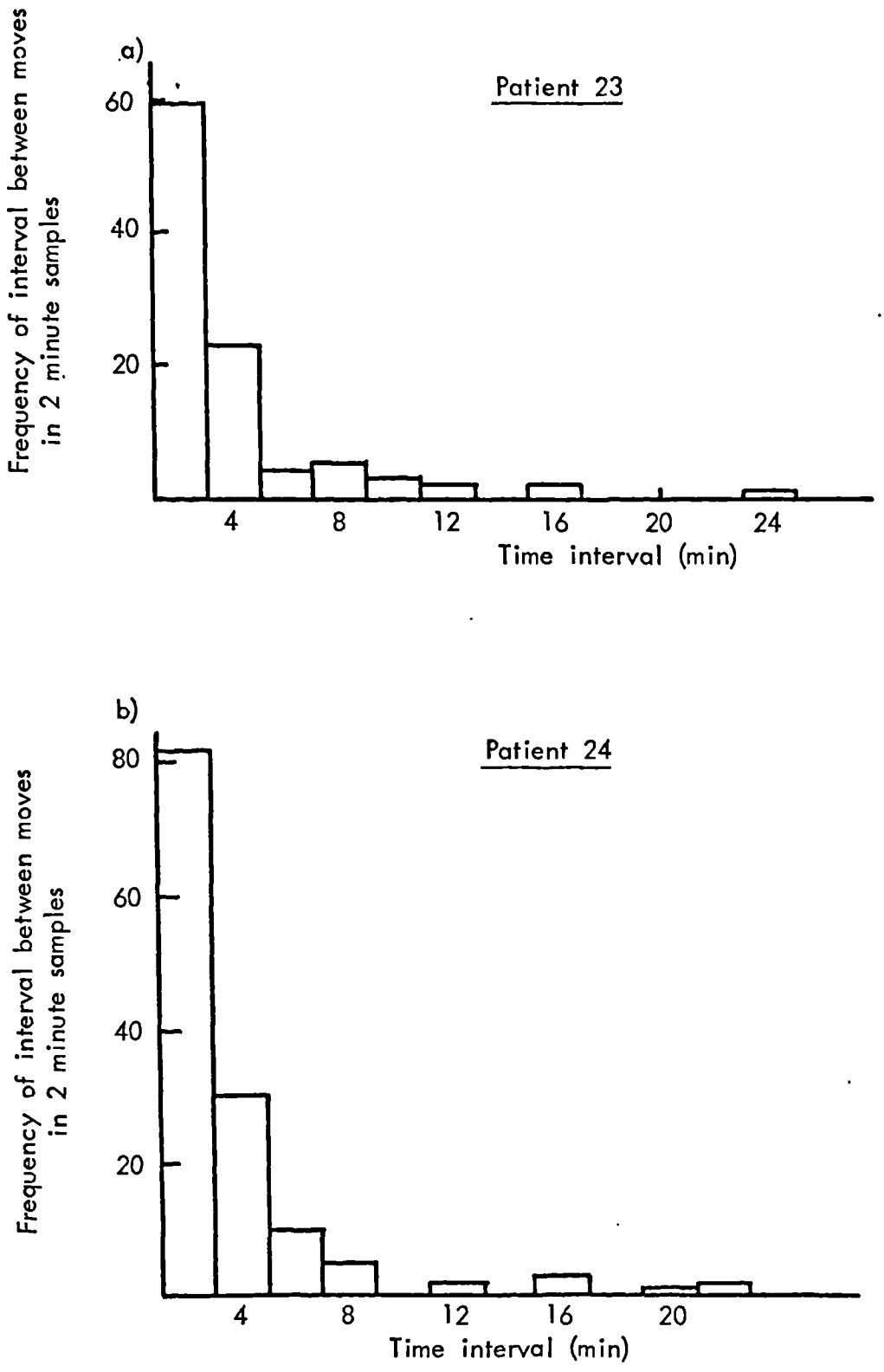


Figure 7.8a,b Distribution of interval between moves in 2 minute samples. a) Patient 23, 3rd night and b) Patient 24, 1st night

and in three cases the patient died during the monitoring session. The results of section 7.3, indicated that the frequency of movements throughout the night was approximately equal and thus, to account for the shortened records, a normalised version of the number of moves (n_m) was calculated,

$$n_m = \frac{N_m}{\text{length of sleep record}} \times K$$

where K = a scaling factor of 10^3, used to convert the the magnitude of the parameter to a convenient size.

In normalised form the characterisation parameters were denoted as n_m, $n_m > T$, $n_m < T$ and $N_m > T / N_m < T$.

When a normalised parameter (with scaling factor) is used, it is important to remember that it is a rate of movement and is not the actual number of movements per se.

7.4.2 Interval between moves

An alternative method of characterising the mobility data involved calculation of the time interval between successive movements. Such a parameter is particularly relevant in a study related to pressure sores where one of the primary causative factors is unrelieved pressure (vide 2.1.1). The distribution of time intervals between movements in two minute sample widths were described for each night's data. To prevent a loss of information relating to short duration moves, the data employed for the calculation of this distribution, was not subject to the grouping together of those moves separated by less than one minute (vide 6.6.2). Figures 7.8a and b are typical examples indicating a large number of movements, separated by less than two minutes, longer intervals occurring with a much reduced frequency.

To enable the mobility information to be characterised in relation to the interval between moves, it was first necessary to establish a parameter. The shape of the distributions, exhibiting no maxima or minima suggested no obvious threshold value(s). In addition, unlike move size (vide 7.4.1), there was no apparent associated clinical or physiological factor suggestive

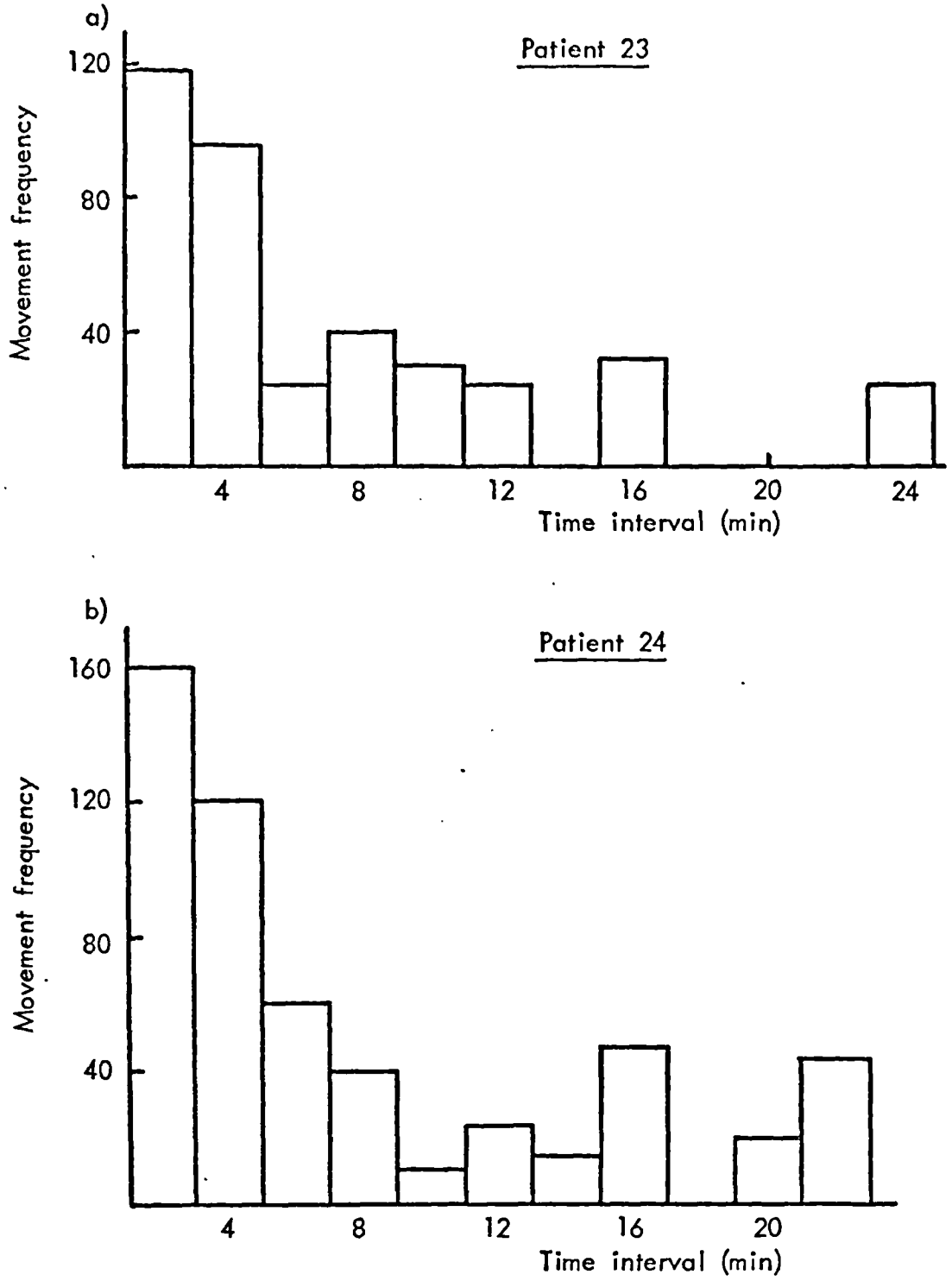


Figure 7.9a,b Distribution of integral transformation of interval between moves, in 2 minute samples. a) Patient 23, 3rd night and b) Patient 24, 1st night

of any threshold. Transforming the distribution by integration (figures 7.9a and b) also failed to provide any evidence of a "natural" division in the data.

The form of the distribution was found to correspond most closely with a lognormal distribution (figures 7.10a and b). Transforming the data using a three parameter lognormal function allows the utilisation of parametric statistics. In addition these parameters, related to the interval between moves, may also be suitable for characterising the mobility records. Bardsley (1977), who monitored the overnight mobility of normal subjects, observed a similar lognormal distribution, and he also utilised the parameters associated with the transformation to characterise the data. However, he established that, despite the accurate representation of the distribution, they were insensitive measures of movement characteristics. This was attributed to the effect of extreme values producing a "skewness" to the distribution. On the basis of this experience, no attempt was made to utilise a lognormal transformation of the distribution.

Thus it was decided to employ an alternative, less sophisticated technique. The data was grouped into three intervals, i_s, i_m and i_L where,

i_s (small) = intervals of less than 10 minutes

i_m (medium) = intervals of 10 minutes to 30 minutes

i_L (large) = intervals of longer than 30 minutes

The size of the intervals was arbitrary, in relation to the shape of the distribution, but were similar to that of Bardsley (1977), who had previously employed them to characterise his own mobility data, finding them to be sensitive to variations between subjects.

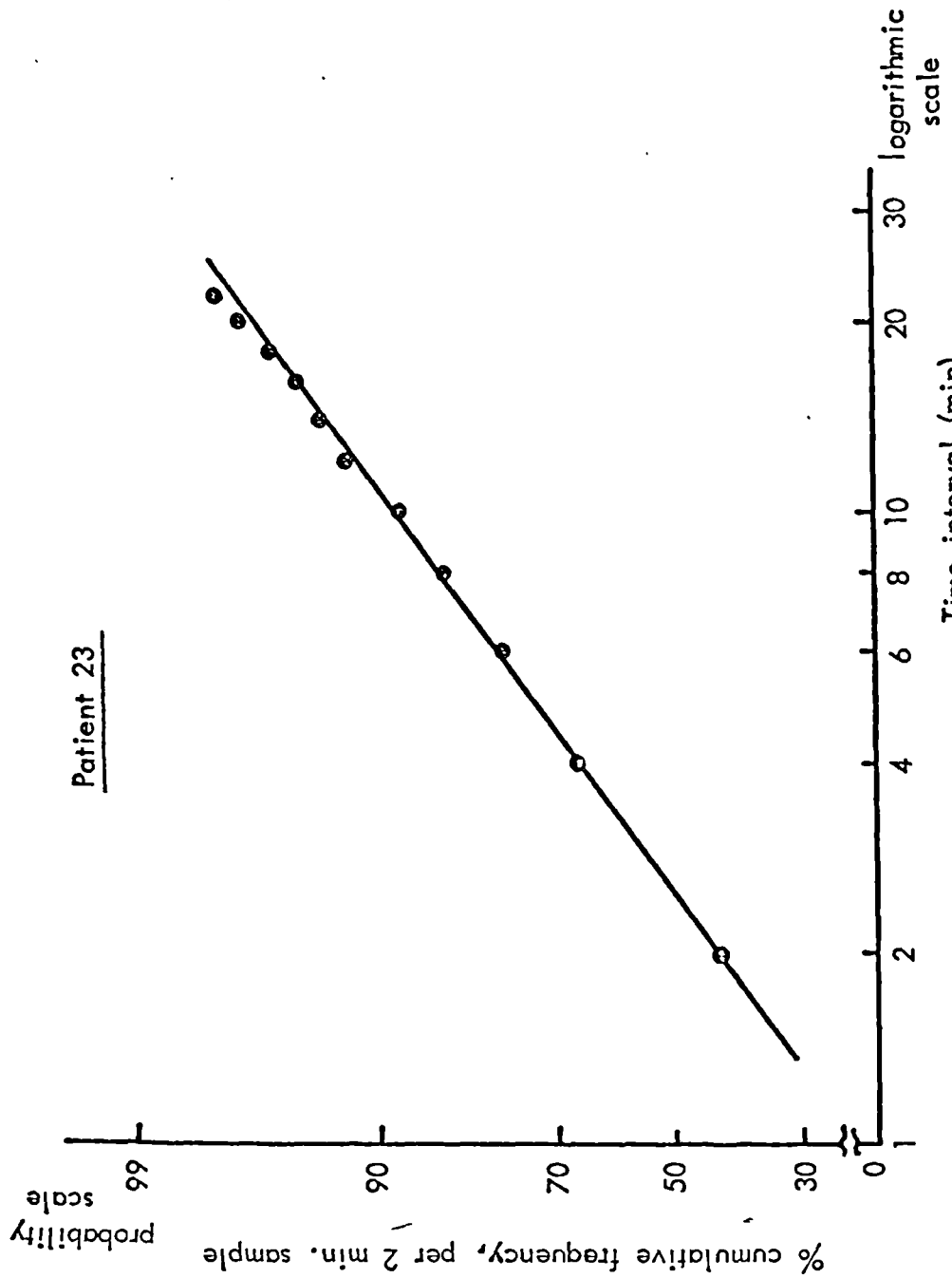


Figure 7.10a Logarithmic distribution of time interval between moves. (Patient 23)

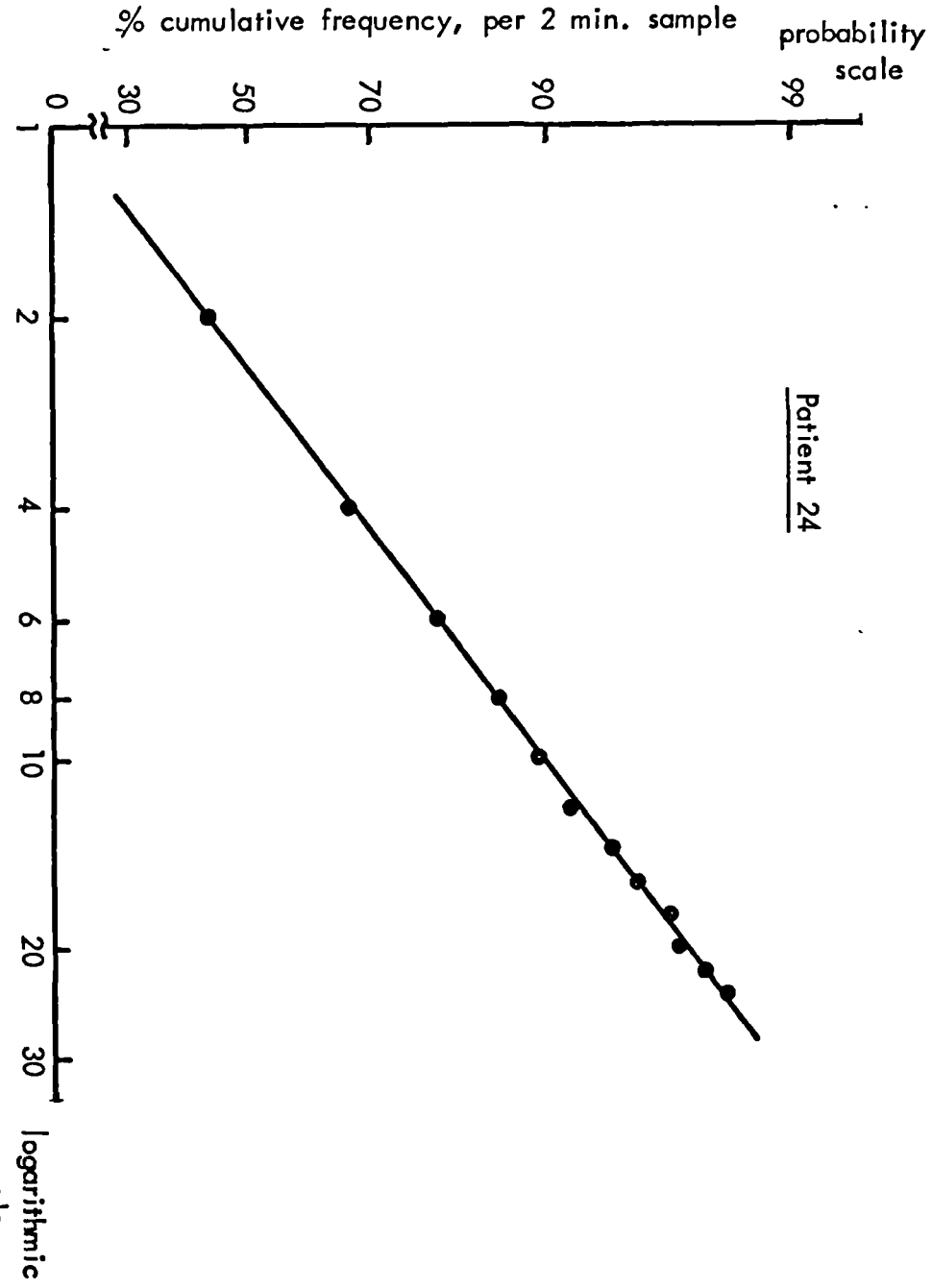


Figure 7.10b Logarithmic distribution of time interval between moves. (Patient 24)

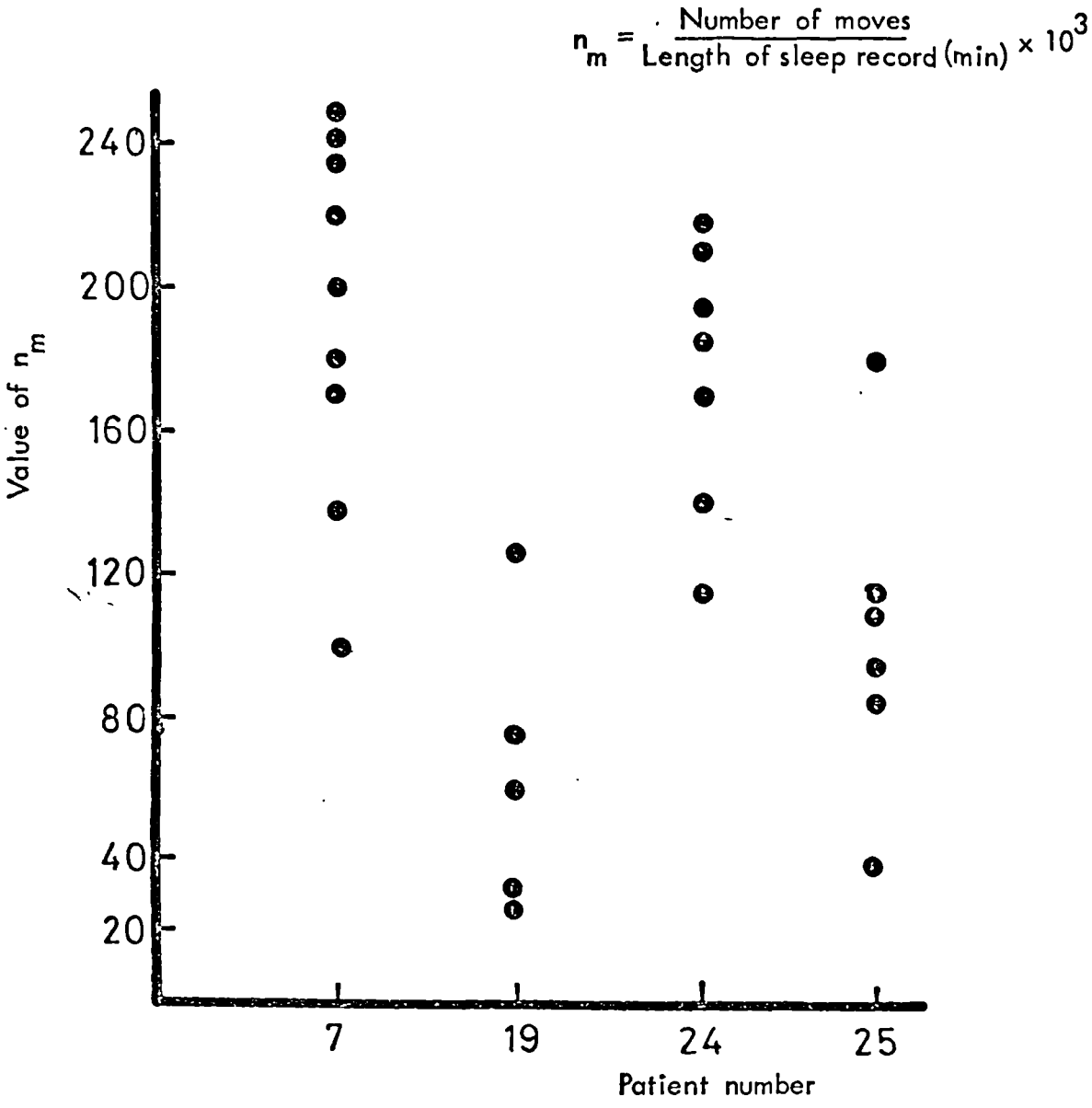


Figure 7.11a Number of moves, normalised, recorded for four of the patients on each night of the monitoring session.

7.5 CHARACTERISATION OF THE SLEEP RECORDS

Having established suitable characterisation parameters they were applied to the data. Values of N_m, $N_m > T$, $N_m < T$ and $^{N_m > T}/_{N_m < T}$ were calculated for each night's data, as were their normalised versions. In addition values of i_s, i_m, and i_L were similarly collected.

7.5.1 Number of moves

Figure 7.11a illustrates the value of n_m of four of the subjects monitored for five or more nights. Figures 7.11b, c and d are the values of $n_m > T$, $n_m < T$ and $^{N_m > T}/_{N_m < T}$ for the same subjects respectively. From these it can be seen that each patient exhibited a wide range in the number of movements during the night, over the period for which they were monitored. The mean range of the number of movements for $n_m = \frac{193}{74}$ (2.6), $n_m > T = \frac{126}{24}$ (5.3), $n_m < T = \frac{100}{36}$ (2.8) and $^{N_m > T}/_{N_m < T} = \frac{3.83}{0.15}$ (25.5). From these graphs it appeared that the results were approximately normally distributed and facilitated the use of parametric statistical tests.

One way analysis of variance was calculated to compare the variations in the values of n_m, $n_m > T$, $n_m < T$ and $^{N_m > T}/_{N_m < T}$, within and between patients. The variance ratio F test was used to determine whether the independent estimates of population variance differed significantly, or whether the samples may be considered to be drawn from the same population (Table 7.1). Characterisation parameters n_m, $n_m > T$ and $n_m < T$ demonstrated significant differences between subjects ($p<0.01$), while $^{N_m > T}/_{N_m < T}$ was significant at $p<0.025$.

The means and standard deviations of each of the movement parameters were calculated for every subject (Table 7.2). Student's t test was used to compare them. Of the 120 pairwise comparisons between the subjects 40 (33%) of n_m, 37 (30%) of $n_m > T$, 55 (44%) of $n_m < T$ and 25 (20%) of $^{N_m > T}/_{N_m < T}$ were significantly different (Tables 7.3a, b, c and d). The grand mean of the parameters relating to the total number of movements during the night and of those associated with move size, were calculated for both the normalised and un-normalised data (Table 7.4).

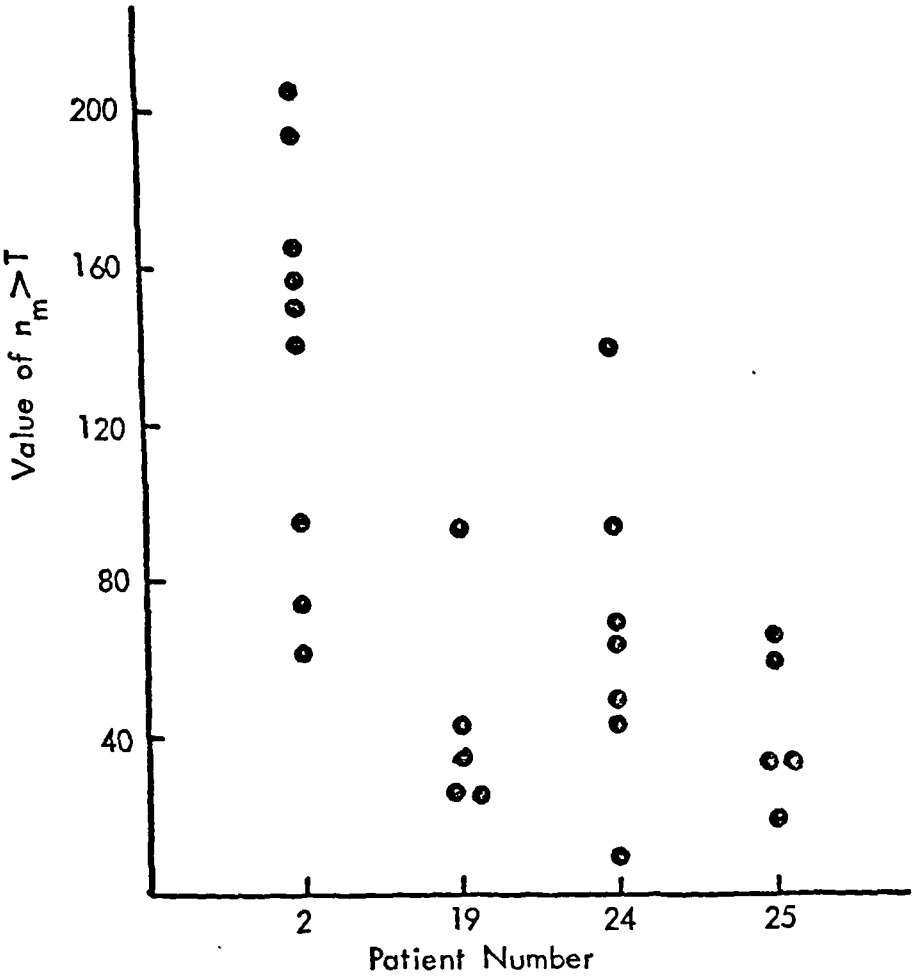


Figure 7.11b Number of moves larger than the threshold, normalised, ($n_m > T$) recorded for four of the patients on each night of the monitoring session.

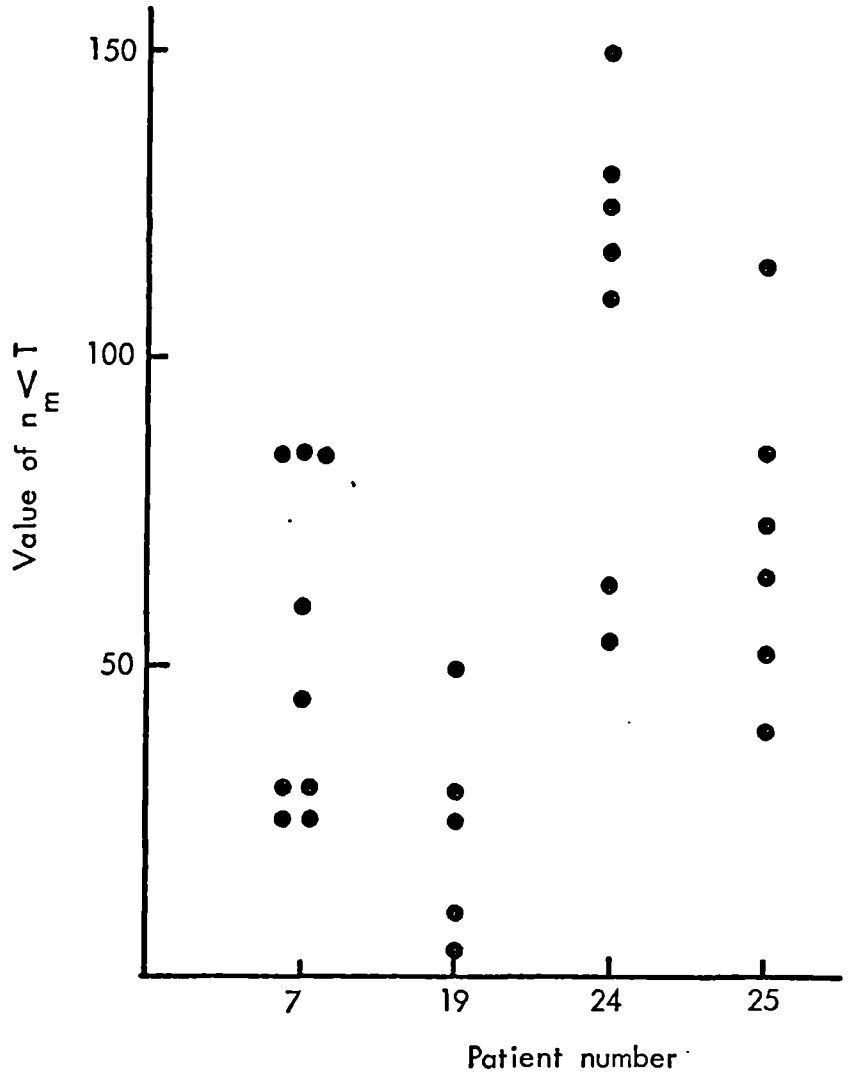


Figure 7.11c Number of moves less than the threshold, normalised, ($n_m < T$) recorded for four of the patients on each night of the monitoring session.

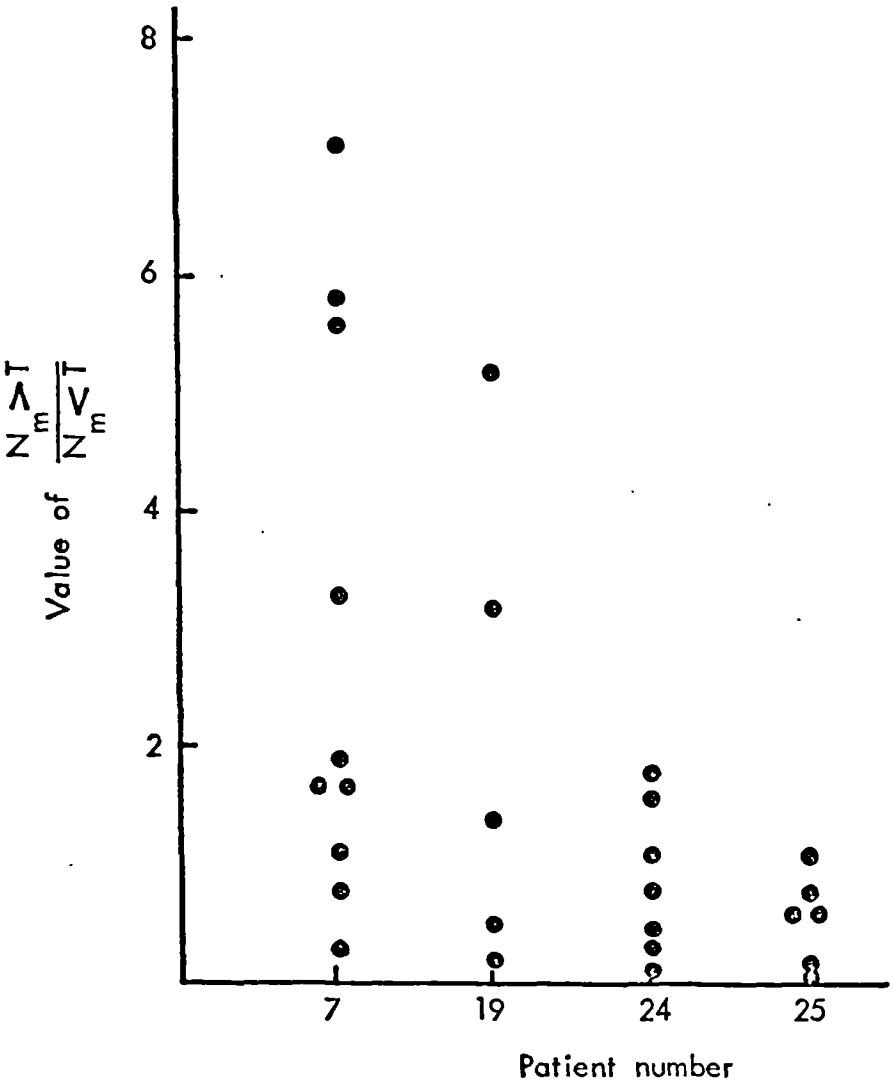


Figure 7.11d Ratio of $N_m > T$ to $N_m < T$, recorded for four of the patients on each night of the monitoring session.

7.5.2 Interval between moves

The time intervals between moves greater than the threshold value, were calculated for each night's recording and grouped into intervals of i_s, i_m and i_L (vide 7.4.2). "Large" moves (>T) were chosen as they were considered to be more likely to represent significant changes in interface pressure, than the total number of movements, which included those very small changes in position, associated with twitching, movements of the head etc. The reduced occurrence of moves separated by greater than 30 minutes (i_L), suggested that normalisation would imply a "bias" towards the i_s group, as they occurred more often throughout the night. For this reason, only non-normalised data were utilised in this section of the analysis, all incomplete nights recordings being discounted.

It had been established (vide 7.4.2) that the distribution of time intervals was lognormal. This precluded the use of parametric statistical tests. For this reason, the non-parametric χ^2 test was employed to compare patients' mobility, characterised by i_s, i_m and i_L.

The results from three of the patients (Patients 7, 23 and 25), monitored for five or more nights, were utilised to study the variation within patient's mobility (Table 7.5). Use of the χ^2 statistic and distribution for significance, requires that the smallest expected value of a contingency table be no less than 5 (Mounsey, 1964). The data relating to Patient 7 satisfied this condition, but that from Patients 23 and 25 did not. However, using "corrected tables" of significance (Craddock and Flood, 1970) which accounted for low frequencies in contingency tables of up to 5 x 5, it was possible to use the data from Patients 23 and 25. Results indicated that of the 44 pairwise comparisons, 19 (43%) proved to be significantly different ($p<0.1$) (Table 7.6). To compare the distribution of move time intervals between patients, the results of each were first pooled together (Table 7.7). Comparison was effected by application of the χ^2 statistic, the data from every patient being compared to each of the others. Of the 78 paired comparisons developed in this way, 52 (66%) were found to be significantly different ($p<0.1$) (Table 7.8).

Mean values of i_s, i_m and i_L per night, were calculated to be 26, 6 and 2 respectively.

7.5.3 Discussion

Characterisation of the data by the normalised number of movements (n_m) performed by the patient during a six hour sleep period, provided a sensitive measure of variations between patients. Further discrimination of the data by grouping relative to the number of "large" movements ($n_m > T$), "small" movements ($n_m < T$) and the ratio of "large" to "small" movements $N_m > T / N_m < T$, provided evidence that these parameters were also capable of characterising the data. The ratio F test of variance for each of the different movement magnitude parameters, indicated that n_m and $n_m > T$ produced similar results, whereas in comparison, those of $n_m < T$ and $N_m > T / N_m < T$ described larger and smaller variations between patients respectively. Comparison of means and standard deviations of the number of movements made by each of the subjects exhibited the same trend.

Subjects were found on average, to execute 63 movements per night (N_m), 33 greater than the threshold ($N_m > T$), 29 less than the threshold ($N_m < T$) with a ratio of large to small movements $\left(N_m > T / N_m < T\right)$ of 2.8. Equivalent normalised values were 134, 77 and 56 for n_m, $n_m > T$ and $n_m < T$ respectively (Table 7.4).

In later sections where the mobility data is compared to the clinical information gathered about each patient, particular emphasis is placed upon characterising the data using the parameter $n_m > T$ since this has been shown to be related to changes in interface pressure, (vide 4.6). In view of measuring mobility as a clinical tool for regular use on the ward, it may be of importance that the total number of movements (n_m), which is perhaps the most easily assessed of all of the parameters, appears to characterise the data in a manner similar to that of $n_m > T$. This result supports other studies, (eg. Exton-Smith and Sherwin, 1961) which have used the number of movements made during the night as the method for characterising their data.

Characterisation of the mobility data using parameters associated with time intervals between moves (i_s, i_m and i_L) also proved to be able

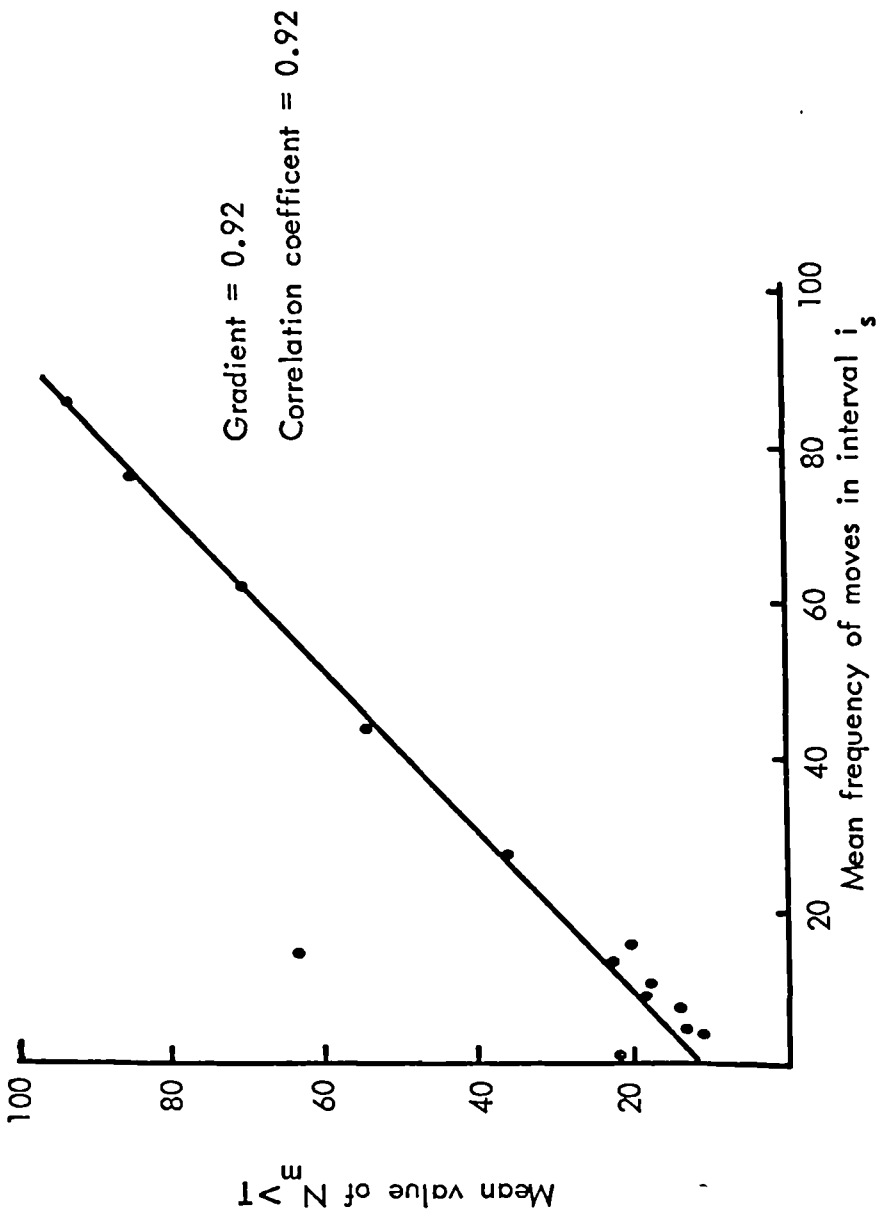
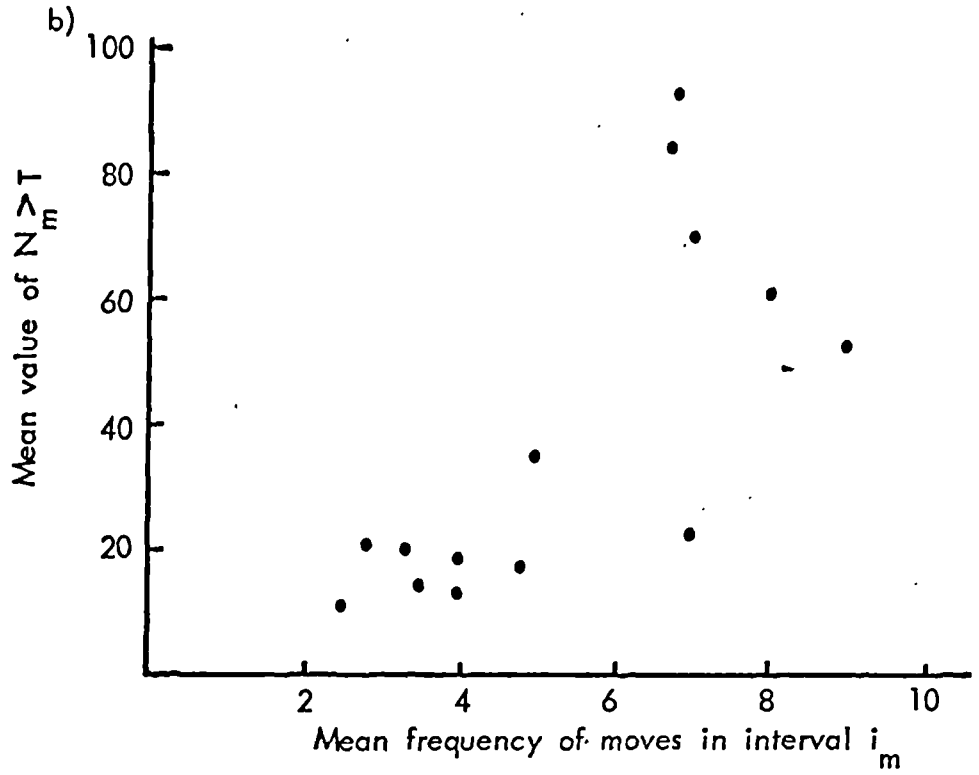


Figure 7.12a Mean number of moves, larger than the threshold, versus the mean frequency of moves in interval i_s for each patient.



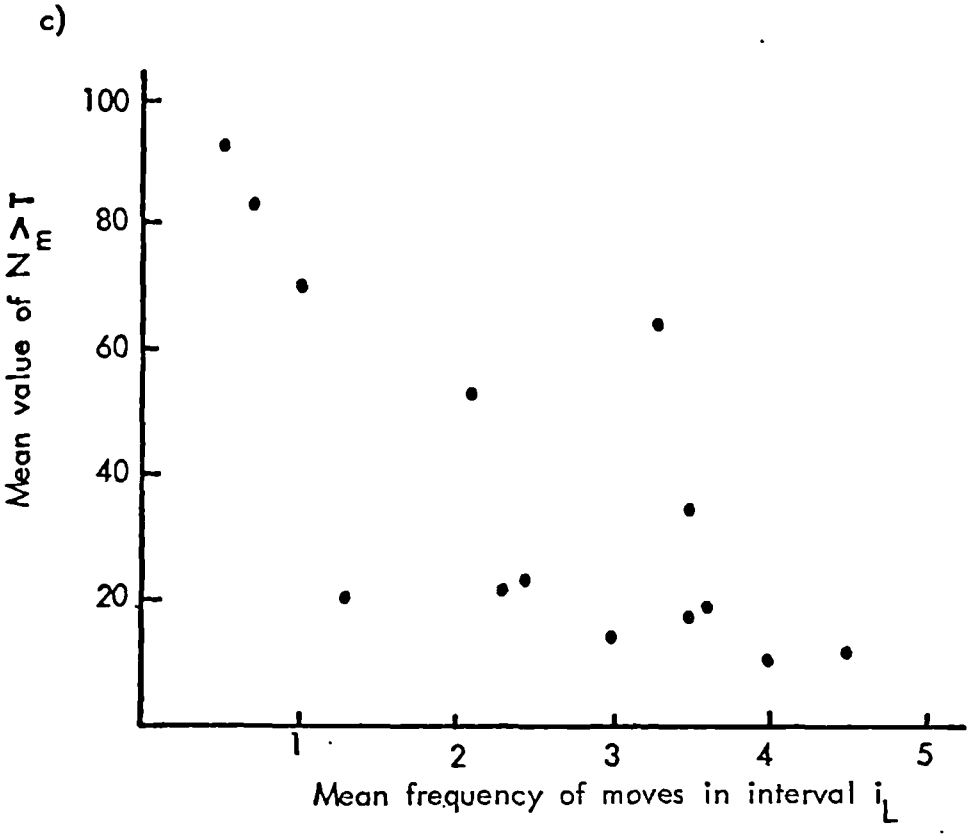
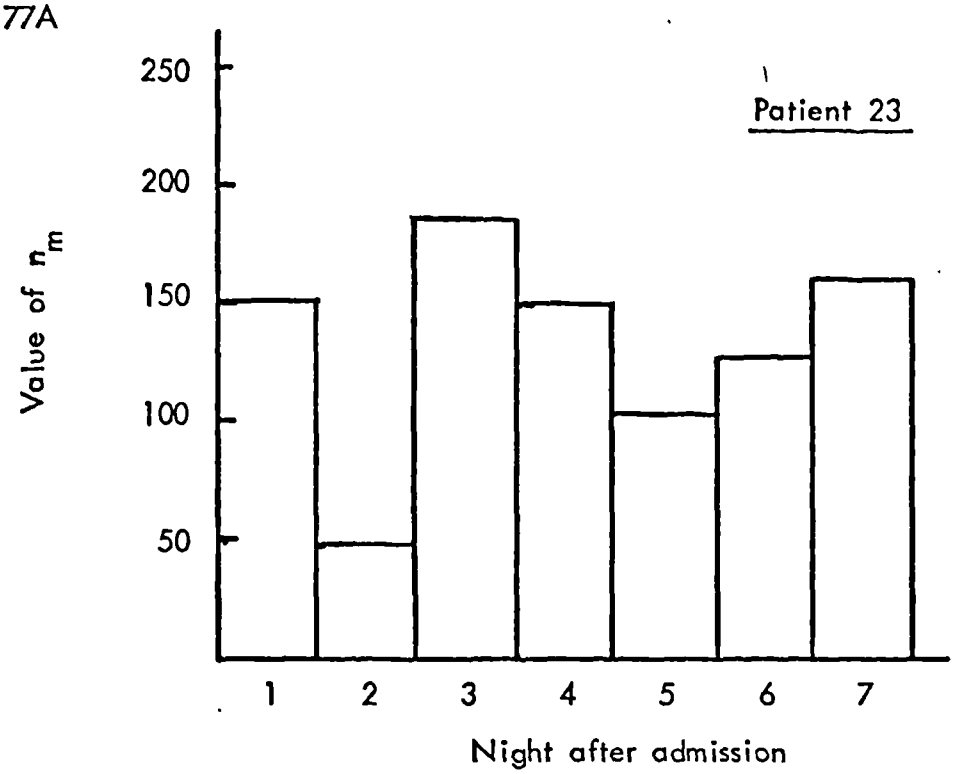


Figure 7.12b, c Mean number of moves, larger than the threshold, versus the mean frequency of movements in an interval, for each patient. b) i_m and c) i_L

to discriminate variations between patients. The number of significant differences between subjects, using this parameter (66%) was higher than that achieved when characterising the data by the number of movements. However, the magnitude of the variation within patients (43%), suggests that this set of parameters (i_s, i_m and i_L) should be employed with caution and that, if used in a comparison, any differences apparent statistically between patients may in fact be attributable to variations within the patient's own night to night mobility. For this reason the parameter relating to interval between movements was considered to be of restricted use in any quantitative analysis, but capable of providing useful qualitative information.

The frequency distribution of moves in i_s, i_m and i_L in two minute sample sizes was compared to the total number of moves, greater than the threshold value for each of the patients (figures 7.12a,b and c). A marked relationship between the total number of moves and the distribution group, i_s was evident (figure 7.12a), although this was not repeated for i_m and i_L. This suggested that the parameter i_s may not be independent and is a function of the total number of moves performed by a patient (n_m).



$$n_m = \frac{\text{Number of moves}}{\text{Length of sleep record (min)}} \times 10^3$$

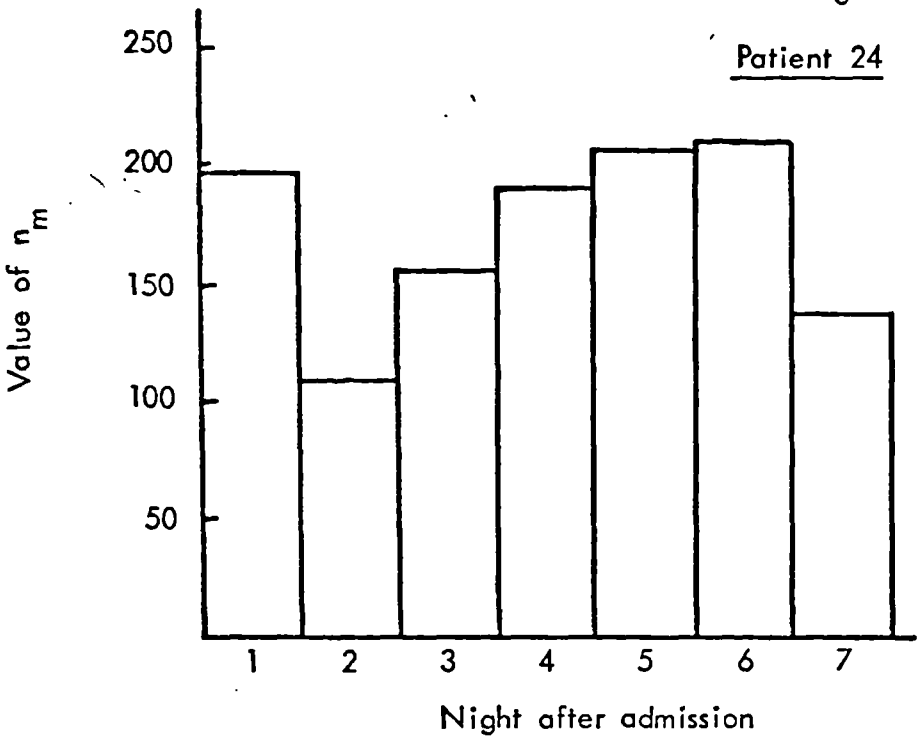


Figure 7.13a Number of movements (normalised) for 7 nights following admission.

Patients 23 and 24 demonstrating a reduction in mobility on the 2nd night of monitoring.

7.6 MOBILITY AFTER ADMISSION

7.6.1 Results

Variations in the total number of movements during sleep, for each recorded night of every subject were displayed. This suggested that some patients (the majority) exhibited a characteristic fall in mobility on the second night (figure 7.13a), although others showed an unchanged or increased mobility (figure 7.13b). Use of the other movement magnitude parameters, ie. $n_m > T$, $n_m < T$ and $N_m > T / N_m < T$ (figures 7.14, 7.15 and 7.16) also provided evidence of a corresponding reduction in mobility in the period following admission. From the information provided by these graphs the patients were divided into two groups;

- those who exhibited a fall in mobility on the second night after admission, followed by a recovery on the third night ("Fall").
- those who showed an increase in mobility on the second night after admission ("No Fall").

The groupings were compared using the χ^2 statistic. Results of the patients in each group were pooled to obtain an estimate of the mean number of moves on each of the first three nights following admission (for values of both n_m and $n_m > T$). Values in the resulting contingency table were used to calculate the χ^2 statistic (Tables 7.9a and b). There was a significant difference between the two groups for both characterisation parameters n_m and $n_m > T$ ($p < 0.001$).

However, there was no observable trend to the data after the third night (figures 7.13 to 7.16) and no evidence of stabilisation of any of the movement characterisation parameters.

7.6.2 Discussion

The distinctive drop in mobility over the period following admission may be explained as a result of sleep loss. On their first night, the anxiety associated with being in a new environment and with their illness, leads to the patient sleeping poorly and having a restless night, as indicated by an increased mobility. However, it has been established (Naitoh et al, 1973)

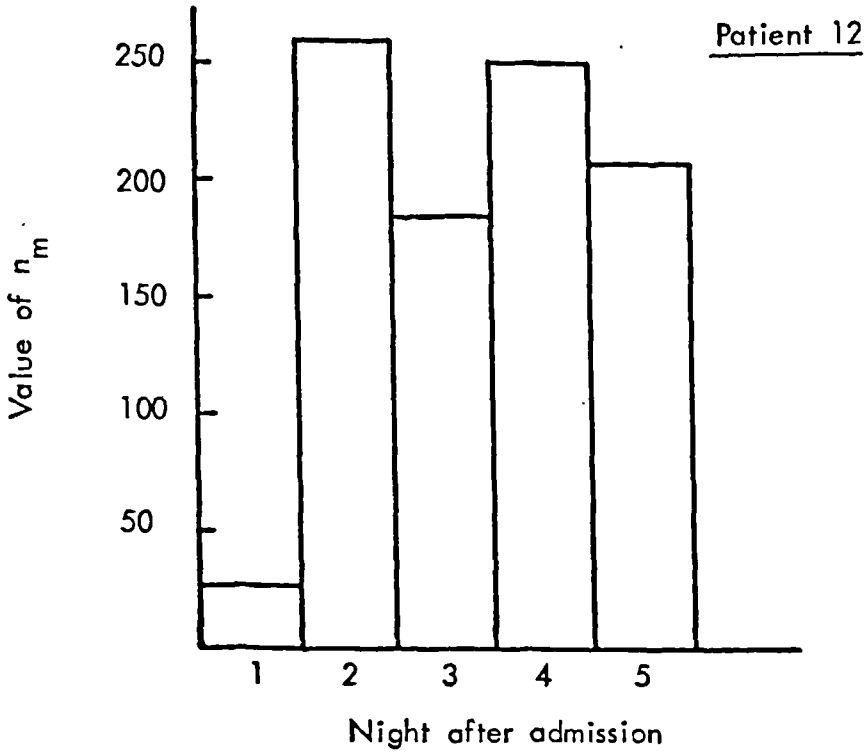


Figure 7.13b Number of movements (normalised) for 5 nights following admission.

Patient 12 , demonstrating an increase in mobility on the 2nd night of monitoring

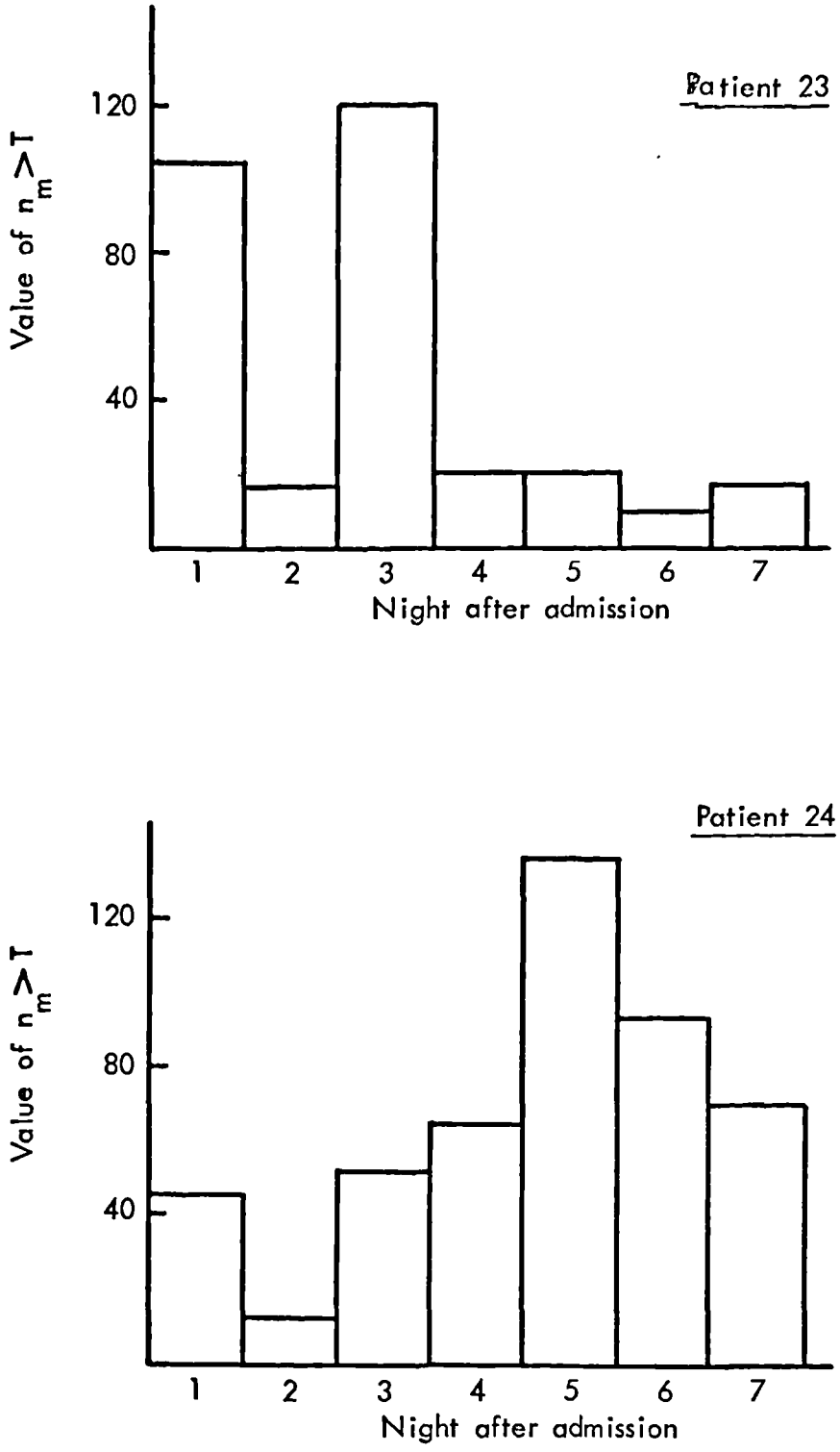


Figure 7.14 Number of moves larger than the threshold (normalised) for 7 nights following admission.

Patients 23 and 24 demonstrating a reduction in mobility on the 2nd night.

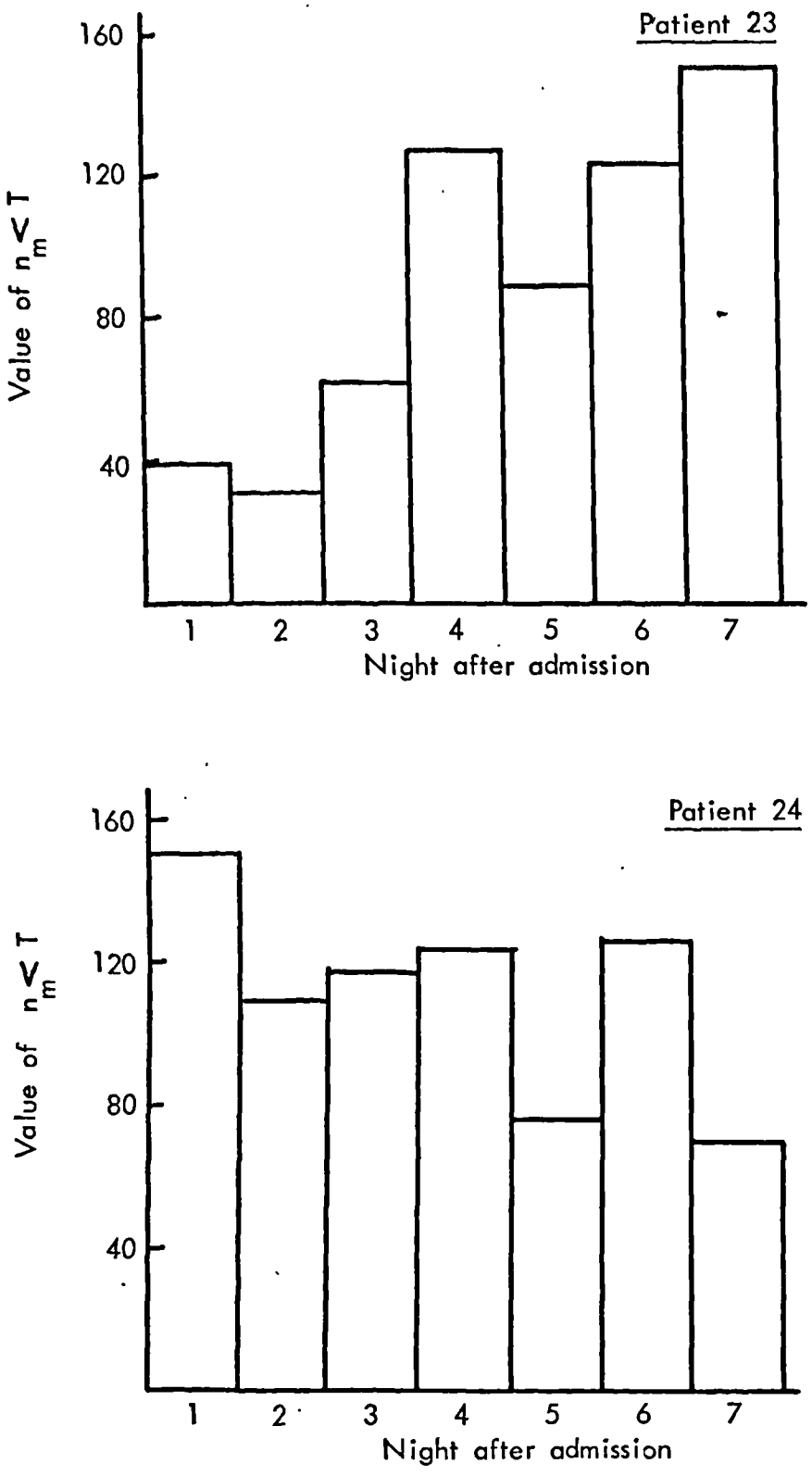
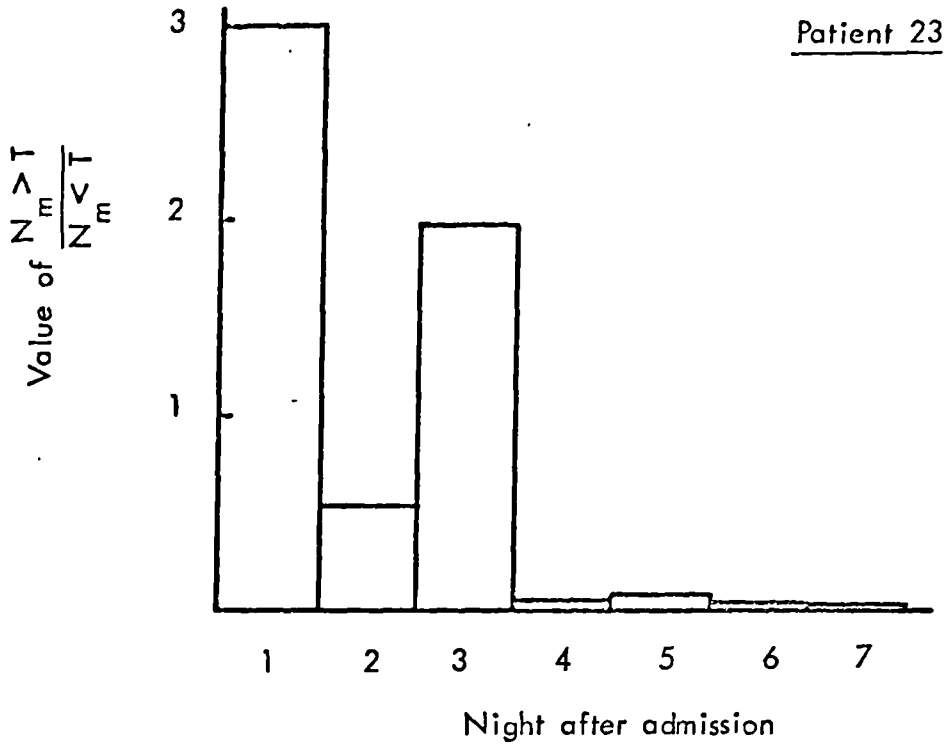


Figure 7.15 Number of movements less than the threshold (normalised) for 7 nights following admission.

Patients 23 and 24 demonstrating a reduction in mobility on the 2nd night of monitoring.



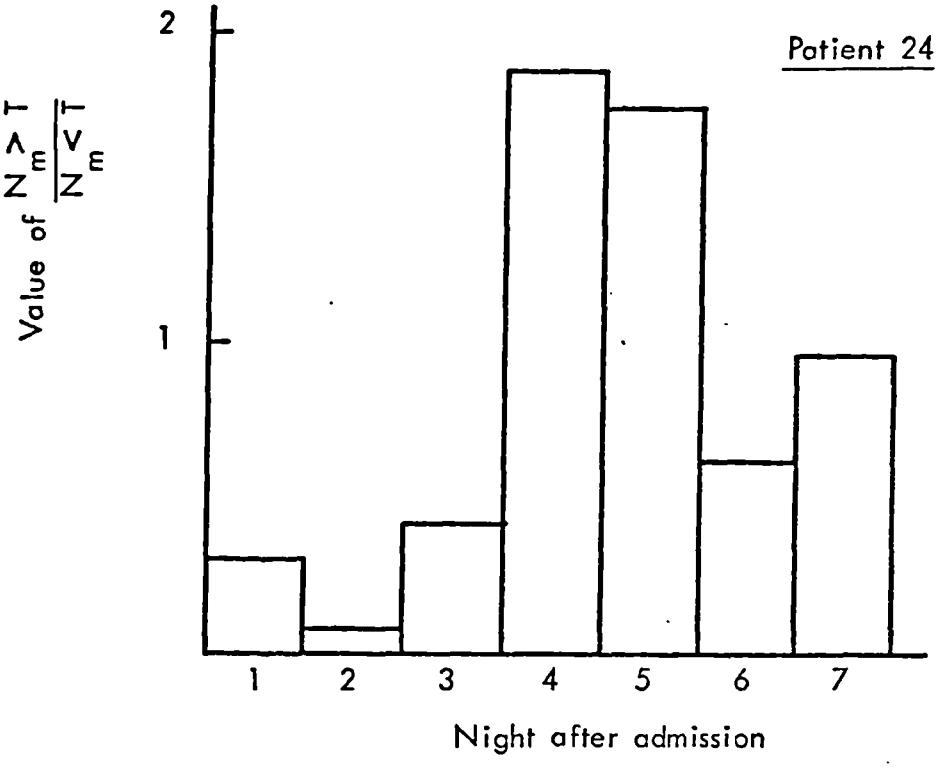


Figure 7.16 Ratio of movements larger than threshold to those smaller than threshold for 7 nights following admission. Patients 23 and 24 demonstrating a reduction in mobility on the 2nd night of monitoring.

that on the first night following a period of sleep loss, there is a significant reduction in the number of body movements, whereas the second night shows a recovery in the number of movements. The results of this study appear to conform to this theory.

It is not clear from the amount of data presented by Bardsley (1977) whether this characteristic was repeated in his study of normal young subjects, although of the three examples he provides, two of them do display a drop in mobility on the second night of monitoring. Additionally, he also mentions that the first night's results were generally unrepresentative of a subject's mobility.

The possible importance of this characteristic is illustrated by the results of Patients 4 and 6 of this study, who on their second night both failed to make even one move larger than the threshold value.

Clinically, these results suggest that those patients newly admitted to hospital be carefully assessed, as to their susceptibility to pressure sores with particular emphasis on their ability to maintain unaided pressure relief of the tissues over the bony prominences. Although a reduction in mobility may not significantly increase the vulnerability for pressure sores of those patients who are naturally highly mobile during sleep, it will be of significantly more importance to those already suffering from poor mobility or who are "at risk" from other factors.

7.7 PATIENT DATA INFORMATION

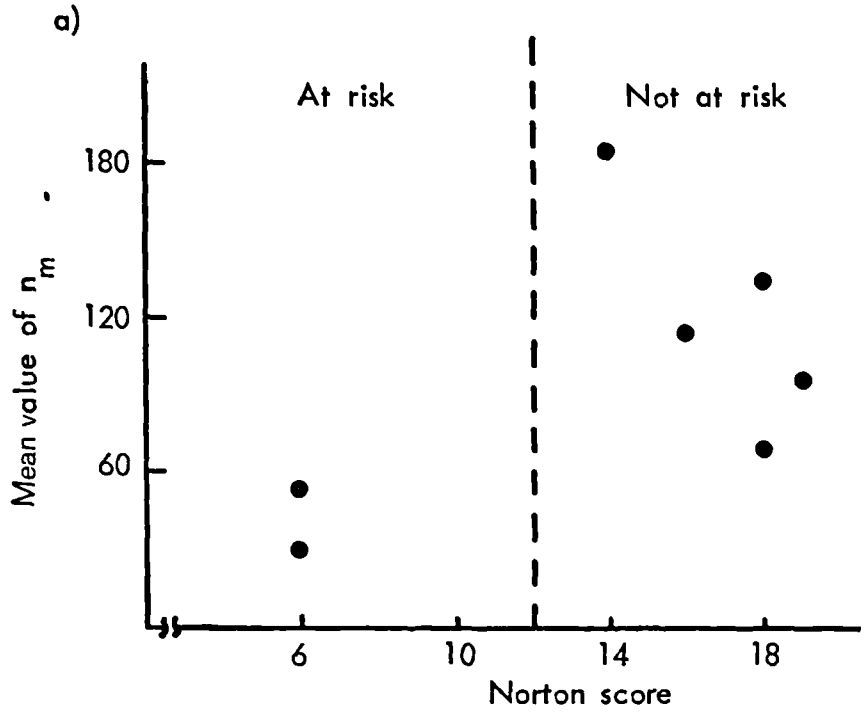
The information collected on the patient record forms (vide 6.3) was grouped together into sections relating to sedation, incontinence, pressure sores, pressure sore prophylaxis and diagnosis. The tests on Patients 4, 5, 6 and 7 employed a modified system of patient data recording and did not contain a daily record of the Norton score, although an overall assessment of the patient's condition was provided.

In total, the overnight mobility of 20 elderly hospitalised patients was monitored for up to ten consecutive nights each. However, due to unforeseen breakdowns of the monitoring apparatus, usually as a result of failure of one of the strain gauge amplifiers (vide 5.7.2), the results from three of these patients were discarded. A further one of the patients refused to be monitored part-way through a series of tests and the results from this patient were also rejected. Additionally, the patient data record form for one of the subjects was incomplete and was therefore not included. Thus, the patient data records of 15 of those monitored were analysed.

- 3 (20%) were under regular sedation, while another was intermittently sedated. Two drugs were used, Melleril and Temazepam.
- 8 (53%) were incontinent. Of these 4 (27%) suffered from urinary incontinence only, 1 (7%) from faecal incontinence only and 3 (20%) were doubly incontinent.
- 3 (20%) had a pressure sore. One of these was classified as a grade 4 sore (Barbenel et al, 1977), another as grade 2, while the third was limited to a persistent patch of redness of the skin, with no tissue breakdown.
- 7 (40%) were receiving regular care for pressure sore prophylaxis. All of these patients were being turned. The nursing staff usually employed a four hourly turning regime for patients without sores and a two hourly one for those with sores. 5 (33%) of the patients were additionally using "large cell" ripple mattresses.
- 10 (67%) of the patients could be classified as belonging to one of two different diagnostic groups. However, information on a patient's illness was confused since in the majority of cases, the subject exhibited symptoms

of more than one complaint. As a result, only those cases were considered in which it was possible to define a primary diagnosis. With this limitation the two most commonly occurring diagnoses were diseases of the circulatory system, 5 (33%) and diseases of the nervous system, 5 (33%).

- 3 (20%) of the patients died during the course of a monitoring session.



$$n_m = \frac{\text{Number of moves}}{\text{Length of sleep record}} \times 10^3$$

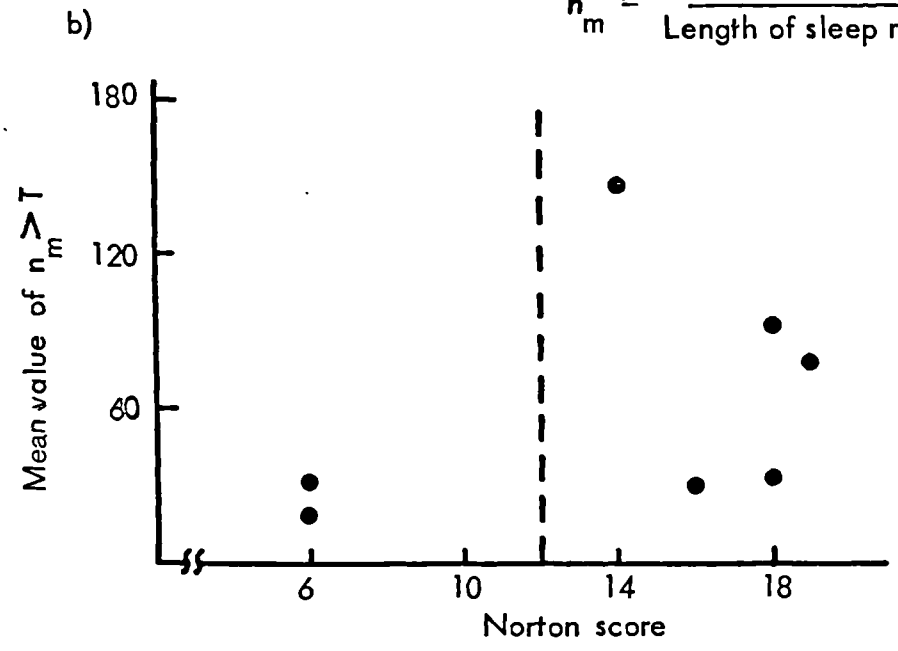


Figure 7.17a,b "Stable"patients mobility relative to their Norton score.
a) value of n_m and b) value of $n_m > T$

7.8 COMPARISON BETWEEN THE MEASUREMENT OF MOBILITY AND THE NORTON SCORE

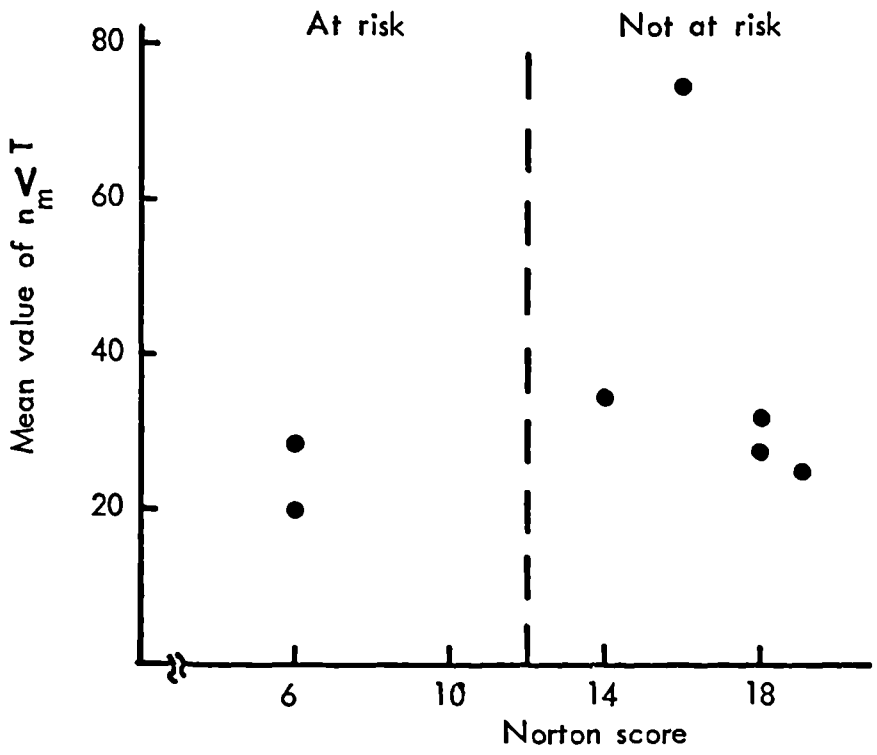
Mobility has been identified as one of the primary factors in the aetiology of pressure sores and other studies have indicated the relation between low mobility and pressure sore incidence (Exton-Smith and Sherwin, 1961). As a first step in establishing the clinical relevance of mobility monitoring it was thought useful to compare values of mobility for each patient, to an acknowledged assessment technique for pressure sores which has been substantiated by quantitative evidence - the Norton score. To achieve this, Norton scores were assessed on each night of the study by one of the nursing staff.

7.8.1 Results

Norton score assessment of the patients suggested two natural groupings. Firstly, those whose score was consistent (within $\pm$ 1) over the duration of the study ("All Stable"). Secondly, those whose Norton score changed by two or more points over the duration of the study. All of the patients in this group exhibited an increase in their score ("Improved"). The group with consistent scores was further subdivided into those with scores greater than 12 ("Stable >12") and with scores less than or equal to 12 ("Stable<12") (Table 7.10).

For the "Stable" group the mean number of moves ($\overline{n_m}$) was plotted against the Norton score for each of the patients (figure 7.17a), if the score changed during the duration of the study, then the smallest value was used. Figure 7.17a demonstrated that the size of Norton score assessed for a patient was related to her overnight mobility, as characterised by n_m. Similar graphs were drawn for parameters $n_m > T$, $n_m < T$ and $N_m > T / N_m < T$ (figures 7.17b, c and d). Figure 7.17b indicated that a corresponding relationship existed between Norton score and the number of moves larger than the threshold, although this feature was less evident than that from the data characterised using n_m. Figures 7.17c and d suggested that when the mobility was characterised by parameter $n_m < T$ and $N_m > T / N_m < T$ there was no corresponding relationship between Norton score and mobility.

c)



d)

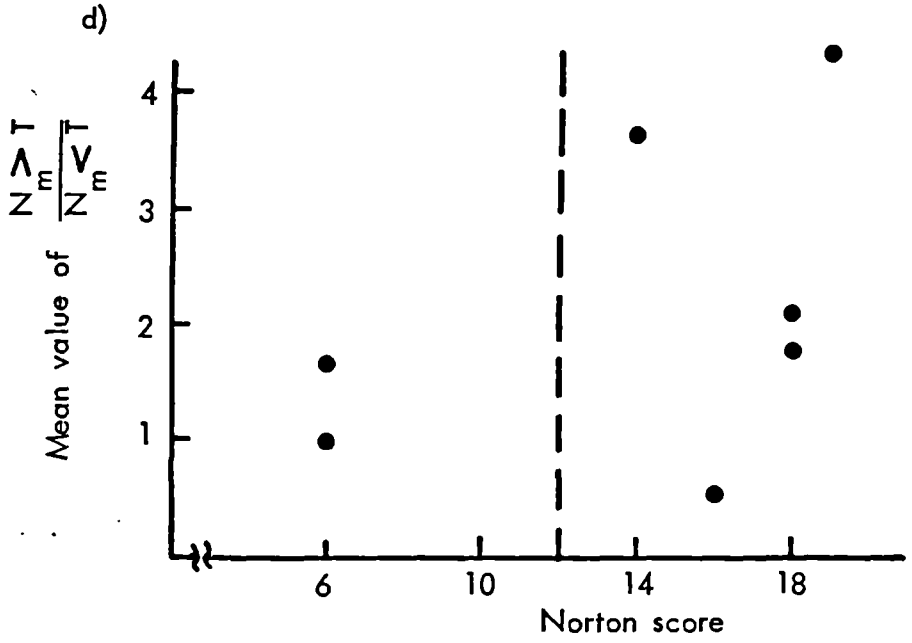


Figure 7.17c,d "Stable " patients mobility relative to their Norton score.

c) mean value of $n_{m<T}$ and d) mean value of $\frac{N_{m>T}}{N_{m<T}}$

The results from the patients in the "Improved" group, were subdivided in relation to the Norton score on the first night of monitoring and on the last. The mean number of movements, corresponding to each subgroup, was then calculated and plotted against the initial and final Norton score, for each patient (figure 7.18a). A similar procedure was adopted for the movement parameters $n_m > T$, $n_m < T$ and $N_m > T / N_m < T$ (figures 7.18b, c and d).

In the case of n_m, for all of the patients, an increase in the Norton score was associated with a corresponding rise in the number of movements, although in some cases this was marginal (Patients 16 and 23). The data characterised by parameters $n_m > T$, $n_m < T$ and $N_m > T / N_m < T$ failed to show any corresponding correlation. The χ^2 test was utilised in a statistical comparison between the "Stable >12" and "Stable<12" subgroups for each of the characterisation parameters n_m, $n_m > T$, $n_m < T$ and $N_m > T / N_m < T$ (Tables 7.11a and 7.12a, b and c). "Improved" and "All Stable" subgroups were also compared, but for n_m only (Table 7.11b).

The increased mobility of stable patients, with a high Norton score, as opposed to those with a low score, evident from figures 7.17a and b, was statistically significant for n_m and $n_m > T$ at $p<0.1$ and $p<0.01$ respectively. However, if the number of movements was characterised using $n_m < T$ and $N_m > T / N_m < T$ there was no significant difference between the groups. The corresponding increase in the value of $\overline{n_m}$ for those patients who showed an improvement in their Norton score, as demonstrated in figure 7.18a, was statistically significant ($p<0.05$).

7.8.2 Discussion

The results suggest that the number of movements made by a patient in bed during sleep, and in particular large movements, significantly correlate with the Norton score, a well established technique for assessing the vulnerability of patients to pressure sores (vide 6.2.3). The study of "small" moves (<T), or the ratio of"large"to"small"moves, did not provide an equivalent identification. This is probably attributable to the assessment criteria for the

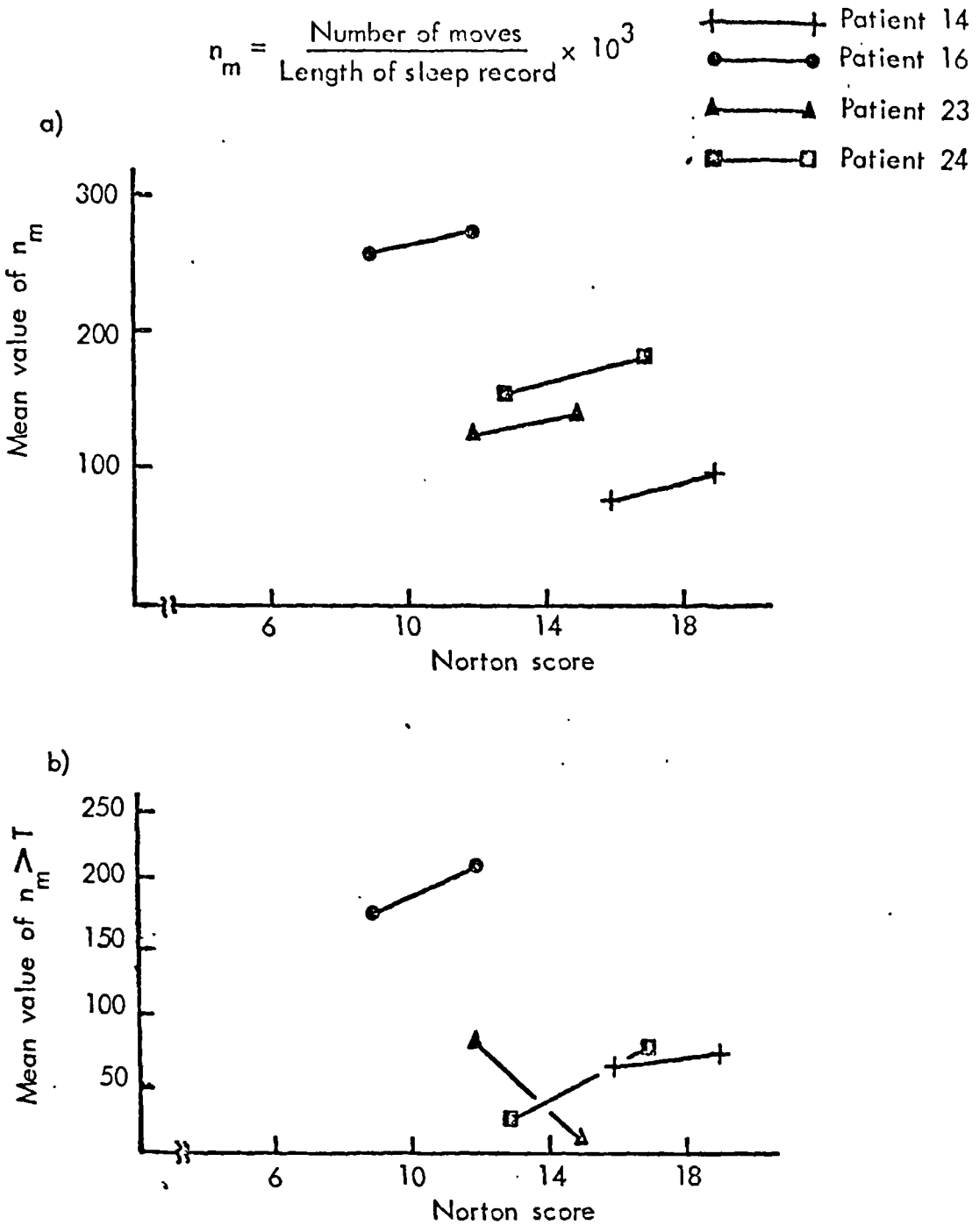


Figure 7.18a,b "Improved"patients mobility relative to their Norton score. a) mean value of n_m and b) mean value of $n_m > T$

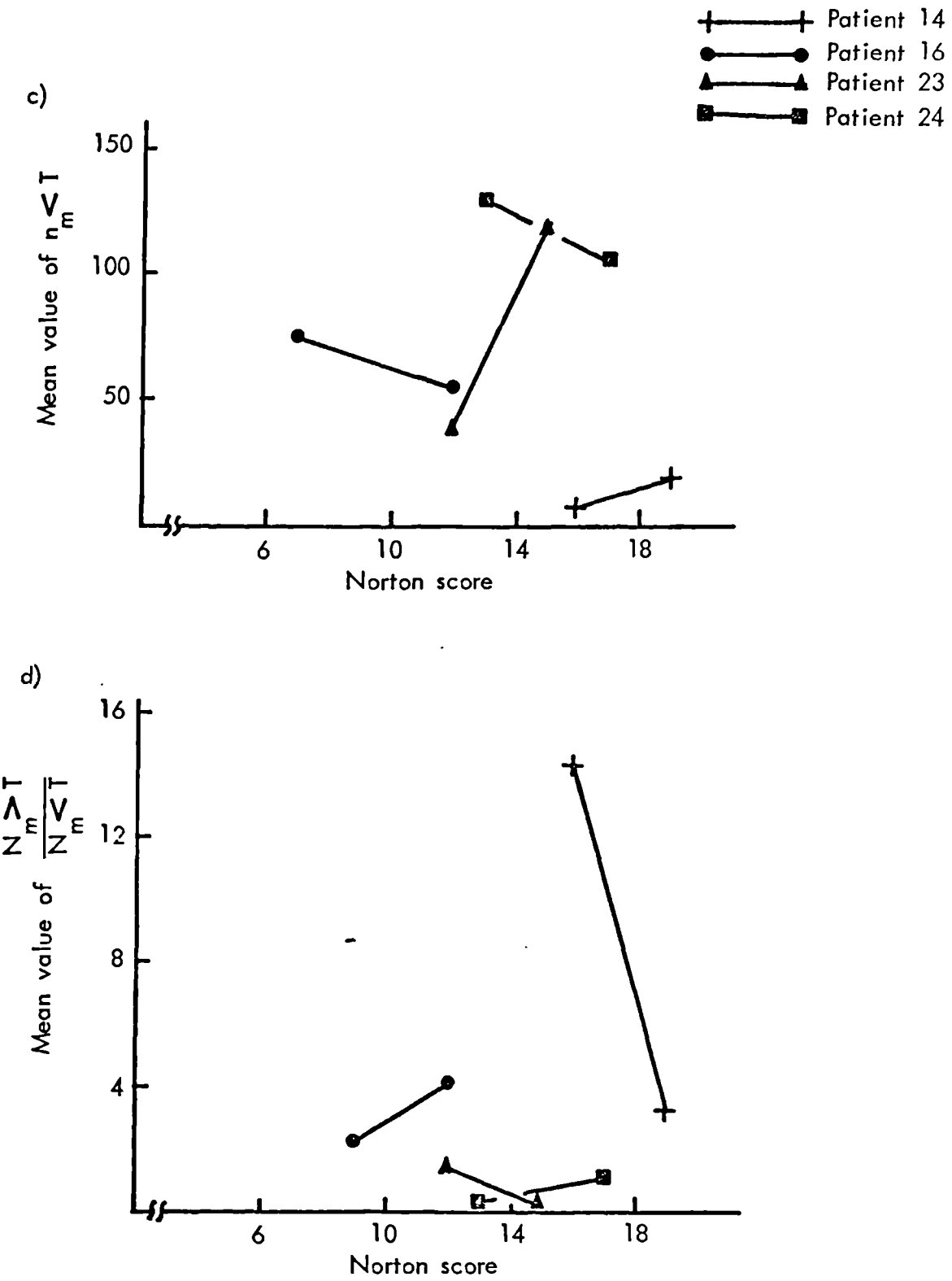


Figure 7.18c,d "Improved" patients mobility relative to their Norton score.

c) mean value of $n_m < T$ and d) mean value of $\frac{N_m > T}{N_m < T}$

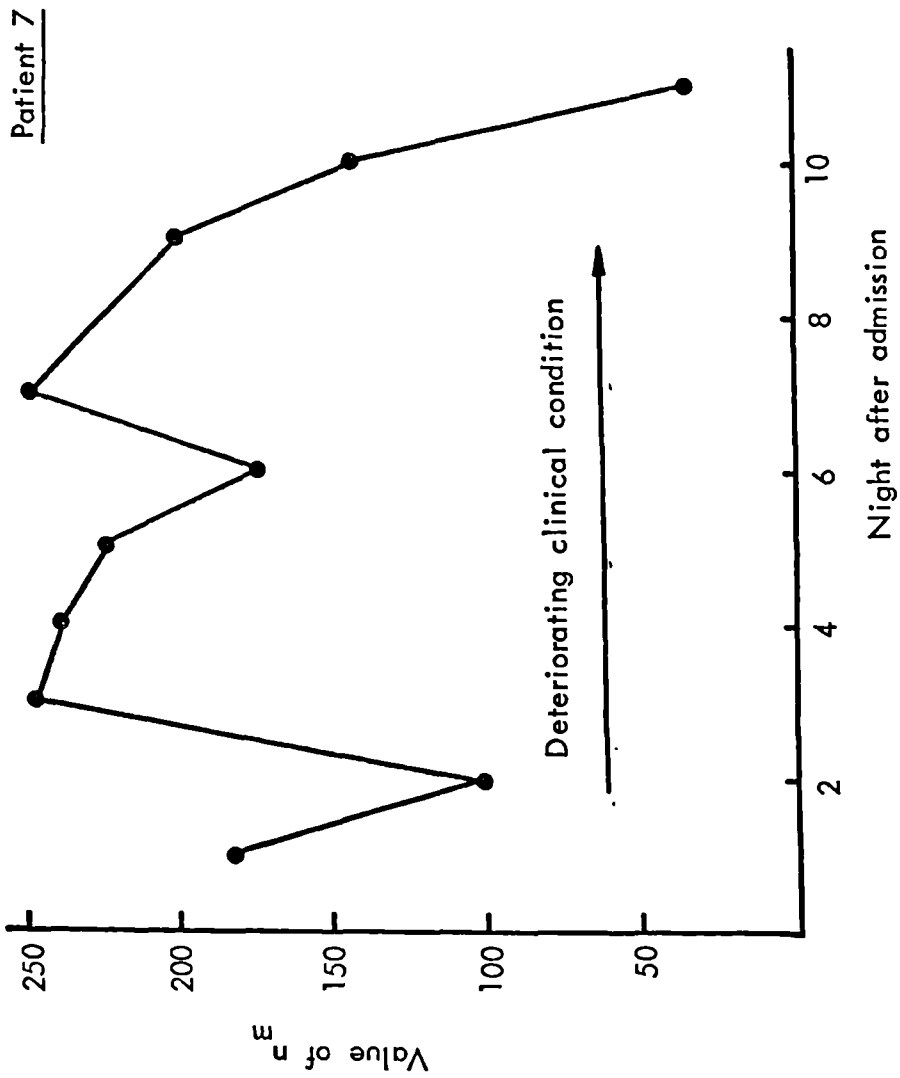


Figure 7.19 Number of movements (normalised) performed by Patient 7 for each night of the monitoring session.

Norton score. This data does not therefore provide evidence for abandoning these parameters in the assessment of vulnerable patients, but their usefulness can only be established by an extensive study of the relationship between mobility parameters and pressure sore incidence.

The marginally significant relationship between the number of movements and an increasing Norton score suggests that mobility may be related to changes in clinical condition. However, the results of this study indicate that only marked changes in clinical condition show as corresponding changes in the patient's mobility. Patient 7 provides an example of this (figure 7.19). A 70 year old lady was admitted to hospital suffering from diseases of the cardiovascular system. She exhibited a typical sleep loss effect in her mobility over the first three days after admission (vide 7.6). Thereafter her mobility remained relatively stable, although her record showed that her clinical condition was gradually deteriorating. However, on the tenth night after admission she suffered repeated atrial fibrillation and although this was reversed, her physical condition deteriorated rapidly and it continued to do so until she died two days later. The patient's overnight mobility showed a sharp decrease over the same period. Thus, for the interval of time that the patient's condition was slowly deteriorating there was no associated change in her mobility and only when the major and rapid deterioration ensued was the mobility affected.

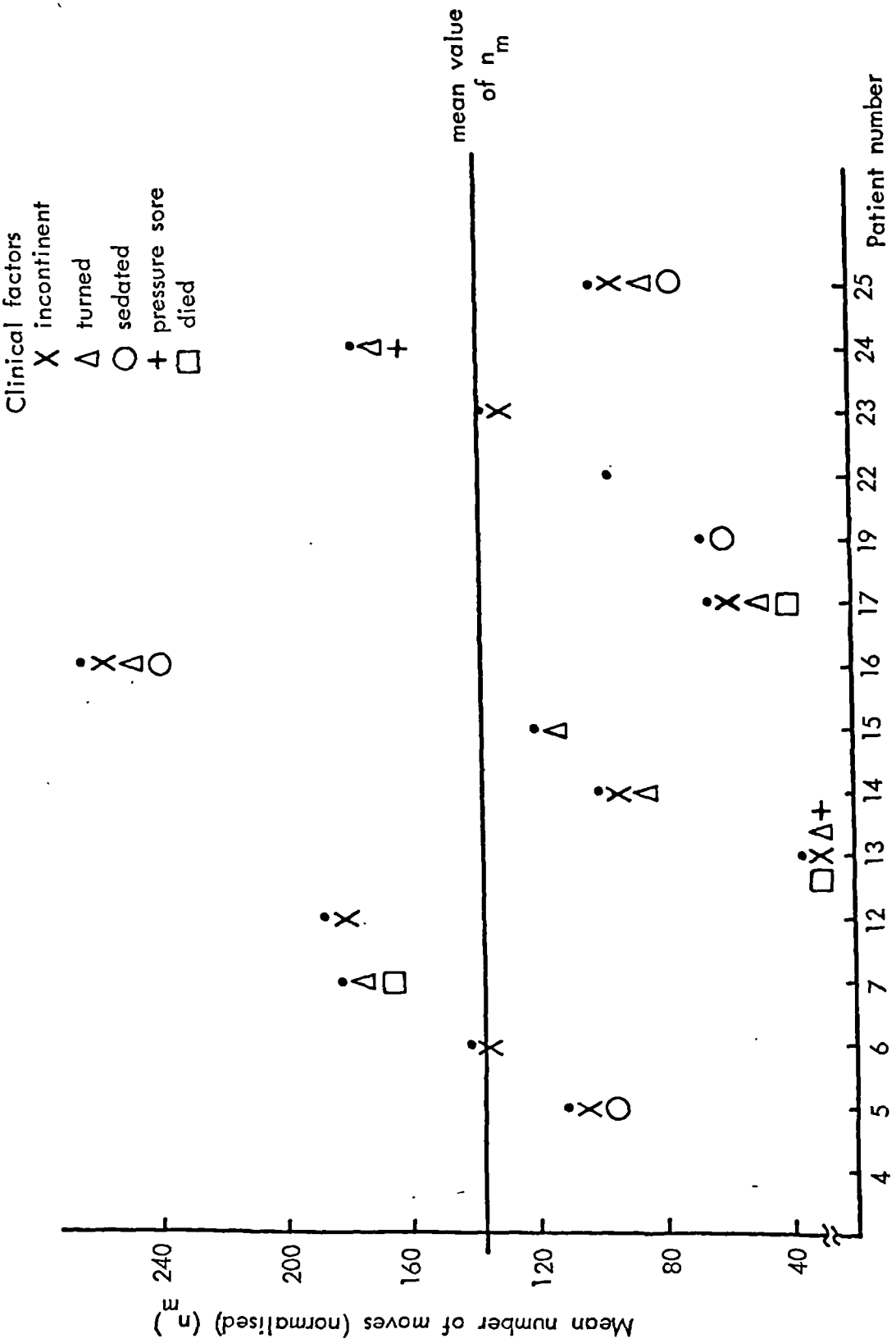


Figure 7.20 Mean number of moves (normalised) and recorded clinical parameters, for each of the patients.

7.9 RECORDED CLINICAL DATA AND MOBILITY

Figure 7.20 displays a summary of the results of the clinical data, indicating those patients who were turned, sedated, incontinent or who died, in association with the mean number of moves (normalised) made by each of them. Additionally the population's mean mobility is included. These results emphasise the wide variability between patients.

7.9.1 Sedation

From figure 7.20 it is evident that below average mobility is associated with the patients receiving sedatives. To provide quantitative evidence of the statistical significance of this observation, the patients were pooled into two groups; those who received sedation ("Sedated") and those who did not ("Not Sedated") (figure 7.21a). The results of those patients in the "Sedated" group were combined and the mean and standard deviation of n_m (pooled) was calculated. The means and standard deviations of n_m for the two groups were then compared using the student's t test (Table 7.13). This demonstrated a statistically significant difference between the groups ($p<0.05$). This correlation between overnight mobility and sedatives was investigated further by employing some of the other characterisation parameters, established in section 7.4 ($n_m > T$, $n_m < T$, $^{N}m > T / N_m < T$) (figures 7.21b, c and d) (Table 7.13). Statistical comparison between the "Sedated" and "Not Sedated" groups showed a significant difference for $n_m > T$ ($p< 0.01$), but comparison between the groups was not significant for $n_m < T$ and $^{N}m > T / N_m < T$ (Table 7.13).

The effect of sedatives on the distribution of time intervals between moves was also investigated. For each group "Sedated" and "Not Sedated", the results of the patients with regard to parameters i_s, i_m and i_L were pooled together (Table 7.14) and compared using the chi-squared statistic. The two groups were found to be significantly different ($p< 0.001$), with an increase in the mean value of i_L and a reduction in the mean value of i_s, for the sedated patients, relative to those not sedated (Table 7.14).

The small number of patients in the "Sedated" group precludes any firm quantitative conclusions about the affect of sedation on mobility.

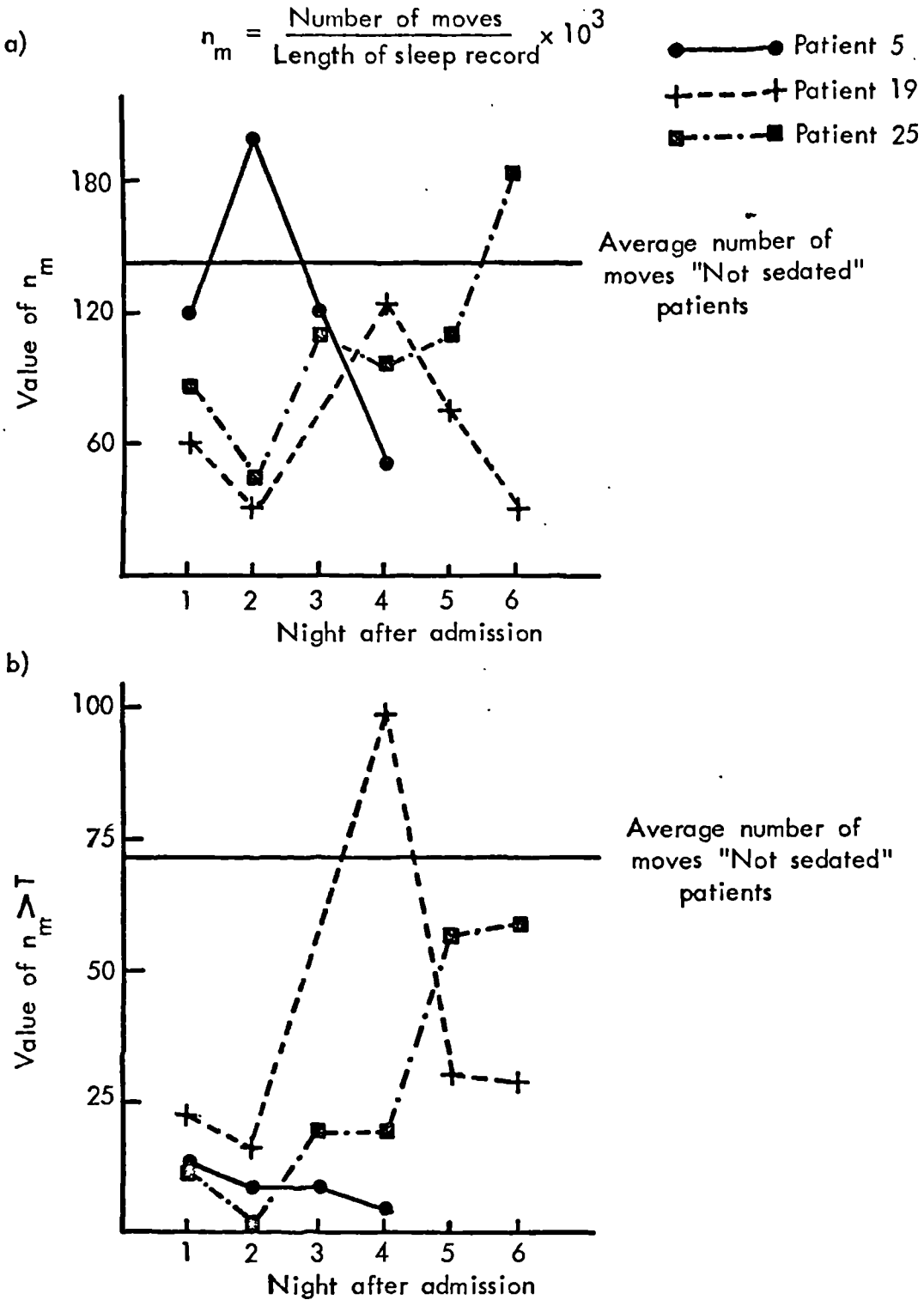


Figure 7.21a,b Mobility of "Sedated" patients compared with the mean value of those "Not sedated".

a) value of n_m and b) value of $n_m > T$

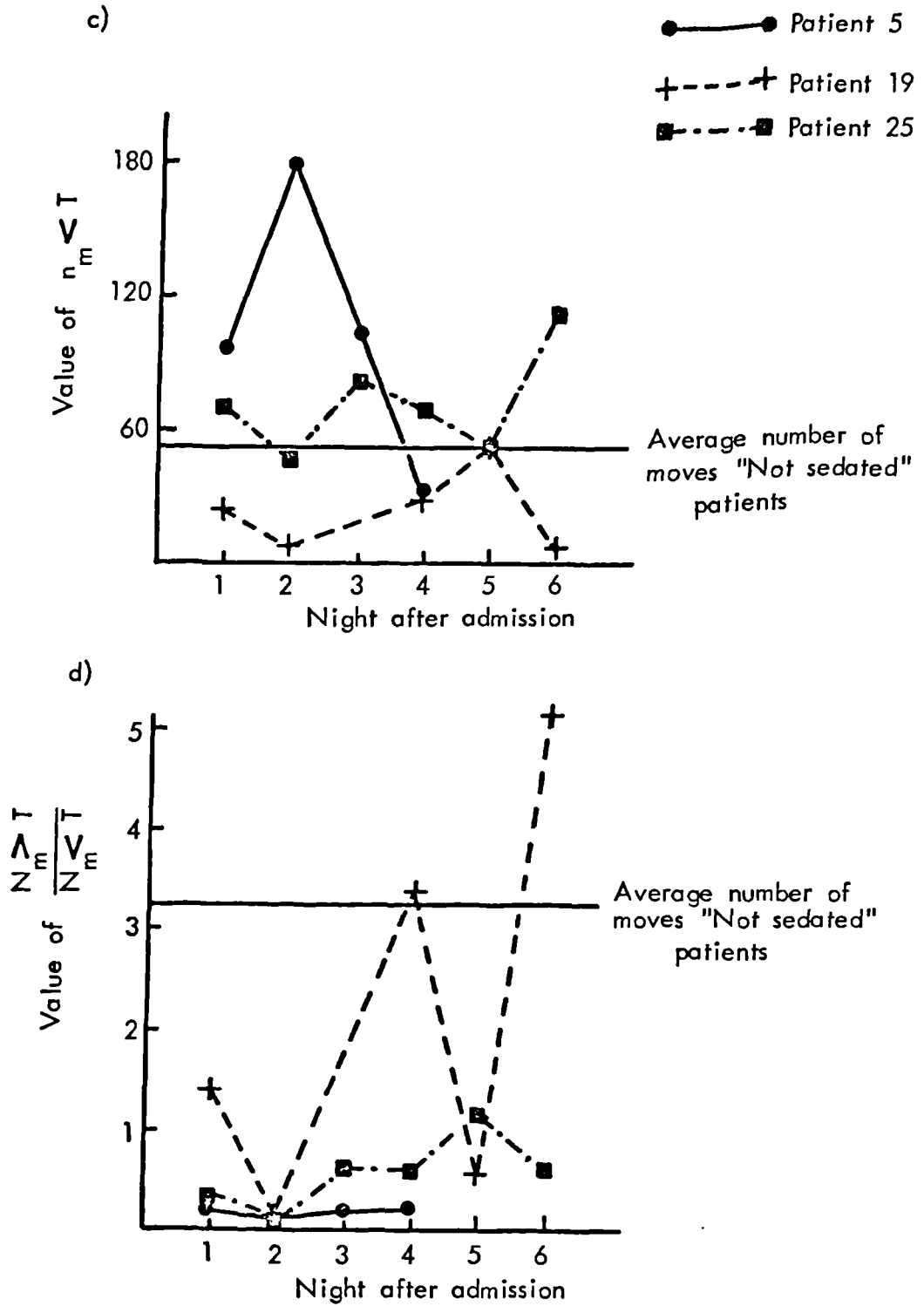


Figure 7.21c,d Mobility of "Sedated" patients compared with the mean value of those "Not sedated".

c) value of $n_m < T$ and d) value of $\frac{N_m > T}{N_m < T}$

However, the trend of the results is in agreement with those of Hinton (1961), and Exton-Smith (1967) who have shown that the use of the vast majority of sedatives results in a reduction of the number of body movements (vide 3.3.4).

The comparison between the "Sedated" and "Not Sedated" groups with respect to movement magnitude, suggests that sedatives may act to decrease the number of larger movements made during the night, while not affecting the number of smaller movements. Such an observation may be of particular importance in relation to pressure sores, since larger movements are responsible for substantial relief of pressure and any diminution in their number may serve to increase a patient's susceptibility to pressure sores.

The distribution of the time interval between movements was altered for those patients receiving sedatives. Clearly, an increase in duration between movements means a longer time between relief of the ischaemia, increasing the risk of damage to the tissues. As the time interval between moves was defined as that interval between large moves only (vide 7.5.2), then the possible relationship between large movements and sedatives is reinforced.

7.9.2 Other clinical parameters

Section 6.2 outlined typical pressure sore assessment techniques for patients in hospital. If used they provided a measure of the possible susceptibility of patients to pressure sores. Based on this information, care could then be ascribed to those patients most requiring it. One of the widely used methods of prevention, involves regular turning of the patient. The results presented in this sample suggest that on the ward at least, those patients who have been selected as "high risk" and are being turned, do not always have a low mobility (figure 7.20). In addition some of the patients with a reduced mobility are not being turned.

Of those patients that died during the course of a monitoring session, two (Patients 13 and 17) exhibited a mobility well below average. This was probably due to their poor clinical condition. However, one of the patients (Patient 7) who died, had a mobility above average. Details of this case were

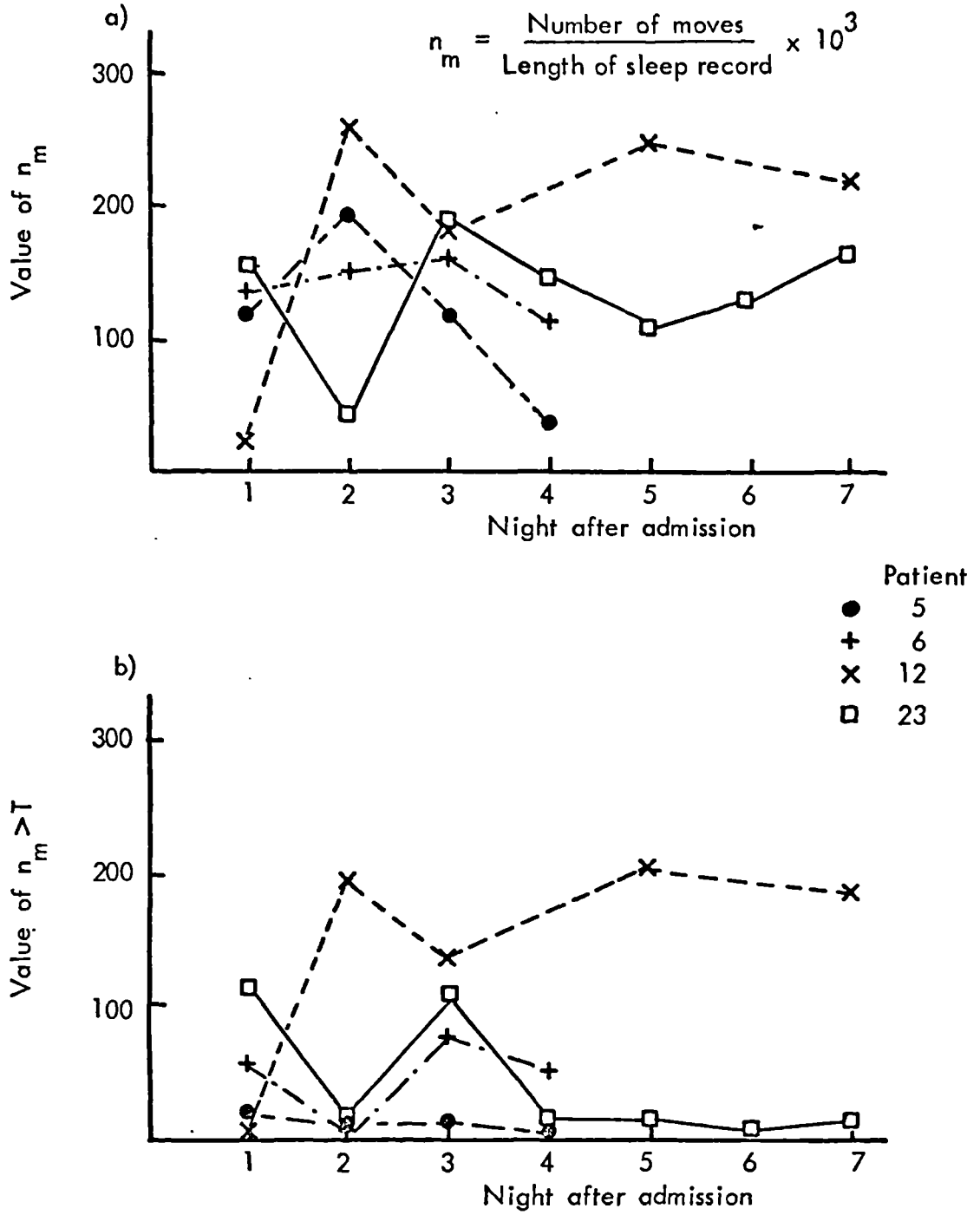


Figure 7.22a,b Number of moves, per night, of those patients suffering from urinary incontinence. a) Value of n_m and b) Value of $n_m > T$

presented in section 7.8. This level of mobility was attributed to the patient being very active in the earlier part of her stay in hospital, before her clinical condition deteriorated.

Both of those patients with sores (Patients 13 and 24) were being turned. One of these (Patient 13) had a low mobility while the other (Patient 24) was above average. However, the results of Patient 24 have been more closely examined (vide 7.2.1) and have shown that on admission to the ward her mobility was reduced and it improved steadily, as did the condition of her sore, leading to an overall mean value above average.

One of the patients (Patient 16) exhibited a markedly high mobility throughout the period of the monitoring session, although her Norton score was only around 12 and she was being turned. However, information from her assessment form (vide 6.3) indicated that she was suffering from a disease of the nervous system (brain tumour) which was causing repeated seizures and muscle spasms, thereby accounting for her high mobility.

Results from the patients in this sample, provided no evidence of a link between mobility and incontinence (figure 7.22a and b).

Parameter	Variance		F Value	Significance Level
	Within	Between		
n_m	3253.17	11600.66	3.57	$p<0.01$
$n_m > T$	2529.88	9977.99	3.94	$p<0.01$
$n_m < T$	51571.62	876.63	6.36	$p<0.01$
$\frac{N_m > T}{N_m < T}$	22.31	47.36	2.12	$p<0.025$

Table 7.1 Variations within and between the sample population.

Patient Number	Parameter							
	n_m		$n_m > T$		$n_m < T$		$N_m > T / N_m < T$	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
4	133.8	46.0	44.3	55.9	89.5	27.3	0.6	0.9
5	117.3	63.3	14.0	5.7	103.3	60.5	0.2	0.1
6	140.8	20.0	45.7	32.4	96.5	37.7	1.0	1.0
7	177.0	69.5	125.0	63.4	51.8	25.9	3.0	2.4
12	186.4	96.9	147.2	84.9	39.2	18.6	3.7	2.5
13	36.0	19.8	18.5	17.7	17.5	2.1	1.0	0.9
14	97.8	72.8	79.2	52.7	18.6	21.1	7.9	7.3
15	120.3	13.0	96.0	17.3	24.3	21.2	1.6	2.2
16	268.0	40.9	220.3	50.0	47.8	32.6	12.8	18.5
17	62.3	30.0	39.7	26.6	22.7	3.5	1.7	0.9
19	66.0	39.4	42.8	31.1	23.2	18.4	2.2	2.1
22	97.6	83.6	77.0	77.3	20.6	12.4	4.4	2.8
23	134.1	44.3	45.7	48.9	88.4	46.7	0.9	1.2
24	177.3	37.7	67.4	39.1	110.4	30.0	0.9	0.7
25	105.0	45.0	34.8	24.1	70.2	25.9	0.5	0.4

Table 7.2 Mean and standard deviation values for each of the patients studied.

Patient Number	Patient Number															
	4	5	6	7	12	13	14	15	16	17	19	20	22	23	24	25
4		0.4	0.2	1.1	0.9	2.4^{+}	0.8	0.4	3.8$^{\bullet}$	2.0^{+}	2.1^{+}	0.7	0.7	0.0	1.5	0.9
5			0.6	1.4	1.1	1.5	0.4	0.1	3.5$^{\bullet}$	1.2	1.3	0.4	0.4	0.5	1.8	0.3
6				1.0	0.8	5.0$^{\bullet}$	1.0	1.3	4.8$^{\bullet}$	3.5$^{\bullet}$	3.0^{+}	1.1	0.9	0.3	1.6	1.3
7					0.2	2.6^{+}	1.9	1.3	2.3^{+}	2.6^{+}	3.1^{+}	1.6	2.0^{+}	1.4	0.0	2.1^{+}
12						1.8	1.5	1.0	1.4	1.9	2.3^{+}	1.2	1.6	1.2	0.2	1.7
13							1.0	4.5$^{\bullet}$	6.2$^{\vee}$	0.8	0.9	1.0	0.9	2.7^{+}	4.5$^{\bullet}$	1.8
14								0.5	3.7$^{\bullet}$	0.7	0.8	0.0	0.0	1.0	2.3^{+}	0.2
15									5.1$^{\bullet}$	2.5^{+}	2.0^{+}	0.5	0.4	0.5	2.3^{+}	0.5
16										6.2$^{\vee}$	6.6$^{\vee}$	3.5$^{\bullet}$	3.5$^{\bullet}$	4.5$^{\bullet}$	3.4$^{\bullet}$	5.2$^{\bullet}$
17											0.1	0.7	0.6	2.3^{+}	4.2$^{\bullet}$	1.3
19												0.7	0.7	2.5^{+}	4.5$^{\bullet}$	1.4
20													0.0	0.9	2.2^{+}	0.2
22														1.0	2.1^{+}	0.2
23															1.8	1.1
24																2.9^{+}
25																

Significance Level

$^{+}$ = $p < 0.1$

$^{\bullet}$ = $p < 0.01$

$^{\vee}$ = $p < 0.001$

Table 7.3a Values of student's *t* test in a pairwise comparison between patients. Data characterised by the number of moves (all), normalised (n_m).

Patient Number	Patient Number															
	4	5	6	7	12	13	14	15	16	17	19	20	22	23	24	25
4		0.9	0.0	2.1^{+}	1.8	0.5	0.9	1.3	4.1$^{\bullet}$	0.1	0.0	0.5	0.7	0.0	0.7	0.3
5			1.7	3.2^{+}	2.8^{+}	0.4	2.2^{+}	7.5$^{\vee}$	7.1$^{\vee}$	1.6	1.6	2.0^{+}	1.5	1.2	2.5^{+}	1.5
6				2.2^{+}	2.0^{+}	0.9	1.0	2.1^{+}	5.1$^{\bullet}$	0.2	0.1	0.6	0.7	0.0	0.9	0.5
7					0.5	2.2^{+}	1.3	0.7	2.5^{+}	2.1^{+}	2.6^{+}	1.4	1.3	2.6^{+}	2.0^{+}	3.1^{+}
12						1.8	1.4	0.9	1.3	1.9	2.3^{+}	1.3	1.4	2.4^{+}	2.0^{+}	2.8^{+}
13							1.4	2.8^{+}	4.5$^{\bullet}$	0.8	0.9	1.1	0.9	0.7	1.5	0.8
14								0.5	3.6$^{\bullet}$	1.1	1.2	0.3	0.1	1.0	0.4	1.7
15									3.5$^{\bullet}$	2.5^{+}	2.4^{+}	0.9	0.4	1.6	1.1	3.5
16										4.8$^{\bullet}$	5.8$^{\vee}$	3.6$^{\bullet}$	3.0^{+}	5.1$^{\vee}$	5.1$^{\vee}$	7.0$^{\vee}$
17											0.1	0.7	0.7	0.2	1.0	0.2
19												0.8	0.9	0.1	1.1	0.4
20													0.2	0.6	0.0	1.2
22														0.8	0.3	1.2
23															0.9	0.5
24																1.6
25																

Significance Level

$^{+}$ = $p<0.1$

$^{\bullet}$ = $p<0.01$

$^{\vee}$ = $p<0.001$

Table 7.3b Values of student's t tests in a pairwise comparison between patients. Data characterised by the number of large movements, normalised ($n_m > T$).

Patient Number	Patient Number															
	4	5	6	7	12	13	14	15	16	17	19	20	22	23	24	25
4		0.4	0.3	2.2^{+}	2.9^{+}	3.0^{+}	3.9$^{\bullet}$	2.9^{+}	1.7	3.6$^{\bullet}$	3.8$^{\bullet}$	2.6^{+}	5.2$^{\vee}$	0.0	1.0	1.0
5			0.2	2.1^{+}	2.0^{+}	1.6	2.6^{+}	1.8	1.4	2.0^{+}	2.5^{+}	1.7	3.2^{+}	0.4	0.2	1.1
6				2.4^{+}	2.6^{+}	2.4^{+}	3.5$^{\bullet}$	2.5^{+}	1.7	2.9^{+}	3.4$^{\bullet}$	2.3^{+}	4.4$^{\bullet}$	0.3	0.6	1.2
7					0.9	1.7	2.3^{+}	1.5	0.2	1.8	2.1^{+}	1.2	2.8^{+}	1.9^{+}	4.0$^{\bullet}$	1.3
12						1.4	1.5	0.9	0.4	1.3	1.2	0.5	1.9^{+}	2.0^{+}	4.3$^{\bullet}$	2.0^{+}
13							0.0	0.4	1.1	1.5	0.4	0.6	0.3	1.9^{+}	3.9$^{\bullet}$	2.5^{+}
14								0.3	1.4	0.3	0.3	0.6	0.2	2.9^{+}	5.4$^{\vee}$	3.2$^{\bullet}$
15									0.9	0.1	0.1	0.2	0.3	2.0^{+}	4.0$^{\bullet}$	2.3^{+}
16										1.1	1.2	0.7	1.8	1.4	2.9^{+}	1.1
17											0.0	0.4	0.2	2.2^{+}	4.5$^{\bullet}$	2.8^{+}
19												0.4	0.3	2.7^{+}	5.3$^{\vee}$	3.1^{+}
20													0.7	1.8	3.7$^{\bullet}$	2.0^{+}
22														3.4$^{\bullet}$	6.8$^{\vee}$	4.1$^{\bullet}$
23															1.0	0.8
24																2.4^{+}
25																

Significance Level

$^{+}$ = $p < 0.1$

$^{\bullet}$ = $p < 0.01$

$^{\vee}$ = $p < 0.001$

Table 7.3c Values of student's t tests in a pairwise comparison between patients.

Data characterised by the number of small movements, normalised ($n_m < T$)

Patient Number	Patient Number															
	4	5	6	7	12	13	14	15	16	17	19	20	22	23	24	25
4		0.8	0.5	1.8	2.1^{+}	0.4	1.8	0.7	1.1	1.2	1.2	2.4^{+}	2.4^{+}	0.3	0.5	0.3
5			1.5	2.2^{+}	2.5^{+}	1.6	1.9	1.1	1.2	2.8^{+}	1.7	4.8$^{\bullet}$	2.7^{+}	1.1	1.9	1.5
6				1.5	1.8	0.0	1.7	0.4	1.1	0.7	0.9	1.8	2.1^{+}	0.2	0.2	1.1
7					0.5	1.0	1.8	0.8	1.5	0.9	0.6	0.3	1.0	2.0^{+}	2.1^{+}	2.4^{+}
12						1.2	1.1	1.0	1.0	1.6	0.9	0.7	0.4	2.4^{+}	2.6^{+}	2.8^{+}
13							1.1	0.2	0.7	0.6	0.6	1.5	1.5	0.1	0.2	1.0
14								1.3	0.5	1.3	1.5	1.1	1.1	2.3^{+}	2.3^{+}	2.3^{+}
15									0.9	0.0	0.3	0.6	1.4	0.6	0.7	1.0
16										0.9	1.1	0.8	1.1	1.5	1.5	1.5
17											0.4	1.0	1.5	0.9	1.3	2.4^{+}
19												0.3	1.4	1.3	1.4	1.7
20													1.0	2.0^{+}	2.9^{+}	4.5$^{\bullet}$
22														2.8^{+}	3.0^{+}	3.1^{+}
23															0.0	0.7
24																1.2
25																

Significance Level

+ = $p<0.1$

• = $p<0.01$

Table 7.3d Values of student's t test in a pairwise comparison between patients. Data characterised by the ratio of large to small moves, non - normalised $\frac{N_m > T}{N_m < T}$.

	Parameter	Grand Mean	Standard Deviation
Non - normalised	N_m	63.0	30.6
	$N_m > T$	33.0	26.8
	$N_m < T$	29.0	17.6
	$\frac{N_m > T}{N_m < T}$	2.8	5.1
Normalised	n_m	133.6	72.1
	$n_m > T$	77.3	66.4
	$n_m < T$	56.3	42.0

Table 7.4 Grand mean of the sample population's mobility, characterised using non-normalised and normalised parameters.

Patient 7

	Night after admission						
	1	2	3	4	5	6	7
$\tilde{i}_s$	26	16	76	49	49	58	22
$\tilde{i}_m$	11	5	11	14	11	5	6
$\tilde{i}_L$	3	6	0	0	1	2	3

Patient 23

	Night after admission					
	1	2	3	4	5	6
$\tilde{i}_s$	36	3	2	2	0	2
$\tilde{i}_m$	9	1	4	4	0	3
$\tilde{i}_L$	3	3	3	3	4	2

Patient 25

	Night after admission				
	1	2	3	4	5
$\tilde{i}_s$	4	10	4	17	14
$\tilde{i}_m$	1	2	6	6	9
$\tilde{i}_L$	4	5	3	2	4

Table 7.5 Values of the parameters $\tilde{i}_s$, $\tilde{i}_m$ and $\tilde{i}_L$ for three of the patients monitored for 5 or more nights.

Patient 7

Night after admission	1	2	3	4	5	6	7
1		3.2	11.7•	5.6+	3.9	9.2+	0.7
2			21.9v	14.6v	11.1•	12.2•	1.8
3					2.3	3.6	9.9•
4					1.3	7.0+	6.3+
5						3.2	3.3
6							5.1+
7							

Patient 23

Night after admission	2	3	4	5	6
1	8.5+	10.6•	10.6•	27.9v	–
2		1.8	1.8	3.6	1.4
3			0.0	5.0+	0.1
4				5.0+	0.1
5					5.2+
6					

Patient 25

Night after admission	1	2	3	4	5
1		0.6	3.1	6.1+	3.9
2			4.6	3.7	3.1
3				4.9	1.6
4					1.5
5					

Significance Level

+ = $p<0.1$

• = $p<0.01$

v = $p<0.001$

Table 7.6 x^2values of a night to night pairwise comparison of mobility characterised by parameters i_s, i_m and i_L. For 3 patients monitored for 5 or more nights.

Parameter	Patient Number												
	4	5	6	7	12	14	15	16	17	19	23	24	25
i_s	50	4	43	296	229	62	55	341	21	8	45	67	49
i_m	13	11	24	13	20	7	10	27	8	5	21	35	25
i_L	10	9	10	15	2	1	7	2	7	9	18	12	18

Table 7.7 Pooled results of characterisation parameters i_s, i_m and i_L, for each patient.

Patient no.	4	5	6	7	12	14	15	16	17	19	23	24	25
4		19.7$^{\vee}$	3.7	28.7$^{\vee}$	34.2$^{\vee}$	10.4$^{\bullet}$	1.2	50.3$^{\vee}$	1.1	9.3$^{\bullet}$	3.7	3.9	3.9
5			13$^{\vee}$	106$^{\vee}$	116$^{\vee}$	47$^{\vee}$	27$^{\vee}$	159$^{\vee}$	10.3$^{\bullet}$	3.5	10.3$^{\bullet}$	17.7$^{\vee}$	10.3$^{\bullet}$
6				66$^{\vee}$	56$^{\vee}$	20$^{\vee}$	7.6^{+}	78$^{\vee}$	1.4	5.3^{+}	2.2	0.3	1.4
7					17$^{\vee}$	3.5	4.4	27$^{\vee}$	17$^{\vee}$	52$^{\vee}$	37$^{\vee}$	20$^{\vee}$	36$^{\vee}$
12						0.5	19$^{\vee}$	0.3	45$^{\vee}$	92$^{\vee}$	71$^{\vee}$	56$^{\vee}$	72$^{\vee}$
14							5.4^{+}	0.7	16$^{\vee}$	32$^{\vee}$	24$^{\vee}$	19$^{\vee}$	24$^{\vee}$
15								30$^{\vee}$	3.9	14.5$^{\vee}$	8.9$^{\bullet}$	7.3^{+}	9.3$^{\bullet}$
16									65$^{\vee}$	134$^{\vee}$	100$^{\vee}$	79$^{\vee}$	101$^{\vee}$
17										3.6	0.2	2.4	0.4
19											3.7	13.1$^{\bullet}$	4.6
23												4.6	0.2
24													3.3

Significance Levels

$^{+}$ = $p<0.1$

$^{\circ}$ = $p<0.01$

$^{\vee}$ = $p<0.001$

Table 7.8 Values of X^2 from a pairwise comparison of the interval between moves, characterised by i_s, i_m and i_L, between each of the patients in the study.

Pooled Results

Sub-group		Number of Moves 1st night	2nd night	3rd night	Total no. nights
Fall		1462	515	1167	9
	Mean	162.4	59.2	129.7	
No Fall		386	737	581	4
	Mean	96.5	184.3	142.3	

Contingency Table

Sub-group	Number of Moves 1st night	2nd night	3rd night
Fall	162.4	59.2	129.7
No Fall	96.5	184.3	142.3

2 degrees of freedom

$\chi^2 = 17.67$

Significant at $p < 0.001$

Table 7.9a Number of moves performed by patients in the "Fall" and "No Fall" subgroups on the first three nights after admission and χ^2 comparison between them. Mobility data characterised by the number of moves (all) normalised (n_m).

Pooled Results

Sub-group		Number of Moves 1st night	2nd night	3rd night	Total no. nights
Fall		909	225	897	10
	Mean	90.9	22.5	89.7	
No Fall		92	281	248	2
	Mean	46.0	140.5	124.0	

Contingency Table

Sub-group	Number of Moves 1st night	2nd night	3rd night
Fall	90.9	22.5	89.7
No Fall	46.0	140.5	124.0

2 degrees of freedom

$\chi^2 = 24.7$

Significant at,

$p < 0.001$

Table 7.9b Number of moves by patients in the "Fall" and "No Fall" subgroups on the first three nights after admission and a χ^2 comparison between them. Mobility data characterised by the number of moves greater than the threshold, normalised $(n_m > T)$.

Norton score grouping	Number of moves				Number of nights
	Parameters				
	n_m	$n_m > T$	$n_m < T$	$\frac{N_m < T}{N_m > T}$	
Stable >12	3912	2439	1479	71.5	28
Stable <12	728	212	516	7.1	9
All stable	4640	-	-	-	37
Improved	3741	-	-	-	23

Table 7.10 Pooled data relating to the mobility of patients grouped according to their Norton score.

a)

n_m	Stable >12	Stable <12	χ^2	Sign. Level
Observed frequency	139.4	80.9		
Expected frequency (fe)	125.4	125.4		
Difference (d)	14.3	44.5		
$d^2/_{fe}$	1.63	15.3	17.4	$p<0.01$

b)

n_m	All Stable	Improved	χ^2	Sign. Level
Observed frequency	125.4	162.7		
Expected frequency (fe)	139.7	139.7		
Difference (d)	14.3	23		
$d^2/_{fe}$	1.46	3.79	5.25	$p<0.05$

Tables 7.11a and b χ^2 comparison of the mobility of subgroups relating to Norton score. Mobility records characterised by number of moves (all), normalised (n_m), a) "Stable >12" and "Stable < 12" and b) "All Stable" and "Improved".

a)

$n_m > T$	Stable >12	Stable <12	χ^2	Sign. Level
Observed freq.	87.1	23.6		
Expected freq.(fe)	71.6	71.6		
Difference (d)	15.5	48.0		
$d^2/_{fe}$	3.4	32.2	35.6	$p<0.001$

b)

$n_m < T$	Stable >12	Stable <12	χ^2	Sign. Level
Observed freq.	52.8	57.3		
Expected freq.(fe)	51.2	51.2		
Difference (d)	1.6	6.1		
$d^2/_{fe}$	0.1	0.7	0.8	N.S.

c)

$\frac{N_m > T}{N_m < T}$	Stable >12	Stable <12	χ^2	Sign. Level
Observed freq.	2.6	0.8		
Expected freq.(fe)	2.1	2.1		
Difference (d)	0.4	1.3		
$d^2/_{fe}$	0.1	0.8	0.9	N.S.

Tables 7.12a,b and c χ^2 comparison of the mobility of "Stable >12" and "Stable <12" subgroups. Mobility records characterised by a) moves larger than the threshold, normalised ($n_m > T$), b) moves smaller than the threshold, normalised ($n_m < T$) and c) value of $N_m > T / N_m < T$.

Parameters		Sedated	Not Sedated	t value	Sign. Level
n_m	Mean	95.3	141.9		
	S.D.	50.1	73.8	2.3	$p < 0.05$
$n_m > T$	Mean	31.9	87.4		
	S.D.	25.1	68.5	3.1	$p < 0.01$
$n_m < T$	Mean	63.3	54.8		
	S.D.	46.6	41.1	0.7	N.S.
$\frac{N_m > T}{N_m < T}$	Mean	1.0	2.8		
	S.D.	3.3	.5.5	1.5	N.S.

Table 7.13 Student's t test comparison between "Sedated" and "Not Sedated" subgroups.

Parameter	Sedated (3)	Not Sedated (11)	χ^2	Sign. Level
i_s	61 (20.3)	1234 (112.2)		
i_m	40 (13.3)	232 (21.1)		
i_L	36 (12)	99 (9)	99	$p<0.001$

Table 7.14 χ^2 comparison of interval between moves (i_s, i_m and i_L) for "Sedated" and "Not Sedated" subgroups.

CHAPTER 8

SUMMARY

Pressure sores, once thought to be a direct consequence of inferior nursing care and neglected by much of the medical profession, have now become a subject of open discussion by clinician, nurse, bioengineer, and industrialist. This increased awareness has resulted in an expansion of resources, ideas and opinions devoted to the topic. However, despite this, much essential research associated with the pathophysiological, psychological and to some extent, economic factors of pressure sores has yet to be undertaken.

Epidemiological studies assessing the prevalence of sores within the hospital and the home communities have established some of the common characteristics of those people who suffer from pressure sores. A high proportion of elderly patients were found to have pressure sores and this group accounted for 66% of all sores recorded. Aetiological factors such as impaired mobility, incontinence, damage to the neurosensory system etc., associated with pressure sore formation were identified in these surveys. Experimental studies on animals have provided data on the tolerance of soft tissue to pressure applied with a range of amplitudes and duration. Evidence supplied by these different investigations repeatedly emphasised the importance of mobility in the aetiology of pressure sores. Many studies have generated quantitative data relating to some of the factors thought to be associated with pressure sores. Those of Bardsley (1977) and Ferguson-Pell (1977) have produced baseline measurements of frequency and size of body movements and variations in body support surface interface pressure during sleep, for healthy young subjects. The study reported in this thesis provides an extension to their work comparing overnight mobility of elderly patients in the clinical environment.

Althought it is generally recognised that there is an association between movement and changes in interface pressure, there is no substantiating quantitative information. An investigation was conducted to establish whether such an association was present, by simultaneously measuring interface pressure and mobility during sleep using the experimental methods of Bardsley (1977) and Ferguson-Pell (1977). The interface pressure monitoring procedure required that transducers be attached to the body and precluded the use of elderly

patients as subjects and as a result unlike the rest of this study, was conducted on normal young subjects.

The minimum resolution of the interface-pressure monitoring system was established (4kPa) and all of the measured changes greater than this threshold were noted. To establish a comparable parameter for the mobility, a range of thresholds were employed. Thus, those moves greater than a threshold were compared to interface pressure changes greater than the minimum resolution of the system. Nine subjects were monitored and comparison between the two sets of data , for each of the subjects, indicated that changes in interface pressure were closely related to the occurrence of moves larger than an optimum threshold level. Statistical analysis of the data showed a highly significant relationship between the two variables. Changes in the position of a subject's centre of gravity greater than 10 mm, were associated with significant changes in interface pressure.

One of the reasons for measuring mobility rather than interface pressure in the clinic, is that current instrumentation systems available for monitoring pressure have a number of limitations. They require the attachment of transducers to the body and are associated with bulky recording apparatus, interfering with normal nursing and clinical treatment of the patient. Therefore, although it is feasible to monitor interface pressures in the confines of a sleep laboratory, it is impractical in the clinical environment. However, the study with normal volunteers showed a close correlation between interface pressure and movement. Therefore, by measuring a subject's mobility it is possible to obtain indirectly, information on periods of prolonged pressure, relief of tissues etc. Similar studies measuring the relationship between movement and pressure relief in the elderly would be valuable in confirming the validity of extrapolating results obtained from the young to the elderly. However, before this can be conducted the limitations in currently available pressure monitoring equipment must be overcome.

In order to study the size and frequency of body movements during sleep in elderly patients in hospital, a suitable monitoring apparatus was developed. On reviewing the literature it was apparent that a number of

different principles have previously been employed in the measurement of overnight mobility. The most popular of these was derived from measuring the displacement of all, or part of the bed, usually the mattress. Other methods utilised ultrasonics, direct observation of the subject by cine camera or television camera, and measurement of mechanical forces generated in, or by, the bed structure. However, the constraints of this study, where the system was required to be compatible with use in the clinical environment, inconspicuous, accurate and fitted to any bed in a ward, were not fully satisfied by any of the established designs. A monitoring system was therefore designed to meet these specifications. The technique of monitoring the reaction force between the floor and the bedlegs, as outlined by Fernie (1973) and Bardsley (1977) was employed. The system developed was in three sections; transducing elements, monitoring the reaction force under each of the bedlegs; electronic processing, combining the outputs from the transducers into a form representative of a movement by the patient in the bed; a means of recording the data, that permitted transfer of the results from the hospital to the laboratory.

Force transducers available commercially were reviewed, but none met all the design requirements such as low profile, electrical output,capable of coupling to a bedwheel etc. A suitable device based on an appropriately strain gauged cantilevered plate was therefore developed for this study. The design consisted of two instrumented cantilever sections, separated by a loading surface. The force transducer was milled out of a steel plate and two aluminium support surfaces were attached to its underside. Evaluation of the device suggested a typical output sensitivity of 0.425μV/V/N, excellent linearity, low hysteresis with a satisfactory rejection of artefacts arising from off-centre loading and cross-effects.

The processing electronics acted as a dedicated single program computer. Strain gauge amplifiers were employed to amplify the signal produced by the force transducers, to one of a suitable magnitude for analysis in further circuitry. Initial designs utilised an SGA 100 amplifier (CIL Electronics, Worthing, U.K.), which were prone to failure after an extended

period of operation and as a result were replaced. The second design employed chopper amplifiers in its first stage, providing high input impedance with low thermal drift. Following stages further amplified the signal and filtered the output. Evaluation of the device showed it to be capable of a gain of over 1000 with satisfactory noise and drift specifications. From the results of the earlier investigation into interface-pressure and overnight mobility, it had been established that changes in signal level characterised the patient's mobility most reliably. Therefore, the electronic processing circuitry was designed to reject high frequency transient signals and to analyse only low frequency changes in signal level. In this way, and by combining the outputs from three of the force transducers, the magnitude of changes in position of the centre of gravity of the patient in the bed was calculated.

One of the major drawbacks of most previous systems has been the dependency of the system calibration upon the patient's weight. However, in this device use of a fourth transducer allowed the weight of the patient to be calculated automatically and the output of the system to be independent of the patient's body weight. Data recording was accomplished utilising an analogue reel to reel recorder with a slow recording speed, fast replay, low noise and a high signal to noise ratio. The device was portable and was transferred between the hospital and the laboratory. Evaluation of the completed monitoring system incorporating force transducers, processing instrumentation and tape recorder, suggested an overall system error of $\pm$ 1.6%. Within this range the system was determined to be independent of the patient's weight (>30kg). This novel feature greatly simplified analysis and interpretation of the results. The apparatus was capable of measuring moves up to a size of 0.27m with an output sensitivity of 14mV/mm.

Data produced by the monitoring apparatus were stored on tape and replayed at approximately 60 times real time into a PDP12 minicomputer, where it was digitised and then stored. To reduce the volume of data, the recorded signal was edited, so that those periods when the patient had been lying immobile were discarded. The edited data, containing information associated with movements only, was later transferred to punched tape and

read into an ICL 1904S mainframe computer for further analysis. Periods of the recording at the beginning and end of the night were discounted as the patient might have been awake. Moves occurring within a minute of one another were grouped together and considered as a single "move complex". Parameters were established to characterise the mobility records. The simplest of these was the number of moves that occurred during the night. A threshold move size (10 mm) established earlier, was used to delineate between "large" and "small" moves. In this way further movement "magnitude" parameters were described including, number of "large" moves, number of "small" moves and the ratio of "large" to "small" moves. Normalisation of these parameters was undertaken. The interval between moves was also investigated and associated parameters were developed.

Results of a study involving 15 elderly patients, indicated that all of the selected parameters were capable of characterising the data and were found to be statistically sensitive to variations in mobility between subjects. However, those related to the interval between moves were found to have a significant variation from night to night within patients, precluding their use in comparisons between patients. The ease of assessment of the number of moves suggests its suitability as a method for characterising the mobility records. Investigation into the distribution of movements within 10 minute and 30 minute intervals indicated that in the majority of cases there was no regular periodic trend, nor was there a consistent change in frequency of moves during the night. This observation is contrary to that obtained in studies on movement of normal subjects in a controlled environment (Bardsley, 1977). This disparity may be attributable to the effect of the hospital environment disturbing normal patterns of mobility or to differences in sleep patterns between the elderly and the young. Examination of the night to night variation in mobility for each of the subjects, demonstrated a marked variation between the first two nights, in the majority of cases. Typically on the first night after admission, the patient performed a large number of moves, while on the second night, her mobility was much reduced. This trend was proven to be statistically significant and was attributable to the

effect of changing environment linked to anxiety at being in hospital leading to a restless first night. However, the result on the second night suggests that compensation occurs with a concomitant reduction in mobility.

Patient data information was compared to level of mobility, characterised by both the total number of moves and the number of large moves. It was observed that those patients regularly receiving sedatives, all had mobility below the sample population's average. Statistical analysis indicated that there was a significant difference between the sedated and not sedated patients' mobility. This was most marked in the number of large moves these patients made.

Comparison between the Norton score assessed on each night of the monitoring session and the patient's mobility suggested that for the sample of patients in this study at least, mobility is significantly related to the Norton score.

The results presented in this thesis, indicating the relationship between interface pressure and mobility and from monitoring the mobility of elderly patients, have shown that it is possible to measure and characterise body movements during sleep in a clinical environment, using simple parameters, (number of moves, large moves etc.), and that there are clinical factors associated with consistently low mobility.

On the basis of these results it is suggested that further investigation in this area is justified. However, only by obtaining more data, providing information on the mobility of a larger population of patients, identifying clinical and diagnostic factors associated with abnormal levels of mobility and a retrospective study to determine the longer term relationship between mobility and pressure sore formation, is it possible to ascertain the clinical value and the limitations of studying the mobility of a patient. To this end the Scottish Home and Health Department (S.H.H.D.) have funded a research project to provide a detailed characterisation of the overnight mobility of elderly patients. This award was based upon preliminary results obtained from the mobility monitoring apparatus described in this thesis.

For this work the research personnel associated with the project have

designed a second-generation monitoring system. This retains the force transducers underneath each of the bedlegs. However, signal processing is achieved with the use of software rather than hardware and has employed a dedicated microcomputer for data acquisition, analysis and storage. Thus, the manual editing procedure has been automated, giving greater accuracy.

In addition it is hoped to use the microcomputer to conduct the refined signal analysis previously undertaken by the ICL1904S mainframe computer. Software has been developed which allows two beds to be monitored simultaneously, providing a marked increase in the rate of data collection. The specifications established by the prototype analogue device, described in this thesis, was used extensively in developing processing software. At the time of writing this device is being employed regularly in the clinical environment.

In the long term, if the data obtained from the current research programme confirms the indications reported in this thesis, it is possible that a "clinical tool" might be developed. Conceptually a device might be placed beside the bed and although the contents of the device would be relatively complex, requiring solid state memory and a microprocessor, its readout could be simply an indication of whether the patient's mobility is high, average or low.

APPENDIX 1

DETAILS OF THE ANALOGUE PROCESSING SYSTEM

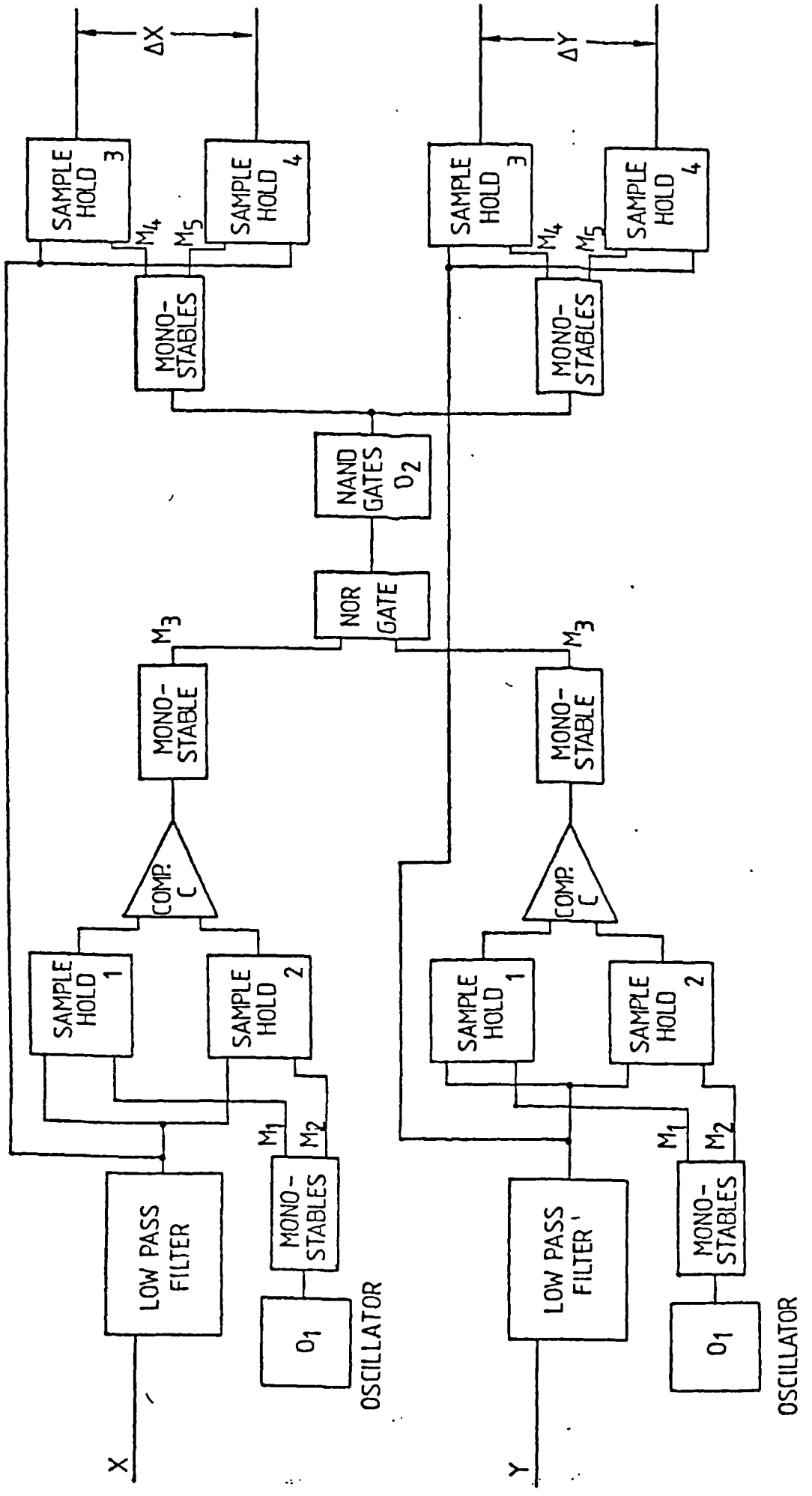


Figure A1.1 Detailed schematic diagram of circuit B

A1.1 CIRCUIT B, DETAILS

A1.1.1 Filtering of the signal

This circuit was designed to calculate the d.c. changes in signal level, while disregarding the a.c. transient component (vide 5.8.2). Figure A1.1 is a detailed schematic diagram indicating the elements comprising the circuit.

To improve the signal to noise ratio, the incoming signal from circuit A, was filtered. The major component of the noise was attributable to mains disturbance with a characteristic 50Hz sinusoidal waveform. It had been previously specified that only the d.c. component of the signal was of importance. Therefore a filter was designed capable of attenuating all signals with a frequency greater than approximately 15Hz while passing lower frequencies unimpaired.

Electronic filters may commonly be classified into four groups.

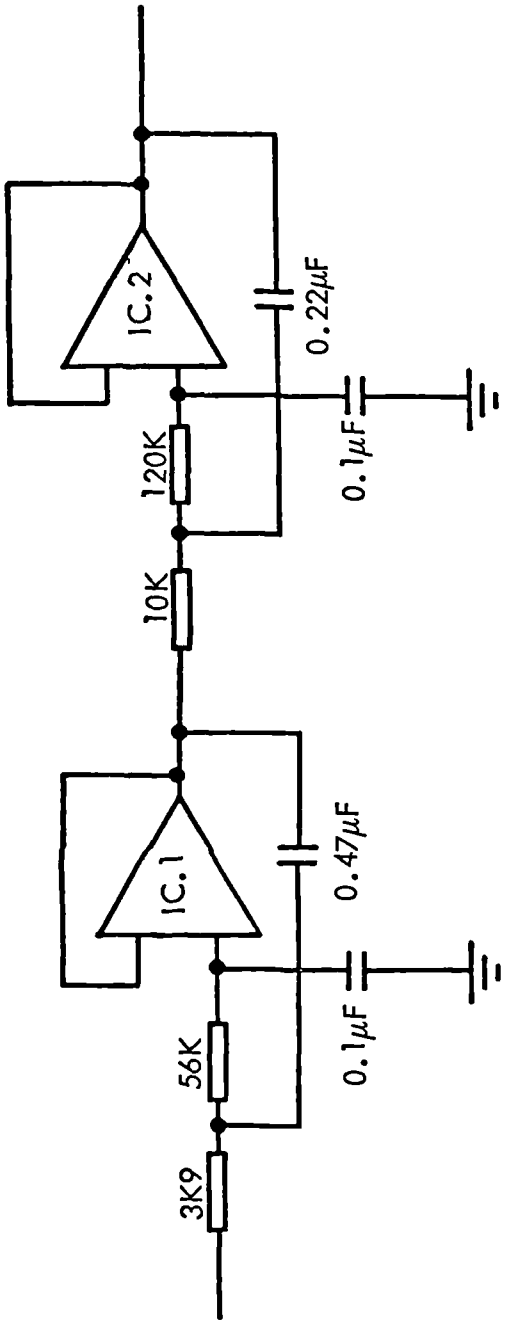
High-pass: Filters of this type attenuate the lower frequencies, while "passing" the higher frequency signals unchanged.

Low-pass: In contrast to high-pass filters, the upper frequencies are attenuated and the lower frequency signal is unchanged.

Band-pass: This type of filter attenuates both upper and lower frequencies, promoting a "band" of frequencies between the two.

Notch filter: This type of filter is applied in situations where a specific frequency is to be attenuated and is the reverse of the band pass device and may be termed "band reject".

With a desired frequency range of d.c. to 15Hz, a low-pass filter was selected for this application. The detailed design of the filter was achieved by the use of a Honeywell 6060 computer and associated filter design package (Geckle, 1973). This program calculated the critical component values for a Butterworth type low-pass filter, based upon a set of parameters describing the required filter characteristics. Thus, information was input to the computer specifying the number of stages, the gain and the cut-off frequency of the filter. The latter is normally defined as that frequency at which the signal has been attenuated by three decibels, where



IC.1 = IC.2 = 741

Figure A1.2 Butterworth low-pass filter, with 3dB attenuation at 14 Hz.

a decibel (dB) is specified as $20 \log_{10} V_o/V_i$ (V_o = output voltage, V_i = input voltage). Thus, for -3dB, $V_o/V_i = 0.707$, an attenuation of the input signal by a factor of 0.707. With this information, the program calculates ideal values for the components of the filter. Often these "theoretical"values do not correspond to standard values, commercially available. Thus, the program was capable of recalculating resistance values, on the basis of adjusted capacitance values. Details of the final design of the filter are described in figure A1.2.

The characteristics of the filter were assessed over a frequency range of d.c. to 60Hz by means of a transfer function analyser (T.F.A.) JM 1600 (Solarton, Hampshire, U.K.). Measurements with this instrument were made by generating a sinusoidal signal as an input to the filter and analysing the output. The instrument consisted of a frequency generator to provide the signal and a "correlator" to measure the response. Results were displayed in three ways, based upon differing coordinate systems, namely, Cartesian, Polar and Logarithmic Polar. The "correlator" converted the analogue output of the filter into digital form and compared it with the input signal provided by the frequency generator. In this manner, the attenuation and phase shift of the filter was determined.

Figure A1.3 provides a graphical representation of the filter output in decibels, against log frequency. From the graph it is evident that attenuation of the input signal by three decibels occurs at a frequency of approximately 15Hz. This was considered as the cut-off frequency. The response curve may be described in the form of two asymptotes (figure A1.3). One of which represents the filter output at low frequencies, the other is an approximation of the output at higher frequencies. Their point of intersection corresponds to the cut-off frequency of 15Hz. The gradient of the high frequency asymptote is known as the "roll-off" characteristic of a filter and is related to the attenuation of the frequencies greater than the cut-off frequency. For this filter the "roll-off" is calculated to be 9.72 dB/octave, where an octave was defined as a doubling of frequency.

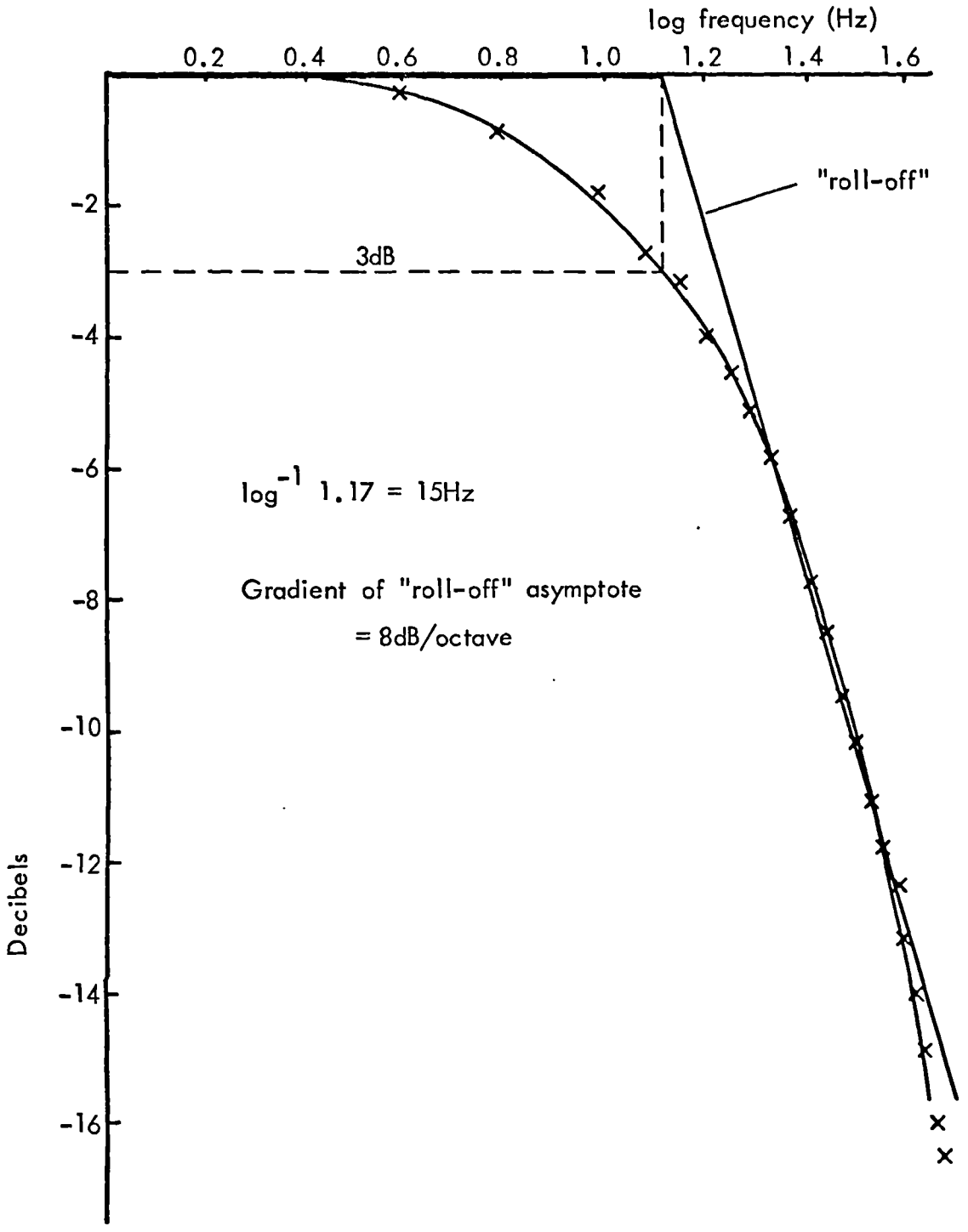


Figure A1.3 Output of the Butterworth low-pass filter utilised in circuit B, indicating a cut-off frequency of 15Hz.

A1.1.2 Signal Processing

The second stage following the filter was a pair of monolithic sample and hold circuits $(S + H)_1$, $(S + H)_2$ (figure A1.1). Characteristically these devices sampled an analogue input voltage and stored (or "held") the sampled signal size. The rate of sampling was controlled by external logic pulses and the duration of signal storing was dependent upon the size of an external capacitor. Selection of a suitable capacitor determined the hold step, acquisition time and droop rate of the device's store facility.

Acquisition time is a measure of the duration necessary for the device to store 100% of the sampled signal. Droop rate refers to the ability of the circuit to maintain the stored voltage level and is usually described in terms of μV/second. A significant error may be provided by dielectric absorption in the hold capacitor. Thus capacitors constructed of materials which suffer from high hysteresis such as ceramics, were not suitable for this application, and a 1 μF polyester capacitor was selected. Under test conditions, this capacitor produced a hold step of 0.01 mV, an acquisition time of approximately 1 ms and a droop rate of better than 8.3μV/s. The logic pulses necessary to activate the sample and hold circuitry were produced by the combination of an oscillator (O_1) and a monostable multivibrator. An oscillator was constructed, based on an RS555 integrated circuit timer (R.S. Components Ltd, London, U.K.), operating in astable mode. By selection of suitable external components, the output of the timer circuit was adjusted to give a regular square wave, with a frequency of 8.3kHz and a pulse width of 60ms.

A monostable multivibrator may be described as a "delay circuit", the output of which stays at zero until there is a sudden change in input voltage. The device then produces a concomitant voltage output for a pre-determined length of time, after which it returns to zero. For this application a dual package RS 4258B monostable was selected. The monostable circuitry was designed such that the device gave a complementary output pulse upon application of a logical 1 to the input. The output pulse width was determined by an impedance timing circuit and was essentially independent of the input pulse width. Each unit had two inputs, one operated by positive-going

voltage changes (non-inverting) and the other by negative-going signals (inverting). In a similar manner, there were two outputs of complementary logic levels. It was essential to the principle of the circuit, that the sample and hold devices $(S + H)_1$ and $(S + H)_2$ did not sample the analogue input signal simultaneously. Both of these devices were associated with a monostable multivibrator (M_1, M_2). By using the signal produced by the oscillator, and selection of differing input ports, the output of the two monostables were designed to be equal in magnitude and out of phase. The output waveform from the oscillator was input to both monostables. However, the inverting input was selected in one case and the non-inverting input in the other. The operation "logic table" as described below,

Input A	Input B	Reset C_D	Output Q
x	x	0	1
0	↓	1	⊓
↑	1	1	⊓

indicates that the monostable inputs to the non-inverting terminal (A) will produce pulses at Q for positive-going signals only. Conversely, inputs to the inverting terminal (B) produce complementary output pulses for negative-going signals only. Thus, the positive-going slope of the oscillator waveform produces an output pulse from one of the pair of monostables, in turn activating one of the sample and hold units. In a similar manner, the second monostable was activated by the negative-going slope of the oscillator signal. Thus, both sample and hold units were provided with an asynchronous logic input, the interval between sampling being governed by the oscillator frequency.

The output from the two sample and hold devices formed a differential input to a comparator (C), in the form of a 741 operational amplifier. Originally the comparator was connected in "open loop" configuration. In this mode, the maximum potential gain of the device was utilised (approximately 50,000).

If the subject lay immobile in bed, then the signal from the force

transducers was constant and the comparator output was zero. When a centre of gravity movement occurred, there was a change in signal level, this being transmitted to the comparator (by means of the sample and hold circuitry). However, a comparator output gain of 50,000 implied that any differential signal input larger than 0.2mV would result in an output of 10V. This may be considered to represent a logic 1 to the monostable multivibrator (M_3) which is the next element of the circuit (figure A1.1). However, under these conditions the comparator's "open loop" mode of operation proved to be unsuitable, producing output pulses associated with signal noise. To reduce the gain, a feedback resistance was added (gain $\approx 1.4 \times 10^4$). Other than the gain, the comparator output and frequency resolution was dependent upon the magnitude of the differential signal and the sampling interval between the aphasic sample and hold devices $(S + H)_1$, $(S + H)_2$.

For example, consider the output of the comparator C to be

$$E_o = (E_1 - E_2).A$$

where E_o = output voltage

E_1, E_2 = differential input voltages

A = circuit gain $\approx 1.4 \times 10^4$

Selecting E_o = 10V as a maximum output value

$$E_1 - E_2 = \frac{10}{1.4 \times 10^4} = 0.7\text{mV}$$

Thus, the minimum differential input voltage to produce an output of 10V was 0.7mV. As a result, the signal noise must be significantly lower than this to prevent artefacts. Signal noise after the filter (determined above) was approximately 0.1mV (peak to peak) and was therefore at a satisfactory level for further processing, involving the sample and hold devices and the comparator.

The frequency resolution may be described as,

$$\frac{\text{Minimum differential signal level}}{\text{Sampling interval}}$$

The sampling interval of the sample and hold devices was 60 ms and the minimum differential signal level was 0.7mV. Thus, frequency resolution

$$= \frac{0.7 \times 10^{-3}}{60 \times 10^{-3}} = 0.012 \text{ Hz}$$

The frequency resolution and the minimum differential input voltage provide critical threshold levels defining the lower limits of the input signal, and thus of the overall system.

During the course of a move there were often brief moments at which the signal would fall outside the described threshold levels. When this occurred the comparator output would fall to zero. To prevent artefacts arising from this situation, the monostable multivibrator (M_3) was adjusted so that its output pulse width was longer in duration than any such fall in signal.

In earlier designs monostable M_3 was followed by three further stages, culminating in another pair of sample and hold devices, $(S + H)_3$ and $(S + H)_4$. The first of these stages consisted of an oscillator (O_2) in the form of three NAND logic gates, connected together with external passive components, producing a nominal square wave output. This provided an input to dual monostables (M_4 and M_5) in a manner similar to that of oscillator O_1, described previously. That is logic pulses were provided for the two sample and hold devices by means of the monostables triggering them asynchronously, dependent upon the oscillator waveform.

Selection of an oscillator (O_2) based on a combination of NAND gates meant that an output signal was applied to monostables M_4 and M_5 simultaneously, with a change in logic level of monostable M_2. The "truth table" for a NAND gate is reproduced below,

Inputs		Outputs
0	0	1
1	0	1
0	1	1
1	1	0

Considering one of its input to be permanently high, if the other input was low, then the combination of NAND gates produced a nominal square wave output. However, if the input was high, the oscillator output was zero, disabling the sample and hold devices. Monostables M_4 and M_5 were identical to M_1 and M_2 in both type and mode of operation. In a similar

manner the pair of sample and hold devices $(S + H)_3$ and $(S + H)_4$ were identical to $(S + H)_1$ and $(S + H)_2$.

The output of circuit B represented $(\Delta (Vs_1 + Vs_2)).Vx$ and $(\Delta (Vs_2 + Vs_3)).Vy$. At a later stage in the analogue processing it was necessary to sum the square of these two values (vide 5.8.3). Unfortunately, although the use of NAND logic gates had provided synchronisation between gates switching and signal sampling, there was no temporal synchronisation between the two channels. This difference resulted from a phase variation between the oscillator (O_2) of each channel. This problem was solved by using a single oscillator (O_2) for both channels, thereby eradicating any possibility of errors, due to phase differences between the timing circuitry for the final pair of sample and hold devices. For this reason, it was necessary to use a NOR gate, to provide a logic control for the oscillator. The input to the NOR gate consisted of the output of monostable M_3 of both channels (figure A1.1). The truth table for a NOR gate as described below,

Inputs		Outputs
0	0	1
1	0	0
0	1	0
1	1	0

shows that only when both inputs are at a logic "0", will the device produce a logic 1 output. Thus, should a movement occur which produces a change in logic level of monostable M_3 of one channel only, the oscillator will be disabled. Only when the analogue signal level is steady and monostables M_3, for both channels are at zero, will the sample and hold devices be activated.

A1.2 ANALOGUE MULTIPLIER

This type of device produces an output voltage or current that is proportional to the product of two or more independent voltages or currents (Sheingold, 1974). The transfer function of such a device may be described as:

$$E_0 = \frac{V_x V_y}{V_r} = K V_x V_y \qquad \text{(A1-1)}$$

where V_x, V_y = input voltages

V_r = reference voltage

K = constant = $\frac{1}{V_r}$

A1.2.1 Error analysis

Equation A1-1 is the ideal output of a multiplier, however, in reality this is modified to:

$$E_0 = K V_x V_y \pm \epsilon (V_x V_y) \qquad \text{(A1-2)}$$

where ϵ is the output voltage error, associated with the input signals V_x and V_y.

There are four major sources of static, or d.c. errors in an analogue multiplier, which may be represented as:

- Input offsets X_{os}, Y_{os}
- Output offsets Z_{os}
- Scale factor K
- Non-linearity f (X, Y)

The transfer function of equation A1-2 may be more accurately rewritten as:

$$E_0 = (K + \Delta K)(V_x + X_{os})(V_y + Y_{os}) + Z_{os} + f(X,Y) \qquad \text{(A1-3)}$$

The product of ΔK with another error term results in a second order term which is regarded as negligible. Thus, equation A1-3 may be rewritten as:

$$E_0 = (K + K)(V_x V_y \quad V_x Y_{os} + V_y X_{os} + Z_{os} + f(X,Y))$$

Considering separate terms:

$K V_x V_y$ represents the ideal output, which tends to zero for V_x V_y tending to zero.

$\Delta K V_x V_y$ is termed a "scale factor error". As previously, the function tends to zero for V_x or V_y tending to zero.

$K V_x Y_{os}$ is known as "linear X feedthrough", and is due to a Y input d.c. offset and is proportional to the magnitude of V_x.

$K V_y X_{os}$ is termed the "linear Y feedthrough" error, which in a comparable manner to the X feedthrough, is due to the X input d.c. offset and is proportional to the size of V_y.

$K X_{os} Y_{os}$ is known as the "output offset" and is independent of the input signal voltages.

$K Z_{os}$ is another output offset, which is also independent of the input signal voltages.

$K f(X,Y)$ is a term resulting from the inherent non-linearity of the device, the magnitude of which is dependent upon both V_x and V_y.

Thus, the errors may be summarised as X and Y input offsets (feedthrough), output offset, scale factor error and non-linearity.

To produce optimum results, the majority of these errors (excluding non-linearity) may be reduced to zero by the provision of equal and opposite offsets and precise adjustment of the scale factor (K). However, the non-linearity inherent to the device is irreducible and provides the critical criteria for its minimum error specification.

The magnitude of the output error of the multiplier is also dependent upon the mode of operation. For example, if the device is connected in

squaring mode, then the accuracy is typically a factor of two better than in the multiplying mode (Analog Devices Inc.,U.S.A.). Conversely, as a square rooter the device is less accurate and due to the circuit principle, often will have a seriously restricted input voltage range.

A1.3 ANALOGUE DIVIDER

Selection of the device to perform the division function was based primarily on the required accuracy, bandwidth and cost.

Since division is the most difficult of the four mathematical functions to accomplish with analogue computing devices, high accuracy implies an associated high level of cost and therefore optimising features of accuracy against cost proved to be particularly relevant. This limitation in accuracy of the analogue divider is due to the inherent nature of division. As the ratio between numerator and denominator reaches limits, that is either one of them approaches zero, the magnitude of their result is either very large, as in the case of the denominator tending to zero, or is very small for the numerator, tending to zero. Thus, ideally, the device must be capable of supplying both infinite gain and infinite dynamic range. In reality, the magnification of drift and noise at low denominator voltages, limits the "ideal" characteristics. Using an analysis similar to that of section A1.2.1, it was possible to categorise some of the errors associated with the analogue division process.

A1.3.1 *Error analysis*

Consider the ideal transfer function to be:

$$E_o = E_{ref} \frac{N}{D}$$

where E_o = output voltage

E_{ref} = reference voltage, often internally scaled to $^{+}$ 10V

N = numerator voltage

D = denominator voltage

Realistically,

$$E_o = (K + \Delta K)\frac{(N + Z_{os})}{D + X_{os}} + E_{os} + f(N, D) \qquad (A1\text{-}4)$$

where K = scale factor (E_{ref})

X_{os} = denominator offset

Z_{os} = numerator offset

E_{os} = output offset

$f(N, D)$ = non-linearity

Rewriting equation A1-4:

$$E_o = (K + \Delta K)\frac{N}{D + X_{os}} + \frac{(K + \Delta K)Z_{os}}{D + X_{os}} + E_{os} + f(N, D) \qquad (A1\text{-}5)$$

Considering each term in equation A1-5 separately,

$$\underline{(K + \Delta K)\frac{N}{D + X_{os}}}$$

may be termed the "scale factor error", resulting from ΔK.

This error is independent of the numerator and denominator voltages. However, an error may also exist as a result of the denominator offset X_{os}. For an arbitrary offset X_{os}, as the denominator (D) approaches a value corresponding to $-X_{os}$, the output of the device will tend to infinity.

$$\underline{(K + \Delta K)\frac{Z_{os}}{D + X_{os}} \approx \frac{K.\,Z_{os}}{D + X_{os}}}$$

assuming second order terms involving K are negligible. This is the "input offset error", which is influenced by the denominator voltage, but not the numerator voltage. As earlier, difficulties occur as values or a combination of values tend to zero. Consider X_{os} as being zero, then the input offset error will tend to infinity as the denominator D tends to zero. For all cases the input offset voltage Z_{os} will be amplified if the denominator voltage (D) is less then the reference voltage (K). Should X_{os} not be zero, then the term will tend to infinity as D tends to $-X_{os}$. To optimise the accuracy of the device, offsets Z_{os} and X_{os} should be zeroed.

$\underline{E_{os}}$, the output stage offset is independent of the numerator and denominator voltages and is not subject to any gain (K). Thus, this term is usually small and can be neglected, although for some strict applications it

should be zeroed.

f(N, D), is related to the output non-linearity and as with the multiplier (vide A1.2.1), is an inherent feature of the design and unlike the other terms irreducible by external trimming.

A1.3.2 Choice of divider

Having defined the possible sources of error in an analogue divider, it was necessary to choose the optimum device for this application. There are three common designs of circuit available for this purpose.

Inverted multiplier: This design is the most common and encompasses a standard analogue multiplier unit. The performance of this type of circuit, particularly with relation to the dynamic range and overall error, is highly dependent upon the specification of the original multiplier unit. For example, a multiplier with an overall error of $\pm$ 1% will have appreciable offsets and non-linearity. In division mode such a multiplier will have a dynamic range of only 20 dB with an overall error of $\pm$ 2.5% at the lower values of denominator voltage. Conversely a multiplier with a specified total error of $\pm$ 0.1% (multiplication mode) will increase this dynamic range to 40 dB and reduce the error to $\pm$ 1% (at the lowest range of denominator value). The AD534K multiplier used in circuit C (vide 5.8.3) was specified as $\pm$ 1% error over a 40 dB dynamic range, in division mode.

Log-antilog: A divider designed using this principle calculates the ratio between variables by subtracting logarithms representing their value,

$$\frac{N}{D} = \epsilon^{(\ln N - \ln D)}$$

The accuracy of these devices is excellent and they are capable of operation over a large dynamic range (several decades). However, log-antilog dividers operate only in a single quandrant and are thus suitable only for comparing positive signals.

Direct or variable transconductance: Dividers utilising this principle are capable of operating in two quadrants, with an error of better than $\pm$ 0.5% over a dynamic range of 40 dB. As with the log-antilog devices,

this is a "dedicated" circuit, capable of division only.

Thus, the inverted multiplier offers the advantage of availability, and flexibility (division being only one of several modes of the basic multiplier unit) with a relatively low associated cost. The "dedicated" devices offer increased accuracy over a wider dynamic range, with limitations in signal polarity. For this application, the numerator and denominator voltages were both positive and therefore the more accurate log-antilog type divider was selected. This was in the form of a 4291H device (Burr-Brown, U.S.A.) which had an overall error of $\pm$ 0.5% over a dynamic range of 40 dB.

A1.4 ELECTRICAL SPECIFICATION OF THE POWER SUPPLY

Type PC500C 15/15 (ITT, Harlow, U.K.)

Output voltage	$\pm$ 12 - 15V
Output current	500 mA
Line stability	500μV/V + 200μV
Load regulation	2mV/V max., 400μV/V typical
Ripple and noise	10mV peak to peak
Input voltage	198 - 264V, 48 - 65 Hz
Operating temperature range	-10°C to 55°C
Temperature coefficient	0.015% /°C max.
Output impedance	Less than 250mΩ , d.c. to 250kHz

APPENDIX 2

CALIBRATION OF THE MOVEMENT MONITORING SYSTEM

The performance of the analogue processing system, in combination with the force transducers, was assessed with regard to changes in load position with a range of different "subject" weights.

A2.1 METHOD

The force transducers were placed underneath a bed similar to those in the hospital ward in which the apparatus was to be used. All the bed clothing and the mattress were removed. To provide a spatial reference a 0.1 metre grid was marked on the bed, emanating from the centre and outlining the X and Y axes (figure 5.1). A selection of accurate 5 kg weights were used to represent the subject's weight, S (vide 5.3.1). The output from the system was monitored by means of a Servoscribe potentiometric chart recorder (RE541.20), adjusted to 5V full scale deflection.

With no weights on the bed (S = 0), the output of circuit D was nulled, that is, the weight of the bed was negated. The system was left to equilibriate and its output monitored to assess drift. A 40 kg load (consisting of 8 of the 5 kg discrete weights) was then applied to the centre of the bed, the position of which was defined as the intersection of the two axes of the grid. The output from the processing instrumentation was zeroed, to permit measurement of the maximum move size. The load was then moved over the bed surface, firstly along the X axis and secondly, along the Y axis. The magnitude of move extended from 10 mm to 300 mm. This procedure was repeated using a range of different loads (40 kg, 30 kg, 20 kg and 10 kg), for move sizes ranging from 75 mm to 300 mm.

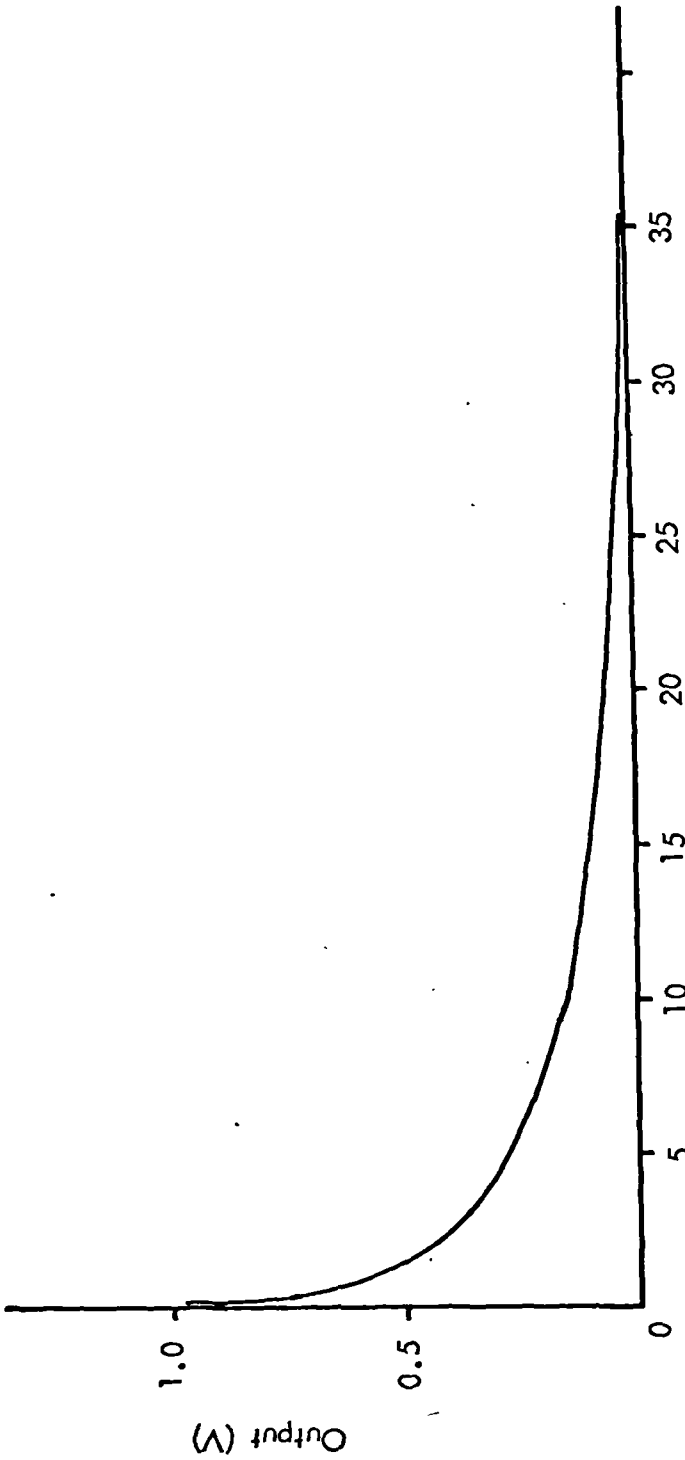


Figure A2.1 Drift associated with the "warm-up" of the monitoring system - equilibrium achieved after approximately 35 minutes.

A2.2 RESULTS

A2.2.1 Drift

The "warm-up" time (period of drift before equilibrium) was considered to be the time interval between switching on the device and it producing a consistent output for a period of at least ten minutes. Figure A2.1 indicates a typical response, with a rapid offset drift in the first 2 - 3 minutes, followed by a slower component. Equilibrium was established after approximately thirty five minutes. The biphasic nature of the equilibrium curve was considered to be the combination of two factors. Firstly, it was necessary for the strain gauge bridge and amplification to warm up, this represented the rapid change in output over the initial five minutes. Secondly, due to heat generated principally by the power supply, a finite time was required to achieve thermal equilibrium.

A2.2.2 Moves along the X and Y axes

Figures A2.2a and b describe the output from the processing electronics for moves along the X axis and Y axis respectively, in both "positive" and "negative" directions. Linear regression of the data suggested a highly linear relationship between move size on the bed and output from the system ($s = 0.99$). System sensitivity, calculated on the basis of the results presented in figures A2.2 a and b, was 14mV/mm and the maximum measurable move was estimated to be 0.27m.

Figure A2.3 is a comparison of the two curves illustrated in figures A2.2 a and b. For low move sizes the output from the electronics was the same for moves along both the X and Y axes. However, as the move size increased a variation between outputs was evident. From the regression calculated previously, this variation, calculated as the difference between gradients, was established to be 2%. This error was attributable to inaccuracies regarding the analogue representation of the bed's geometry.

A2.2.3 Effect of varying load on system output

The mobility monitoring system was designed to be independent of the

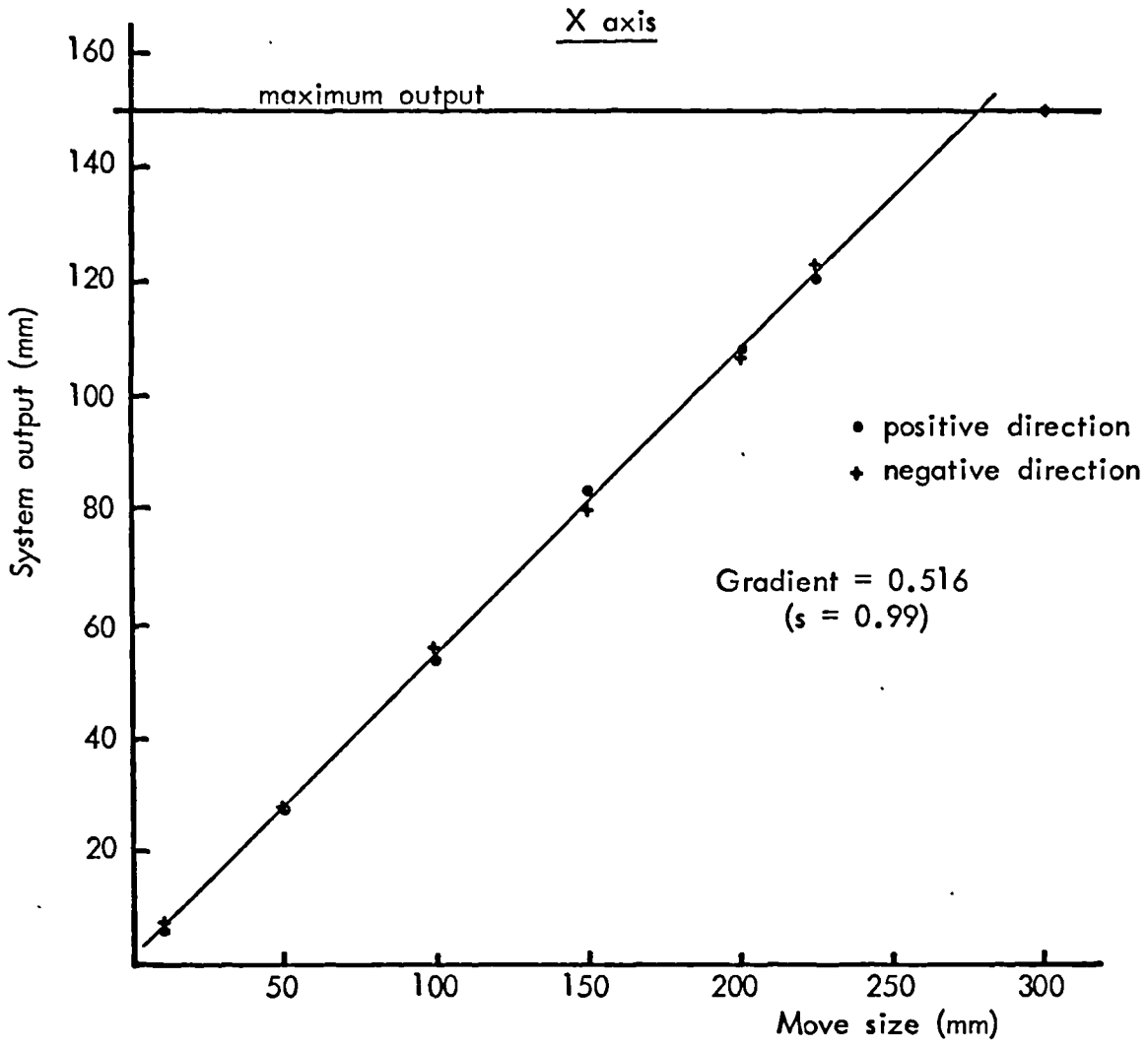


Figure A2.2a Mobility monitoring system calibration - output versus move size for moves along the X axis in both positive and negative directions.

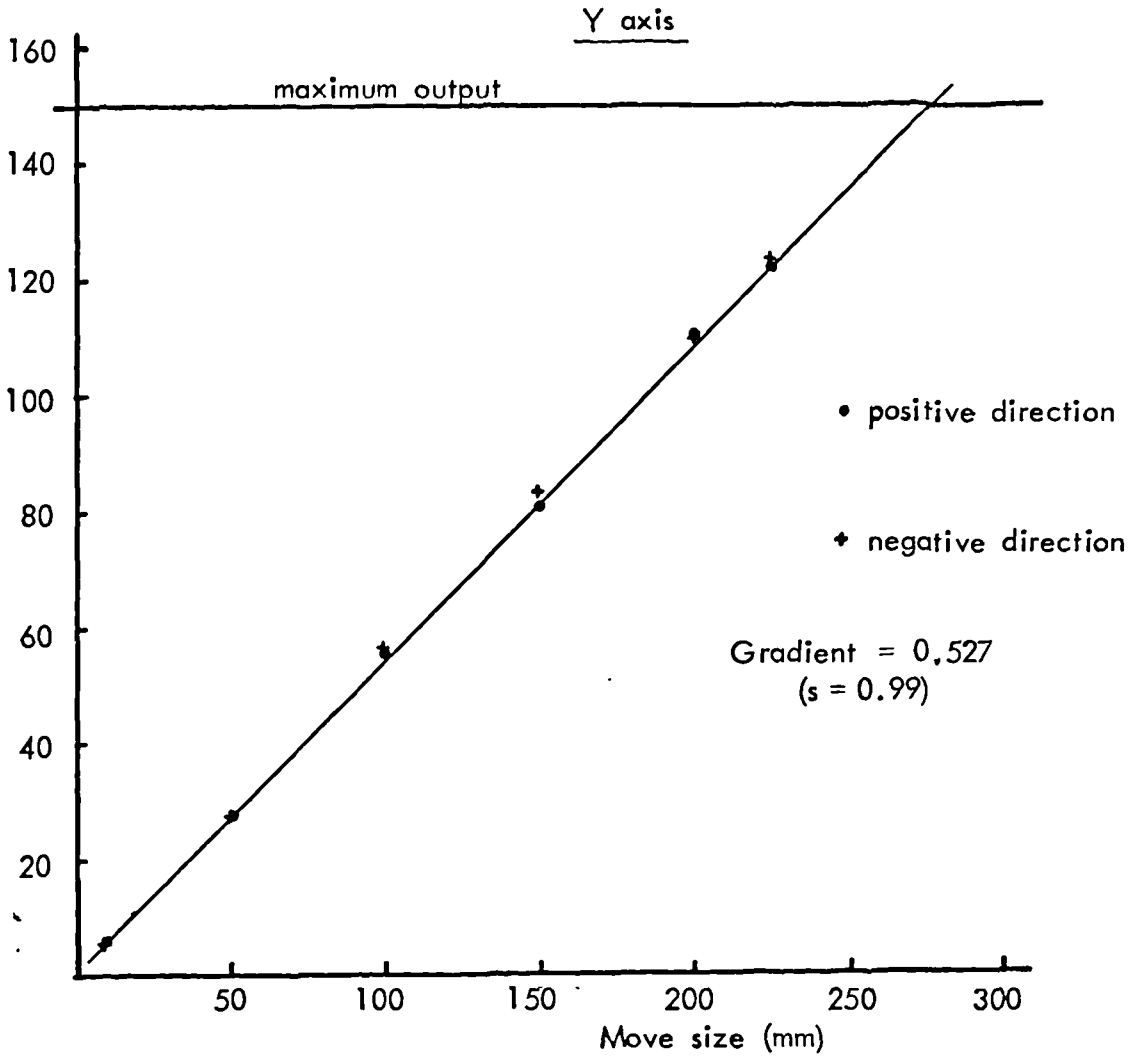


Figure A2.2b Mobility monitoring system calibration - output versus move size for moves along the Y axis in both positive and negative directions.

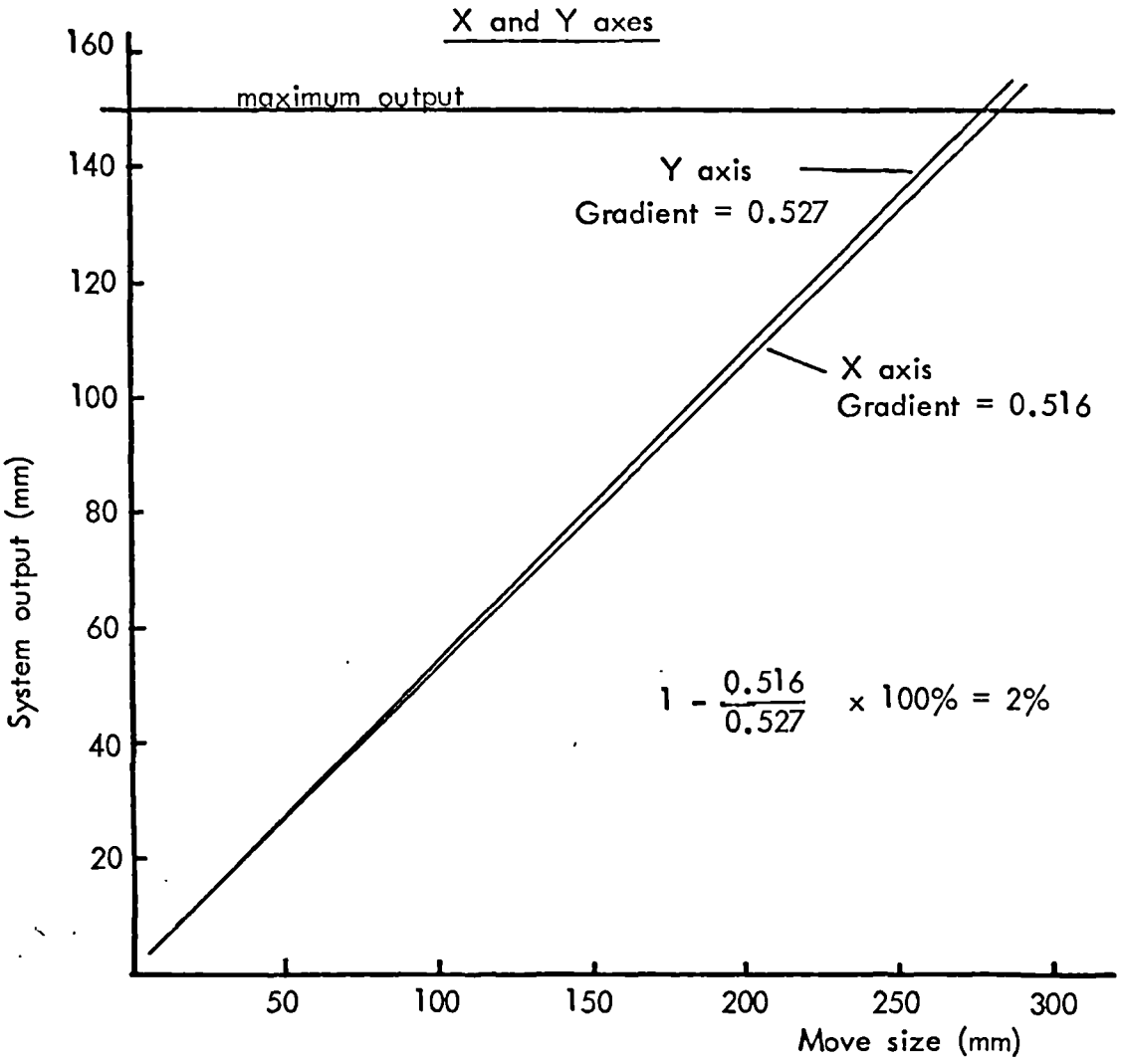


Figure A2.3 Mobility monitoring system calibration - output versus move size for moves along the X and Y axes. Demonstrating an increasing variation between the two.

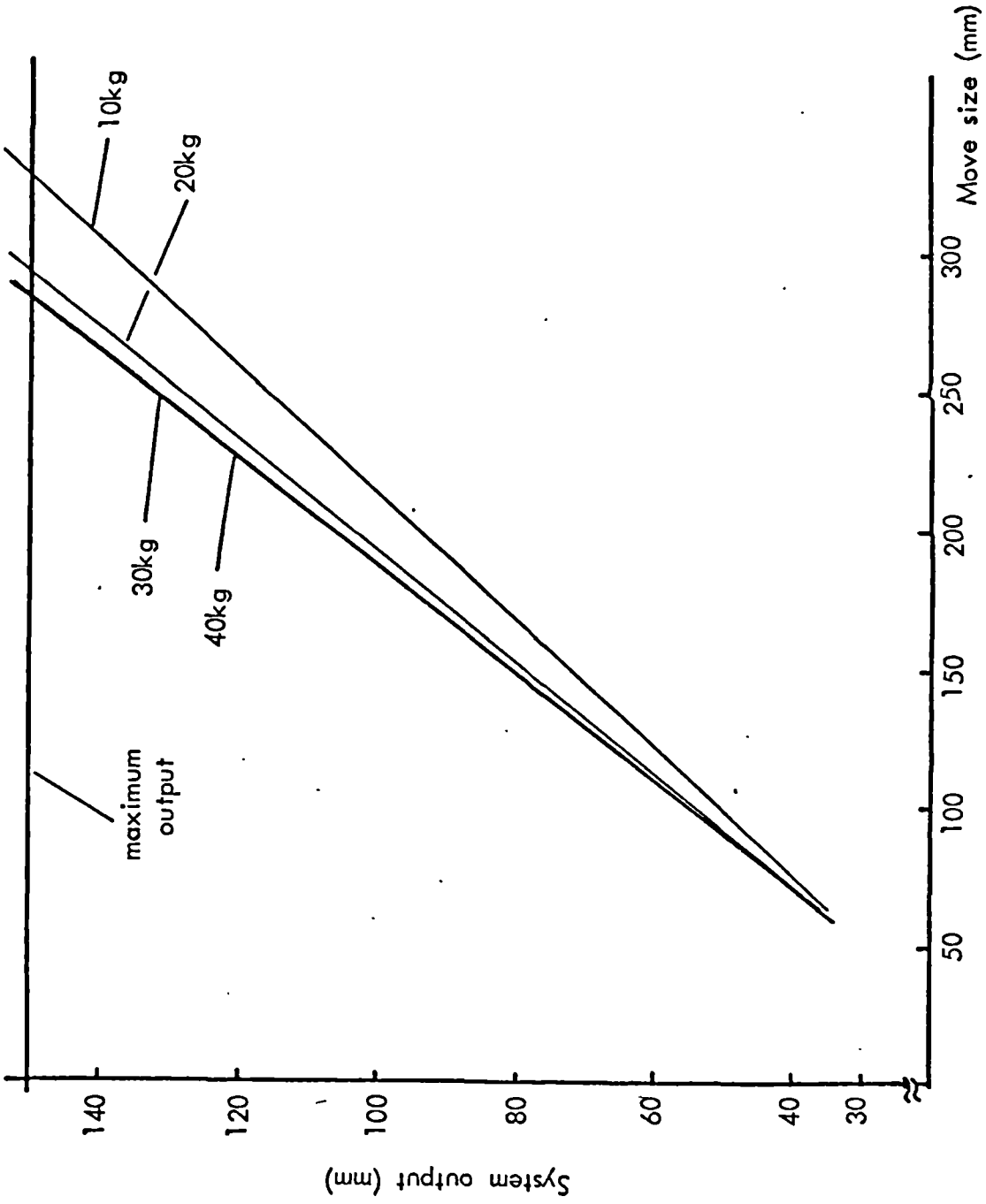


Figure A2.4 System output for a range of different loads. Indicating an increasing variation with decreasing load.

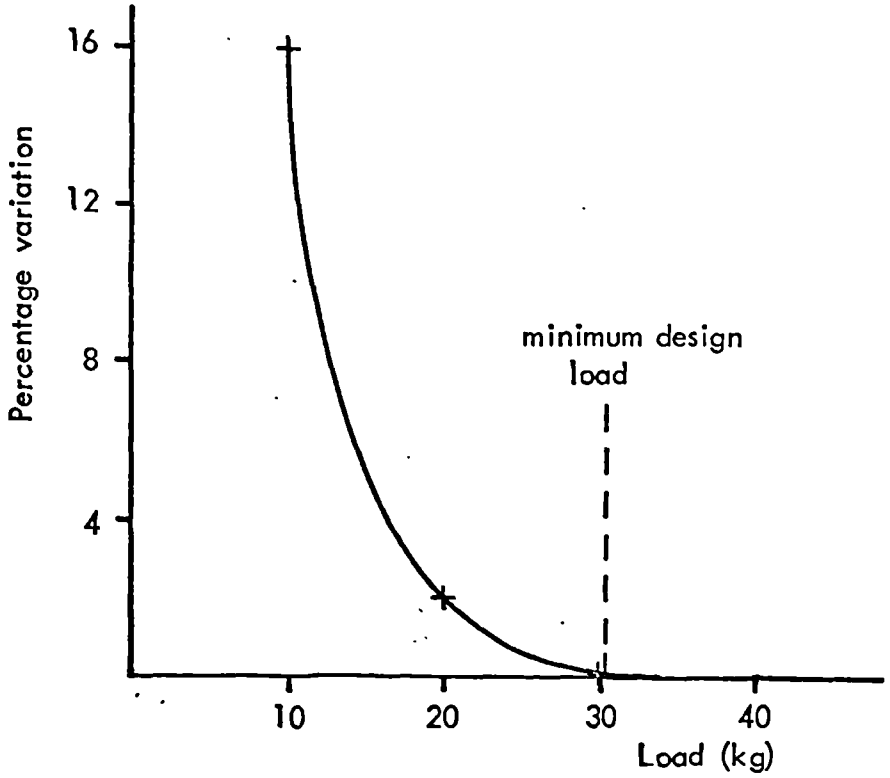


Figure A2.5 Percentage variation in output for equivalent move sizes for a range of different loads. Demonstrating a rapid increase for loads less than 30kg.

patient's weight, in the range of 30 kg to 100 kg (vide 5.2.1).

Figure A2.4 describes the response of the system to varying movement magnitudes with loads of 10 kg, 20 kg, 30 kg, and 40 kg. Results indicated that the output of the system at low loads was reduced in relation to larger loads. Having established the response of the system using a 40 kg load (vide A2.2.2), the output associated with 10 kg, 20 kg and 30 kg loads were compared to it. The results were characterised in terms of the gradient of the lines which were established by linear regression. Figure A2.5 describes this comparison as a percentage difference, in relation to the 40 kg results. From this it is evident that low loads can lead to substantial errors, whereas loads of 30 kg and larger produce the same output. This result was to be expected since the minimum design load of patient weight for the monitoring system was chosen as 30 kg (vide 5.2.1). The increased error at low loads is related to the inherent non-linearity of some of the analogue components in the processing system for low input voltages.

A2.2.4 Summary

The results of the calibration tests suggested that the apparatus was capable of measuring changes in position of load on a bed surface, with a linear response. Although an error was apparent when measuring moves along the different primary axes, this variation was within 2%. Additionally within its designed working range (>30 kg) the system was determined to be independent of the patient's weight.

APPENDIX 3

PATIENT RECORD FORM

Details of some of the clinical and environmental variables associated with the aetiology of pressure sores was collected for each patient studied (vide 6.3). This appendix contains an example of the record form used to collect this data.

Page 1 was completed by the experimenter. Pages 2 (i) - 2 (iii) were completed by the clinical staff. Pages 3 (i) - 3 (v) were completed by the nursing staff. In addition the nursing staff were asked to make a daily assessment of the patient's susceptibility to pressure sores using the Norton score and to record any drugs administered on the evening prior to, or during the course of, a test (page 4).

BIOENGINEERING DATA

TEST NO. ☐

PATIENT NO. ☐

For information in boxes; 1 = yes, 0 = no

MATTRESS TYPE:

- Water Bed ☐
- Water Mattress ☐
- Polyflotation ☐
- Spring Interior ☐
- DS 184 ☐
- Ripple (small cell) ☐
- Ripple (large cell) ☐
- Other ☐

Details .....

MATTRESS COVER:

- Stretchable ☐
- Waterproof ☐

Details .....

MATTRESS THICKNESS (in cms.) ☐

PATIENT SUPPORT SURFACE :

- Pillow packs ☐
- Draw sheet (linen) ☐
- Draw sheet (plastic) ☐
- Cotton sheets ☐
- Sheepskin ☐
- Other ☐

Details .....

.....

COMMENTS:

CLINICAL DATA

(To be filled in as soon as possible after admission)

TEST NO.

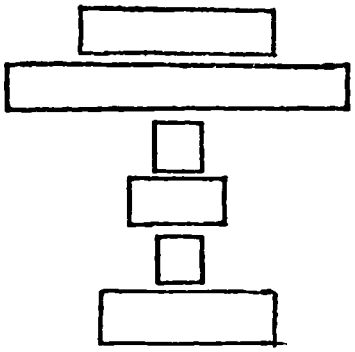
PATIENT NO.

SEX M or F

AGE in years

DOES THE PATIENT LIVE ALONE ? 1 = yes, 0 = No

WEIGHT in kgs.



DIAGNOSIS: Please put a tick in the appropriate box and add details in space provided below :

- Infective and parasitic diseases ☐
- Neoplasms ☐
- Endocrine, nutritional and metabolic disorders ☐
- Diseases of blood forming organs ☐
- Mental disorders ☐
- Diseases of nervous system and sensory organs ☐
- Diseases of circulatory system ☐
- Diseases of respiratory system ☐
- Diseases of digestive system ☐
- Diseases of genito-urinary system ☐
- Diseases of skin and subcutaneous tissue ☐
- Diseases of musculo-skeletal system and connective tissue ☐
- Congenital anomalies ☐
- Post surgical patient ☐
- Accidents ☐
- Other ☐

Details .....

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CLINICAL DATA (contd.)

PROPOSED TREATMENT :

Details .....

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DRUGS : (Please tick appropriate box and then add details in space provided below - (e.g. names of drug etc.))

- Alimentary system ☐
- Cardiovascular system ☐
- Central nervous system ☐
- Musculo-skeletal system ☐
- Hormones ☐
- Genito-urinary system ☐
- Infections and infestations ☐
- Nutrition ☐
- Respiratory system ☐
- Ear, nose and oropharynx ☐
- Eye ☐
- Allergic disorders ☐
- Skin ☐
- Metabolism ☐
- Surgical ☐
- Diagnostic agents ☐
- Dressings and appliances ☐

Details .....

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CLINICAL DATA (contd.)

PLASMA PROTEIN	Level in g/l	
PLASMA ALBUMIN	Level in g/l	
PLASMA GLUCOSE	Level in mmol/l	
HAEMOGLOBIN	Level in g/100 ml	

NUTRITIONAL STATUS .....

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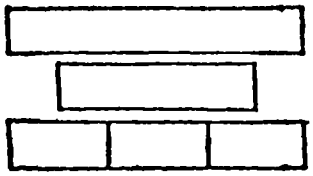
COMMENTS :

NURSING DATA 236

1. PATIENT NO.
2. TEST NO.
3. DATE OF ADMISSION



PRESSURE SORE HISTORY OF PATIENT

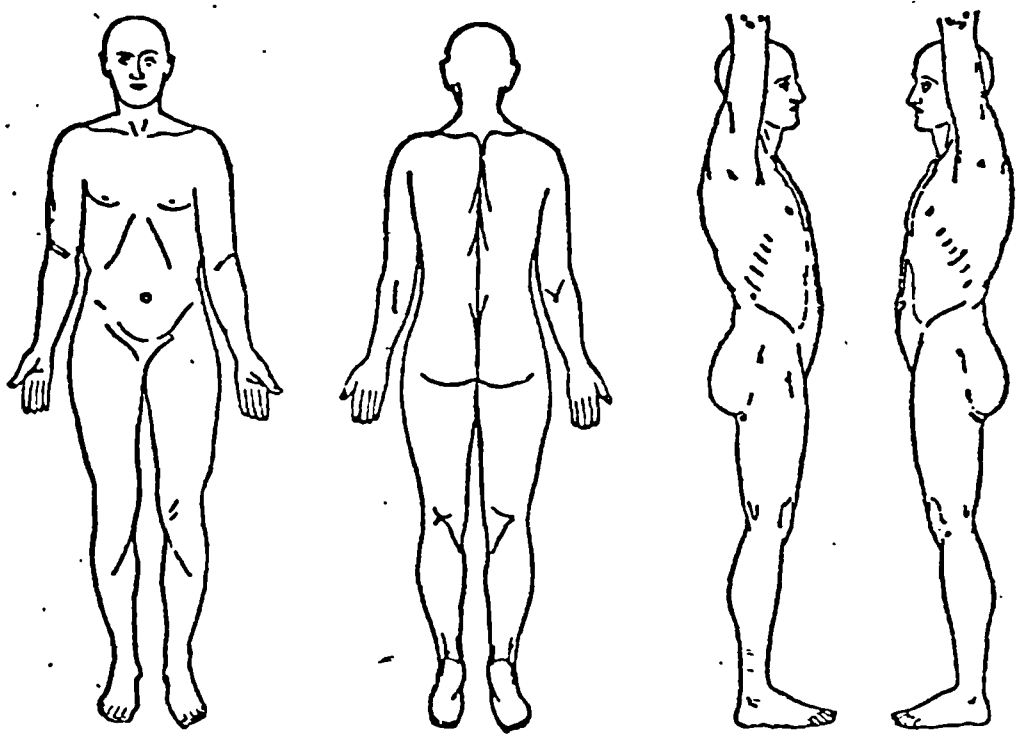
If the patient has no pressure sores. go to section 6, and if no history of pressure sores go to section 7.

4. PRESENT SORES :

The following system has previously been used to grade pressure sores :

Skin discolouration - transient	= 1
- persistent	= 2
Superficial pressure sores	= 3
Destruction of skin - no cavity	= 4
- cavity	= 5

Could you mark on the diagram the position of any existing pressure sore and number it using the above grading.



Details .....

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NURSING DATA (contd.)

5. TREATMENT FOR PRESENT SORES :

Could you tick those boxes that cover the treatment given for the present sores and give details in the space provided.

Medications ☐
Topical Applications ☐
Special Mattress ☐
Special Cushions ☐
Sheepskin ☐
Pillows ☐
Incontinence Pads ☐
Relief of pressure over area damaged ☐
frequency ☐ in hrs.
Other ☐

Details .....
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6. PREVIOUS SORES :

Could you tick the relevant box if any pressure sore in known to have occurred, and give details if possible.

	LEFT	RIGHT
Elbow	☐	☐
Trochanter	☐	☐
Ischial T.	☐	☐
Knee	☐	☐
Ankle - inner aspect	☐	☐
outer aspect	☐	☐
Heel	☐	☐
Sacrum	☐	
Coccyx	☐	
Occiput	☐	
Other	☐	

Details .....
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NURSING DATA (contd.)

7. The 'risk' of developing a pressure sore based on the nurse's decision using the NORTON SCORE.
This score is based on the use of five categories, all relevant to the development of pressure sores. Each category has four grades. The grade relevant for the patient on day of assessment should be put into the appropriate box.

(N.B. 'Physical Condition, means general state at the time and not the degree of disease or prognosis).

DATE OF ASSESSMENT :

A.	PHYSICAL CONDITION :	Good	= 4	
		Fair	= 3	☐
		Poor	= 2	
		V. Bad	= 1	
B.	MENTAL CONDITION :	Alert	= 4	
		Apathetic	= 3	☐
		Confused	= 2	
		Stuporous	= 1	
C.	ACTIVITY :	Ambulant	= 4	
		Walk/Help	= 3	☐
		Chairbound	= 2	
		Bedfast	= 1	
D.	MOBILITY :	Full	= 4	
		Sl. limited	= 3	☐
		V. limited	= 2	
		Immobile	= 1	
E.	INCONTINENT :	Not	= 4	
		Occasionally	= 3	☐
		Usually	= 2	
		Doubly	= 1	
	TOTAL SCORE		=	☐

A score of 14 or below is classed as 'at risk'

NURSING DATA (contd.)

8. SORE PREVENTION : (if applicable)

Please tick any box relating to preventative measures taken against the development of pressure sores and give details in the space provided.

Care of pressure points (e.g. massage, cleansing etc.)				☐
Special Mattress			☐	
Special Cushions			☐	
Sheepskin			☐	
Pillows			☐	
Incontinence Pads			☐	
Regular turning	☐	frequency	☐	in hrs.
Other			☐	

Details .....

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9. CONTINENCE

Please tick appropriate box.

Fully continent	☐
Occasional accident (twice or less in five days)	☐
Urinary incontinence (no indwelling catheter)	☐
Catheter in situ - to keep area clean and dry	☐
- for incontinence	☐
- both the above reasons	☐
Faecal incontinence	☐
Ileostomy or Colostomy	☐
Other	☐

Details .....

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NURSING DATA (contd.)

10. POSTURE : NIGHT-TIME IN BED - Please tick relevant box to describe patient's posture and add any details in space provided below.

	Prone	☐
	Supine	☐
	Lateral	☐
	Foetal	☐
	Other (specify)	☐
	DAYTIME	
If Bedfast	Prone	☐
	Supine	☐
	Lateral	☐
	Foetal	☐
	Sitting Upright (propped with pillows)	☐
	Semi-recumbent	☐
	Other (specify)	☐
Chairbound	Wheelchair	☐
	Geriatric Chair	☐
	Other (please specify)	☐
	Footrest	☐
Ambulant	No help	☐
	Nursing help	☐
	Zimmer	☐
	Other	☐

If up during day, for how long in hours approximately. ☐

Details .....

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DAILY REPORT SHEET - to be completed in evening

TEST NO. ☐

RUN NO. ☐

PATIENT NO. ☐

DATE ☐☐☐

HAS THE PATIENT BEEN UP TODAY - 1 = YES, 0 = NO ☐

HOW LONG IN HOURS ☐

If you think there has been any change in the patient's general condition.

Could you please note any change in A, B, C, D or E

in the NORTON SCORE

A ☐

B ☐

C ☐

D ☐

E ☐

TOTAL SCORE ☐

DRUGS ADMINISTERED .....

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TIME OF ADMINISTRATION : ☐

BODY TEMPERATURE in _°C (Taken in axilla for 1 min.) ☐

COMMENTS :

APPENDIX 4

CRITERIA FOR THE ASSESSMENT OF HISTOLOGICAL SPECIMENS OF SKIN AND MUSCLE

A 4.1 Introduction

A 4.2 Criteria

A 4.2.1 Skin

A 4.2.2 Muscle

A4.1 INTRODUCTION

In an effort to quantitatively evaluate histological specimens of skin and muscle, Daniel et al (1981) developed a set of "critical criteria". Dinsdale (1970) provided an outline of criteria for assessing specimens of swine skin subjected to pressure. However, comparable criteria for evaluating muscle were not available. On reviewing the literature, the detrimental effect of ischaemia on muscle is well recognised. Experimental studies by Harman (1947) and Strock and Majno (1969) have demonstrated the pathologic changes which follow ischaemia in muscle. Husain (1953) has investigated the effect of pressure on soft tissues, including skin and muscle. Harman (1947) states that ischaemia of 2 - 4 hours duration causes the muscle fibres to become individualised, with the disappearance of longitudinal striations, and the appearance of cross striations as a conspicuous cytological feature. After long periods, abnormal anisotropic disks appear and involve the muscle fibres in increasing numbers for up to 18 hours of ischaemia, at which time they are nearly ubiquitous. Husain (1953) conducted a series of investigations to determine whether short periods of pressure would induce damage of the skin and its underlying tissues. He concluded that pressure caused loss of muscle striation, conversion of sarcoplasm into homogenous material and eventual necrosis of the muscle fibres. Absorption of the necrosed part of the muscle fibres resulted in empty sarcolemmal husks.

Based upon this literature and their own observations, Daniel et al (1981) developed a set of criteria for evaluating histological sections of skin and muscle.

A4.2 CRITERIA

A4.2.1 Skin

Epidermis:

1. Inflammation - infiltration with polymorphonucleocytes and monocytes
2. Oedema - of the basement membrane
3. Blistering - separation of layers in the epidermis
4. Ulceration - loss of epithelium
5. Necrosis - focal changes

Dermis:

1. Oedema
2. Haemorrhage - red blood cells and side vessels
3. Thrombosis of vessels - arterioles and veins
4. Inflammation
5. Necrosis

A4.2.2 Muscle

1. Loss of longitudinal striations
2. Infiltration - white blood cells, giant cells
3. Vessel thrombosis and/or inflammation
4. Widespread individualisation and separation by structureless spaces
5. Necrosis

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