



**A search for the pathophysiology of the
Non-specific Occupational Overuse Syndrome (RSI).**

A Research Project undertaken in the
Department of Orthopaedics and Trauma, Royal Adelaide Hospital
and the Department of Surgery, University of Adelaide

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Abstract

Non-specific Occupational Overuse Syndrome — pain associated with activity for which there is no specific diagnosis and often referred to as Repetition Strain Injury (RSI) — continues to divide medical opinion. Previous accounts of its pathophysiology fall into two broad camps: those researchers who claim the syndrome is associated with some form of physical pathology and those who deny the presence of physical pathology, associating the syndrome instead with either fatigue or psychological pathology. This thesis presents findings which challenge both positions. It argues that fatigue cannot provide an adequate explanation and that the symptoms are an expression of neither psychological nor physical pathology. Rather, an alternative theory is developed which accounts for the pain by reference to an as yet unrecognised physiological control mechanism, the Physiological Activity Limitation Process (PALP). The thesis presents evidence that:

- some degree of pain or discomfort associated with activity is widely distributed in “normal” populations,
- that a significant proportion of the population experiences some degree of activity-related pain which does not disrupt normal activity,
- that the experience of these non-disruptive symptoms does not increase the risk of the development of the more disruptive symptoms typically associated with Non-specific Occupational Overuse Syndrome.

The thesis argues that pain or discomfort so widely experienced in “normal” populations cannot, in all cases, have a pathological basis and that, therefore, there must be a non-pathological cause. It proposes a “normal” pathological process (PALP) which generates unpleasant sensations (discomfort or pain) which function to limit activity to a level appropriate for an individual’s level of fitness. That is, when the intensity of some activity is greater than that appropriate for an individual’s level of fitness, then this physiological process generates pain or discomfort which typically causes the individual to limit that activity. The individual thereby avoids the injury which might follow if they consistently exceed their tolerance for the activity. The theory developed from this concept makes it possible to account both for cases of Non-specific Occupational Overuse Syndrome which respond to treatment and for those which prove more refractory. As well, a possible aetiology is suggested for other activity-related conditions which have not yet received generally accepted explanations such as Fibromyalgia, Myofascial Pain Syndrome, Reflex Sympathetic Dystrophy and Focal Dystonia.

This work contains no material which has been accepted for the award of any other degree or diploma in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text.

I give consent to this copy of my thesis, when deposited in the University Library, being available for loan and photocopying.

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Chapter 1. Introduction.

1.1. Introduction

Non-specific Occupational Overuse Syndrome - activity-associated pain for which there is no specific diagnosis - has certainly had a long history. It is, however, only in the past few decades that the syndrome - often referred to as RSI (Repetition Strain Injury) - has come to widespread public prominence with the emergence of apparent epidemics in Australia, the United States [Thompson and Phelps, 1990] and the United Kingdom [Prochak, 1993].. Typically associated with workers involved in process work and key -boarding, but also with musicians, these "epidemics" have resulted in a loss of productivity and a dramatic increase in workers' compensation claims.

Medical opinion has, up to the present time, remained fundamentally divided as to the pathophysiology of the syndrome, with some researchers claiming that pathological changes to peripheral tissue are involved while others have denied the presence of physical pathology and have proposed that the basis of symptoms is either fatigue or psychological pathology. This thesis reports on a research project into the syndrome which was triggered by a dramatic increase in the number of reported cases in Australia in the mid 1980s. It presents findings which challenge all previous hypotheses. It argues that the causes are neither psychologically nor physically pathological, that the pain does not imply the presence of disease or injury and that fatigue is not an adequate explanation either. Rather, the pain of Non-specific Occupational Overuse Syndrome is, in the majority of cases, an active physiological control mechanism which functions to limit the amount of regular normal activity within bounds safe for the individual.

1.2. Background

In the early 1980s two separate but related topics attracted widespread interest in the medical literature. These were the development of what appeared to be an Australian epidemic of upper-limb pain associated with occupational activity and a resurgence of interest in the musculo-skeletal problems of musicians. The major problem reported to affect musicians was that of upper-limb pain, again associated with their occupational activity.

The awareness of the Australian epidemic came from the increasing number of successful compensation claims awarded to workers because of upper-limb pain which was alleged to be caused by occupational activity. In New South Wales the number of these claims increased from 762 in 1978-79 to 2263 in 1981-82, the majority of which were not qualified by the presence of a specific diagnosis [Harris, 1984]. Consequently, the impetus for the

development of an understanding of Non-specific Occupational Overuse Syndrome came out of the medico-legal environment in which it became apparent.

Workers compensation claims in Australia, as in the United Kingdom and the United States, are conducted within the context of an adversarial legal system. Opposing parties (employer vs employee) typically seek to find medical experts who adopt a perspective supportive of their interests and who will accordingly offer strong support for their side of the case. Understandably, this adversarial legal context gave rise to a marked polarisation of opinion concerning the nature of the Non-specific Occupational Overuse Syndrome. Medical science, however, had not at the time found reproducible, objective data to explain the nature of the Overuse Syndrome and thus both the public debate and the court findings had to rely on opinion and conjecture rather than fact. This division of opinion has remained to the present day and has been reflected in a number of recent rulings in British courts. (Mughal vs Reuter News Agency, British High Court, 1993)

Researchers have taken various positions on the cause of Non-Specific Occupational Overuse Syndrome as a result of the Australian debate. These positions can be seen to divide the researchers broadly into those who take the view that pathological damage of peripheral tissue is involved and those who deny physical pathology. The latter can be divided into those who contend that physiological fatigue is an adequate explanation for the source of symptoms and those who suggest that symptoms are caused by psychological pathology. According to the former viewpoint, excessive occupational activity can cause pathological changes in the peripheral tissues involved in activity with potentially irreversible consequences. Thus, the employer is responsible for demanding excessive productivity and the employee should be compensated for both the pain and suffering caused and for the inability to return to work due to the damage produced. The psychological pathology side argues that cases of Non-specific Occupational Overuse Syndrome are due to psychological factors and do not result from occupational activity. Thus, the employer can not be held responsible. Those who favour the fatigue theory argue that fatigue is the expected result of activity and so the employees should accept it as inevitable. Both of these arguments imply that no physical damage or injury has occurred and consequently the employee is not eligible for compensation. Theories about the nature of the process which leads to the experience of pain in the Occupational Overuse Syndrome, its pathophysiology, have been proposed to support all sides.

In the past there has been considerable confusion in both the labelling and classification of occupation and activity-related pain syndromes. The terms which have been used to refer to this phenomenon have included tenosynovitis/tendonitis, occupational cervicobrachial disorder, occupational overuse injury, occupational musculo-skeletal disorder, cumulative trauma disorder, regional pain syndrome, repetitive/repetition strain injury (RSI) and occupational overuse syndrome. In 1986, in an attempt to limit this confusion, the Australian National Occupational Health and Safety Commission recommended that the term, "Occupational

Overuse Syndrome” be used as the umbrella term. The Commission's term applied to all causes of pain associated with occupational activity and thus included both pain due to known pathological causes as well as the most common situation where no specific diagnosis could be made. Although the Commission's definition created an umbrella term it also implied a primary subdivision into specific and non-specific forms. The following is the definition of Occupational Overuse Syndrome as presented in the Australian National Occupational Health and Safety Commission's publication “Repetition Strain Injury: A report and model code of practice 1986”.

“Repetition Strain Injury (RSI), also known as Occupational Overuse Syndrome, is a collective term for a range of conditions characterised by discomfort or persistent pain in muscles, tendons and other soft tissues, with or without physical manifestations. Repetition Strain Injury is usually caused or aggravated by work, and is associated with repetitive movement, sustained or constrained postures and /or forceful movements. Psycho-social factors, including stress in the working environment, may be important in the development of Repetition Strain Injury. Some conditions which fall within the scope of Repetition Strain Injury are well-defined and understood medically, but many others are not, and the basis for their cause and development is yet to be determined. It occurs among workers performing tasks involving either frequent repetitive and/or forceful movements of the limbs or the maintenance or fixed postures for prolonged periods, e.g. process workers, keyboard operators and machinists.”

The primary focus of confusion and controversy was with cases of pain for which there was no specific diagnosis and consequently, a more precise and detailed terminology than that provided by the Commission's umbrella term was required to distinguish this.

Some authors unilaterally chose to limit the use of the term RSI or Occupational Overuse Syndrome to the situation where a specific diagnosis could not be found [Fry, 1986b] whereas others added the qualifier “non-specific” to the term to make this distinction [Magdulski, 1985].

As the focus of this study was the non-specific sub-category, the latter approach was adopted to indicate this primary subdivision of occupational overuse syndrome. Hence, Occupational Overuse Syndrome (OOS) was subdivided into two sub-categories, Specific Occupational Overuse Syndrome (SOOS) (such as carpal tunnel syndrome, peritendinitis crepitans) and Non-specific Occupational Overuse Syndrome (NOOS). Despite the vast array of differing terminology used these terms have been used in this thesis to describe the work of other authors where a clear distinction has been made.

A number of previous researchers attempting to clarify the features of the Non-specific Occupational Overuse Syndrome have proposed systems for staging or grading severity [Browne et al, 1984][Fry, 1986b]. All of these systems have, however, proved to be in some way problematic or controversial, being criticised, for example, by other authors [Wright, 1987] because they were based on symptoms and signs rather than on objective measurements or tests, or because unproven prognostic value had been placed on the varying levels of staging.

At first the intention was to avoid the use of any form of subdivision of subjects classified as having Non-specific Occupational Overuse Syndrome and to simply analyse differences between affected and unaffected individuals. When collecting data early in the research, however, it became apparent that there were compelling grounds for making at least a secondary subdivision of those subjects who were in some way affected.

The research subjects came from two sources: patients who presented seeking treatment for pain associated with activity and a control group made up of students who presented to the University Health Service for the free first year preventative health check-up. Obviously, the first group of "patients" would be classified as having Occupational Overuse Syndrome since the syndrome was defined on the basis of the presence of symptoms, but it was soon discovered that a sub section of the control group also reported having experienced discomfort or pain associated with activity. There was, however, an important feature separating the majority of these control-group individuals from the "patients".

Careful questioning of the majority of those from the control group who did report symptoms, indicated a significant difference in the effect of those symptoms from the effect of the symptoms reported by the "patient" group. The critical feature of this difference was that those from the control group found their symptoms to be of no consequence in that they had felt neither the need to alter their activities in any way nor to seek treatment for those symptoms from any third party such as a doctor or physiotherapist.

Since Non-specific Occupational Overuse Syndrome is defined simply by the presence of symptoms, both the "patients" and this section of the control-group of individuals fell under this broad heading. But there were clearly significant differences between the "patients", those whose symptoms were of consequence in that they had altered their activities or sought medical intervention, and those whose symptoms were of no consequence. The stark difference between the effect of symptoms on these two groups strongly suggested a difference between them which should not be ignored by the research design and indeed should be investigated.

Accordingly, the cases of Non-specific Occupational Overuse Syndrome were divided into two classes, with the division being made on the basis of the effect of the symptoms on the lives and activities of subjects reporting some degree of activity-related pain. To make this process of classification explicit and systematic, symptoms were defined as "Inconsequential" or "Consequential". "Inconsequential" symptoms were those which were of no consequence in that they did not cause the individual to seek intervention from a third party such as a doctor or physiotherapist, nor did the symptoms affect their activities in any way. "Consequential" symptoms were defined as being of some consequence by either inducing the individual to seek help, or by affecting the activity involved in some way. Affected individuals were placed into

one of these two sub-categories according to whether their symptoms were "Inconsequential" or "Consequential".

Musicians were included in the study for the following reasons. Firstly, by 1985 the literature [Hochberg et al, 1983][Owen, 1985a] suggested that Non-specific Occupational Overuse Syndrome was a significant occupation risk for this group and thus they constituted a logical target group for investigation. Secondly, this population group could provide a useful and potentially revealing contrast with the other populations experiencing high levels of Non-specific Occupational Overuse Syndrome. In particular, they provided the possibility of testing the hypothesis that the syndrome is in some way causally connected with the presence or absence of job satisfaction. Ireland [Ireland, 1986a], for example, has suggested that as the "occupations in which RSI is prevalent are usually dull, monotonous, repetitious and boring - the development of the condition has more to do with job satisfaction than the activities involved". Most people would recognise that musicians are usually intensely interested and involved with their art and so lack of job satisfaction is unlikely to be the cause of problems affecting them. If the problem were found with a significant incidence in this population then this would cast doubt on the proposition that this problem is limited to uninteresting occupations.

1.3. Research Orientation and Methodology

In March 1986 a study was commenced with the aim of discovering whether or not data collected by routine clinical assessment of individuals would provide objective evidence in favour of any of the previously proposed theories. These theories were analysed to identify the assumptions they relied on and hypotheses they implied. On the basis of data to be collected by the study, a series of related hypotheses were constructed to enable the previous theories to be tested.

The subjects were selected from two sources. The first was chosen to provide the control group or a group of "normal" individuals. These subjects were taken from the first year students who presented to the University Health Service for the free first year student preventive health checkup. All music students over a two year period and a random selection of students from other faculties were included. The second source was that of patients who presented seeking treatment for pain associated with activity and who were classified as having Non-specific Occupational Overuse Syndrome.

The collection of data thus took four years of full-time research. The first two years were primarily involved with the initial assessment of the music students of 1986 and 1987 during the first year of their study. The last two years were primarily involved with the follow-up assessment of the music students during their final year of undergraduate study; the 1986

students completed in 1988 and the 1987 students completed in 1989. Within this time frame other subjects were assessed and the patient group treated.

Assessment was by use of standard clinical history and examination and a series of self administered psychological questionnaires. A standardised protocol for the examination of the upper-limbs for sites of tenderness was used irrespective of presence or absence of symptoms.

The study was conducted in three parts.

Part 1. A cross-sectional analysis of subjects taken from a "normal" population comparing musicians with non musicians and comparing individuals with and without symptoms consistent with Non-specific Occupational Overuse Syndrome.

Part 2. A case-control analysis of affected and unaffected individuals, created by adding a patient group to the subjects in part 1 comparing musicians with non musicians, and individuals with and without Non-specific Occupational Overuse Syndrome .

Part 3. A prospective analysis of the instrumental music students from the control group assessed initially at a time when they did not have "Consequential" symptoms of Non-specific Occupational Overuse Syndrome and followed for three years to identify differences between those who subsequently developed such symptoms and those who did not.

1.4. Overview of Research Findings

The results have been analysed in three stages.

1.4.1. Cross-sectional analysis

A summary of the major findings of the cross sectional analysis follows.

1. The prevalence of "Inconsequential Symptoms" in the control group was 37% in the instrumental musicians and 15% in the non-musicians.
2. The prevalence of symptoms associated with writing in both groups was similar (14% versus 15%) - the higher overall prevalence of "Inconsequential Symptoms" in musicians was due to symptoms associated with musical performance.
3. In both occupational groups 56% of those with no symptoms had presence of tenderness.
4. The prevalence of Specific Occupational Overuse Syndrome in the instrumental music student group was 2%.

1.4.2. Case-control analysis

A summary of the major findings of the case-control analysis follows.

5. There were 43 individuals classified as "Inconsequential symptoms", 15 of these recalled a recent modification to or change in intensity of the associated activity (35%). There were 37 individuals classified as "Consequential symptoms", 20 of these recalled a recent modification to or change in intensity of the associated activity (54%).
6. There were significant differences in the mean number of tender sites between each diagnostic category (ANOVA $p=0.001$). "No Symptoms" no. of sites =2.5, "Inconsequential symptoms" no. of sites =7.7, "Consequential Symptoms" no. of sites =16.2
7. There were significant differences in the prevalence of tenderness in asymptomatic areas between each diagnostic category (ANOVA $p<0.0001$).
8. In any one individual an asymptomatic area contra-lateral to a symptomatic area was significantly more likely to be tender than the other asymptomatic areas. (Paired Student's t-test, $p<0.0001$)
9. There were no differences in the mean number of tender sites nor sensitivity of areas to palpation comparing musicians with non-musicians within each diagnostic category.

1.4.3. Cohort Prospective analysis.

A summary of the major findings of the prospective analysis follows.

10. Those who initially had "Inconsequential" symptoms were not more likely to develop "Consequential" symptoms than those who initially had No symptoms. (Chi-square: 2 degrees of freedom = 2.88 $p= 0.24$)
11. Those with higher number of sites of tenderness did not have a higher risk of developing "Consequential symptoms" (ANOVA $p = 0.18$)
12. Females with low grip strength had an increased risk of developing "Consequential symptoms" (ANOVA $p = 0.03$)
13. The values for the measure of psychological profiles were within the normal range for all diagnostic categories.

14. Indicators for Conversion Hysteria - "Denial", "Disease Conviction" and "Affective Inhibition" showed no significant differences between categories.

15. There were significant differences between categories on the psychological profile "General Hypochondriasis" (Phobic concern about one's state of health) (ANOVA $p = 0.009$)

16. There were significant differences between categories on the psychological profile "Psychological vs Somatic Concern" (ANOVA $p = 0.013$)

17. There were significant differences between categories on the psychological profile "Affective Disturbance" (Tendency towards anxiety) (ANOVA $p = 0.038$)

1.5. Discussion

These results suggest that the symptoms of Non-specific Occupational Overuse Syndrome are the result of some physical process. This process would seem to be affected in some complex way by a number of physical and psychological factors. Evidence for its presence would seem to be detectable in a broad spectrum of the population including asymptomatic individuals. It would appear to be functioning in all people irrespective of the presence of muscular fatigue.

These results are thus incompatible with the theory that the process causing Non-specific Occupational Overuse Syndrome is purely psychological. Conversely they are also incompatible with the theory that this process involves pathological changes leading to progressive damage to tissues. Physiological fatigue does not seem to be an adequate explanation of the range of findings nor the spectrum of severity of symptoms. Consequently an alternative theory is proposed which allows more moderate conclusions to be drawn than might be wished by those on the opposing sides of the medico-legal debate.

The proposed theory is that the process which leads to the pain of Occupational Overuse Syndrome in the majority of cases is a physiological control process the function of which is to set a limit to the amount of regular normal activity. This process acts by modifying the sensitivity of peripheral structures so that when that sensitivity reaches a certain threshold then tenderness can be detected and when it exceeds a higher threshold then symptoms are experienced. The term "Physiological Activity Limitation Process" is proposed to describe this process. It is expected to function via the expression of discomfort/pain with the anticipated effect of limiting activity. The term "Physiological Activity-related Pain" is proposed to describe the mechanism by which this process exerts its controlling influence. An individual's actual response to Physiological Activity-related Pain will be affected by various psychological factors.

The conclusion that this theory suggests is that the pain of Non-specific Occupational Overuse Syndrome is due, in most cases, to the activities in the workplace but it does not imply the presence of disease or injury. In addition, neither employer nor employee can entirely excuse themselves of responsibility in the prevention and management of the condition.

Acting on the basis of this theory, employers would have to provide ergonomically designed equipment and utilise appropriate work practices in order to minimise the physical and psychological stresses of the tasks required. This is, fortunately, simply the requirements of good occupational health and safety guidelines. It may be that, in addition to this, employers could also provide facilities for employees to help increase their fitness in a way appropriate to the tasks involved.

Similarly, employees would have to take responsibility for utilising the facilities provided to make the demands of the job reasonable. They would also have to ensure that they achieve and maintain the level of fitness necessary for the activities required.

The medical practitioners responsibility would be to assess individuals who develop significant occupationally related discomfort in order to discriminate between those who are experiencing Physiological Activity-related Pain and others who have developed some form of pathological condition which is giving rise to their symptoms.

1.6. Conclusion

The findings of this study have suggested a new theory to help explain the origin of activity-related pain. It proposes an, as yet, unrecognised physiological process (the Physiological Activity Limitation Process). When the intensity of an activity is greater than that appropriate for an individuals level of fitness for that activity, then this process generates unpleasant sensations, Physiological Activity-related Pain, the function of which is to limit that activity. Consideration of the implications of this theory provides not only an explanation for the commonly seen cases of Non-specific Occupational Overuse Syndrome where symptoms do respond to appropriate management but can also provide reasons why some cases prove refractory to treatment. In addition it suggests a possible aetiology for a number of other activity-related conditions associated with Non-specific Occupational Overuse Syndrome which have not as yet received generally accepted explanations. These include Fibromyalgia (Fibrositis), Myofascial Pain Syndrome, Reflex Sympathetic Dystrophy and Focal Dystonia.

Chapter 2. Literature Review

2.1. Introduction

This study seeks to understand the phenomenon of activity-related occupational upper-limb pain which has been variously labelled as Occupational Overuse Syndrome, Repetition Stain Injury (RSI), Regional Pain Syndrome and Reversible Fatigue Syndrome, amongst others. For reasons explained elsewhere the term Occupational Overuse Syndrome has been selected for use in this thesis.

This chapter describes how this study was initiated by reference to the literature and published research findings which existed at that time. The social, medico-legal and media environment present in Australia which created a situation of interest and controversy concerning Occupational Overuse Syndrome will be discussed. There will also be discussion about the serendipitous occurrence of the development of international interest in musculo-skeletal problems of musicians and the relevance of this to the problem of Occupational Overuse Syndrome. Finally, the features contrasting the various conflicting theories relating to the pathophysiology of the Occupational Overuse Syndrome are highlighted.

2.2. The mass media

Initially, the dissemination of medical information occurred through mass media reports of presentations at medical seminars, conferences and meetings rather than in the medical literature. Although widespread public debate started in the early 80's it was not until 1986 that this began to be fully represented in peer review medical journals.

The problem of occupationally associated arm pain appears to have entered Australian society's consciousness by way of a booklet produced by the Liverpool Women's Health Centre, Sydney in 1976. This booklet entitled "Your job ... His profits or Your Life." purported to give information on the occupational risks of "Tenosynovitis".

In 1979 an article published in *New Doctor*, reporting the work of a Sydney Women's Health Centre, provided the headline "TENOSYNOVITIS, A crippling new epidemic in industry" [Walker, 1979]. Although the description of symptoms and signs included most of the structures of the arm, the term "tenosynovitis" was nevertheless used. The author proposed that the cause was repetitive movements or sustained contractions. In the article he "postulates" that the pathology was that of inflammation of tendons and chronic muscle strain. In addition he claimed that ergonomic deficiencies and poor work organisation were factors involved.

Over the next few years numerous articles appeared in the popular press. Such headlines as "Risk in the keyboard" [Smith, 1981] "Repetition Injury: a growing concern" [Gatehouse, 1981] and "Woman crippled by work wins hope for thousands" [Clarke, 1982] raised public awareness and concern about what appeared to be a new disease affecting workers in repetitive types of occupations. Initially the terms "tenosynovitis and tendonitis" were used to describe the condition. By 1983, however, it was widely accepted that the condition did not appear to be localised to the tendons and so more general terms came to be used. These included the term which has become the most controversial, Repetition Strain Injury (RSI), as well as numerous others such as Overuse Injury, Occupation Overuse Syndrome, Regional Pain Syndrome, Reversible Fatigue Syndrome. Despite the fact that the tendons rarely appeared to be involved, the original concept of Walker that the condition was due to pathological changes in tissues at the site of symptoms was largely followed during that time.

As awareness of the problem became increasingly widespread in the early 1980s, there was a parallel increase in concern about rises in workers compensation costs. [Thomas, 1982, "Compo: why costs are so high."] and [Kissane, 1985, "RSI"]. Epidemiological observations based on the figures from state workers compensation statistics indicated that the incidence of the problem was dramatically increasing, "Keyboard cripples: the avalanche looms." [Kavanagh, 1984]. With this came a drive for increased understanding and a call for research into this puzzling phenomenon. A number of anomalous features became apparent and alternative theories relating to the nature of the pathological processes involved were developed. These were first presented at scientific meetings, and because of the high public interest in the problem at that time, were subsequently reported in the public press.

The work of Quintner and Elvey was reported in the Sydney Morning Herald, June 1, 1985. The article "A pain in the arm is a pain in the neck" described their theory that the origin of symptoms was not the muscle or tendons but was due to tethering of the neural structures in the arm and brachial plexus.

At a seminar in Sydney in 1981, Lucire first presented her theory of RSI as a psychiatric condition due to conversion neurosis. She was reported in "The Australian" of December 10, 1985, "Scare tactics cripple the unwary". The hypothesis that she put forward was that the primary source of symptoms was conversion neurosis and that the epidemic was a form of mass hysteria.

2.3. Upper-limb pain in musicians

At the same time there occurred an international increase in interest in the occupational problems of musicians. This would seem to have been triggered in the first instance by the development of activity related arm pain in two internationally renowned concert pianists who were treated in Boston, USA. This led to a paper by Hochberg et al in the Journal of the American Medical

Association, April 1983 [Hochberg et al, 1983]. Here they reported a series of 100 musicians similarly affected. In 1983 in Australia an interested group of doctors formed the "Performing Arts Medicine Society", leading to a series of meetings and seminars in both Melbourne and Sydney. This society initiated two studies to investigate the medical problems of musicians in Australia.

The first study was based in Sydney, Australia, and was reported by Owen in the new *Journal of Occupational Health and Safety - ANZ* [Owen, 1985a]. Owen reported on his personal assessment of 110 musicians with upper-limb pain. He found that although "violinists and pianists made up the majority" of cases "all musical instruments were seen to induce specific repetition strain injuries peculiar to that instrument". As a consequence of his findings he contended that "music can be a physically damaging occupation". The second study was undertaken by Hunter Fry and involved a survey of musicians throughout Australia. The early results of this study were first reported in the popular press by Burnbauer in "The Age", December 1985, in "music charms failed to soothe savage breast". He surveyed a series of over 900 musicians from six symphony orchestras and nine music schools by personal interview and examination. He found that over 40% of the musicians in his survey were experiencing upper-limb pain of some consequence.

In October 1985 a seminar on medical problems of musicians was run by the International Conference of Symphony and Opera Musicians in San Francisco, USA. At this seminar it became clear that the problem of upper limb pain associated with their occupational activity was common amongst musicians throughout the world.

2.4. The Medical Journals

Prior to the commencement of this study there were only 5 peer reviewed articles published directly relating to the Australian problem. All but one of these took the point of view that the cause of symptoms was due to some pathological process occurring local to the area where symptoms occurred. In contrast to this there were 44 letters to the editor in the *Medical Journal of Australia* alone, where a wide range of opinions and theories were presented.

2.4.1. Peer reviewed articles

The first peer-reviewed article in the medical literature relating to the Australian epidemic appeared in the *Medical Journal of Australia*, December 1983, by William Stone. He described a series of 100 patients with repetitive strain injury and related their symptoms directly to their employment. He described the condition as a collection of different diagnoses including carpal tunnel syndrome, tenosynovitis, epicondylitis and ulnar neuritis. He also described a form of the condition which he called static strain injuries, where he could not localise the pathology but did not clarify the relative incidence of the various diagnostic labels he had discussed. He did

not make any suggestion about the nature of the pathology associated with the non-specific form.

In March 1984, the occupational repetition strain injuries advisory committee of the Division of Occupational Health, New South Wales Government, Department of Industrial Relations presented their results in an article of the Medical Journal Association entitled "Occupational Repetitive Strain Injuries: guidelines for diagnosis and management", [Browne et al, 1984]. This report recognised the high incidence of this problem in Australia and commented on the difficulties with terminology, both in the literature and amongst the medical community, in terms of reporting for the purposes of epidemiological statistics. This article again concluded that the condition was directly attributable to occupational activity and that it was a collection of various conditions including peritendinitis, tenosynovitis, epicondylitis, peripheral nerve entrapment as well as a substantial group with poorly localised discomfort which was attributed to the long term effects of static muscle loading. The article contained a comment that the pathology of this non-specific form was unclear. It proposed an "arbitrary" three level system of staging based on the symptoms reported.

- stage 1 Aching and tiredness of the affected limb which occurs during work, but settle overnight and on days off. There is no significant reduction of work performance nor any physical signs. This condition can persist for weeks or months and is reversible.
- stage 2 Recurrent aching and tiredness which increasingly occur earlier in the work shift and persist longer. Symptoms fail to settle overnight, causing disturbance of sleep and are associated with reduced capacity for repetitive work. Physical signs may be present. This condition usually persists for months.
- stage 3 Aching, fatigue and weakness persist at rest and pain occurs with non-repetitive movement. Symptoms cause disturbance of sleep. The affected person is unable to perform light duties and experiences difficulty with non-occupational / activity tasks. Physical signs are present. The condition may last from months to years.

The implications of this system of staging was two fold. Those whose problem could be classified by this grading system were all affected by the same condition at varying degrees of severity. Progression from less severe to more severe grades was to be expected if appropriate intervention were not undertaken. The authors concluded that "advanced repetition injury may mean permanent incapacity for repetitive work".

In the same journal David Ferguson wrote an editorial entitled "The 'New' Industrial Epidemic" [Ferguson, 1984]. He again discussed the problems with terminology. He listed a variety of specific diagnoses included within the spectrum of conditions labelled by others as RSI but went on to say that the majority of cases were not of localised syndromes but of a more diffuse disorder, apparently of muscles. He described the symptoms relating to this non-specific condition to be those of aching, weakness and tenderness of muscles and listed the various terms which have been used to describe it including occupational myalgia, myositis, myopathy, fibrositis, fibromyocitis, muscular rheumatism, non-articular rheumatism, myofascial

syndrome and tension myalgia. He commented that this syndrome was identifiable from occupational cramp but with which it may co-exist. He further commented that “although this ill-defined muscle aching was extraordinarily prevalent, little was known of its aetiology pathogenesis or pathology”. He commented that when the condition became well established it appeared “to persist despite prolonged rest of the affected parts”.

At this stage it became clear that the phenomenon of occupational related hand pain consisted of a variety of different conditions which had been attributed to excessive occupational activity. In a number of cases it appeared that this had led to specific localised pathological changes for which a specific diagnosis could be attributed. The majority of cases, however, appeared to be of a more non-specific nature, without localising clinical features. It was this group in which the pathogenesis and pathology were unknown.

Owen [Owen, 1985b] suggested that previous work looking at muscle changes in the study of exercise physiology, as well as previous studies of peritendinitis crepitans, had shown histological muscle changes and concluded that the pathology of RSI was some form of injury to the muscle tendon unit due to excess of activity.

The American industrial rheumatologist, Norman Hadler, undertook a study tour of Australia in 1985 supported by the World Health Organisation, to study the problem. He took a new position from the previous authors and questioned the presence of localised pathology. His conclusion was "that the pathophysiology of the upper extremity use-associated discomfort is indeterminate and that the symptom complex defies current nosology". He claimed that neither carpal tunnel syndrome nor tenosynovitis were appropriate diagnoses for this phenomenon and questioned the causal relationship between activity and symptoms. He formulated the hypothesis that the problem was a form of "fatigue". Interestingly he seemed to take a somewhat defeatist attitude to the problem and suggested that "perhaps the underlying pathophysiology of use-associated arm discomfort is not worth pursuing". Nevertheless he recognised the workers right to a "comfortable workplace" whether injury or disease was the cause of discomfort or not. This work was first reported in the American literature [Hadler, 1985] and later in the Medical Journal of Australia [Hadler, 1986b].

2.4.2. Letters to the Editor, Medical Journal of Australia

Although “letters to the editor” do not generally have the rigour or detail of fully fledged peer reviewed articles, an analysis of the collected correspondence published on the problem of Occupational Overuse Syndrome in the Medical Journal of Australia gave a number of valuable insights into the nature and extent of the problem. The impact of the problem on the medical profession in Australia is also demonstrated. The more radical and speculative theories as to pathophysiology which could not be expected to be presented in the form of an article, could also be appreciated. The following is a review of this literature (44 letters) in the period leading

up to the commencement of this study. The debate which occurred in these letters focused on the contentious area of Occupational Overuse Syndrome, in particular that of Non-specific Occupational Overuse Syndrome.

This body of work can be seen as an informal discussion between colleagues over time, in which different “speakers” put forward their ideas and others reply either supporting or criticising those ideas. By analysing both the form and content of this discussion it is possible to gauge the difficulties faced in attempting to comprehend this problem, and in deciding on a systematic approach with which to undertake research whereby the arguments might be unravelled.

Analysis of the content of the arguments allowed the ideas as to pathology to be grouped into 4 main concept areas. Each area also contained a wide diversity of opinion. The following is a table systematically listing the various ideas with the number of advocates for each. Criticisms tended to be directed at whole concept areas, those in favour of psychological or absent pathology arguing against physical pathology and visa versa. The number of critics are therefore listed against the overall concept area.

Main Concept Area	Specific Ideas	No. of Advocates	No. of Critics
Physical pathology	Musculo-ligamentous pathology		5
	Direct localised damage or injury	19	
	Chronic compartment syndrome	1	
	Pathology of sympathetic nerves	2	
	Pathology of peripheral nerves	1	
	Fibrositis (Fibromyalgia) / Myofascial Pain Fluorosis	1 1	
Psychological pathology	Neurosis unspecified	4	1
	Compensation neurosis	3	
	Conversion Hysteria	2	
	Psychosomatic	1	
Absence of pathology	Simple disbelief	3	5
	Malingering	3	
	Pain due to fatigue	1	
Missed pathology	Persistent problems due to the presence of a missed specific diagnoses.	5	0

Consideration of both the arguments for and against each idea and concept are revealing.

2.4.2.1. Physical Pathology

The most common opinion favoured the original idea of Walker that the primary cause of pain was due to some physical pathology. The absence of clinical signs which could localise the site of pathology to the tendons led to a range of variations on this theme, various possible sites

were proposed for the expected pathological changes or injury as required by the most commonly used but universally scorned terminology, "Repetitive Strain Injury".

Indeed the first letter to the editor was written by Lowy in December 1983, suggesting that the pathology might be of the sympathetic nervous system.[Lowy, 1983]. Lowy reported personal clinical observations of patients with "RSI" and proposed the theory that "many of the symptoms and signs in occupational overuse and RSI-type disorders result from abnormal neurovascular activity caused by dysfunction of the autonomic receptors". The observations he reported include the presence of symptoms such as "pain and burning, paraesthesiae, altered sensation, restriction of movement, swelling and tenderness" and also in "severe cases, changes such as discoloration and mottling, hyperhydrosis, temperature alteration, oedema, and alteration in sensitivity". Stone was asked by the editors to comment on this theory and replied that such changes were only "infrequently associated" with Repetitive Strain Injury and stated "No direct link between repetitive strain injury and reflex sympathetic dystrophy is known to exist" [Stone, 1983b December]. Graham and Mills [Graham and Mills, 1984 March], however, supported Lowy's theory stating "Our experience covering more than 350 cases ... indicates that sympathetic overactivity in the affected limb is common". Awerbuch [Awerbuch, 1984 June] subsequently contradicted this, arguing that "signs of sympathetic overactivity are not synonymous with RSD (reflex sympathetic dystrophy)". "The neurotic patient (hysterical or otherwise) with an obsessive reluctance to move the limb will invariably demonstrate a cold blue or dusky upper limb."

Magdulski [Magdulski, 1985 April] commented on the theory espoused by Littlejohn and reported in the woman's magazine "New Idea" 20/10/84 that "RSI" is a form of localised "fibromyalgia" or "myofascial pain syndrome". Unfortunately this proposition did not help with the task of localising the site of pathology. As recently as 1989 Goldenberg, in a review of current treatment of fibromyalgia syndrome, stated "fibromyalgia is an idiopathic disorder which causes chronic pain and manifests few objective clinical features. The basic pathophysiological abnormalities in fibromyalgia are unknown". [Goldenberg, 1989a]

Schultz [Schultz, 1985, April] proposed that if "RSI" is due to "chronic muscle strain" then the primary pathology may be that of chronic compartment syndrome. Chronic overuse of muscle may lead to "muscle hypertrophy within non-stretch fascia". Patkin [Patkin, 1985a September] reported the work of Quintner and Elvey in which the theory of peripheral nerve tethering was proposed as the primary pathology. Patkin described the pain of Non-specific Occupational Overuse Syndrome as "bratiatica", equating it as the upper-limb counter part of lower-limb sciatica. Smith proposed that "some cases of RSI may result from deposition of apatite crystals in and around synovial sheaths and tendons" and that this could be caused by excessive fluoride intake. [Smith, 1985c]

The common theme of the arguments questioning the theories of physical pathology was summed up by the statement of Rush. He contended that Non-specific Occupational Overuse Syndrome (RSI) “is a clinical condition the diagnosis of which relies entirely on the patient’s complaints of pain. There are no clinical signs. There are no known pathological features and there is no satisfactory treatment.” [Rush, 1984 October]

2.4.2.2. Psychological Pathology

Block wrote the first letter to the editor which cast doubt on the physical pathology theory. He observed that in cases of Non-specific Occupational Overuse Syndrome, “Stressful personal or leisure activities may continue unabated. Return to work duties neither tiring nor repetitive, is declined or barely attempted. Resolution has followed on satisfactory court awards.” He denied the possibility that non-traumatic muscle activity could cause damage. “Overuse of skeletal muscle is prevented by physiological mechanisms.” “Repetition is the essence of conditioning and recovery from fatigue is rapid and total.” He asked, “Is RSI an entity or a figment of vested interests and politics?” Although he did not come to any specific conclusion as to an alternative pathology the implication from the article was that compensation neurosis was likely. This opinion was confirmed by Rush who stated “The condition is almost invariably seen in workers who are making a worker’s compensation claim.” [Rush, 1984, October]

Awerbuch also contended that “It is physiologically impossible to cause permanent injury to striated muscle either by isometric or dynamic movements”. He pointed out that “real pain does not mean real injury”. “There may be no physical stimulus for an individual’s subjective pain experience”. “The neurotic patient (hysterical or otherwise) with an obsessive reluctance to move the limb will invariably demonstrate a cold blue or dusky upper limb.” [Awerbuch, 1984, June] This writer also did not clearly specifying an alternative pathology. The implication would seem to be, however, that the primary pathology was conversion hysteria.

Wilson suggested, however, that the non-specific form of Occupational Overuse Syndrome should be divided into three groups. The first group included those who rapidly responded to appropriate intervention, he cited four cases. The second group included those with true chronic physical pathology. The third “would consist of people with significant pre-existing problems with home life or work conditions which they are unable to resolve.” These cases “may be more appropriate to explore in terms of social pathology.” It is unclear what this writer meant by “social pathology” but the resolution of psychological conflicts by the development of physical symptoms seems to imply conversion hysteria. He thus allowed for the possibility that physical and psychological pathology may occur in different patients. [Wilson, 1985]

Other writers made general reference to “nervous disorders” and “neurosis” without specific detail to permit a more specific psychological pathology to be implied. “Are we pandering to a neurotic Australian work force? Should we drop the term RSI and revert to the old designation of ‘craft neurosis’.” [Sharrod, 1985, March] “The use of industrial psychology clinics would help re motivate people” [Pervan, 1985, April]. “Overseas industrial psychologists over a decade ago found high incidence of nervous disorders and muscle cramps and spasms in machine-paced workers especially keyboard operators who lacked personal control in pacing their work”. [Ghosh, 1985, September]

Snell complained that “conditions with a clear clinicopathological pattern (Specific Occupational Overuse Syndrome) are now all being grouped together with patients exhibiting strange, diffuse, non-specific pains which they claim are due to repetitive strain.” “The psychosomatic group of patients are increasing in numbers alarmingly”. [Snell, 1985, January] Snell was clearly indicating the opinion that the primary pathology of Non-specific Occupational Overuse Syndrome was psychosomatic.

2.4.2.3. Absence of Pathology

The third concept was that the symptoms of Non-specific Occupational Overuse Syndrome may occur in the absence of any true pathology, either physical or psychological.

The most famous letter which apparently set out this opinion was that of Awerbuch in which he proposed to rename RSI as “Kangaroo Paw”. This letter is unfortunate in that the message was not at first apparent. The style was sarcastic and mocking and this tended to hide its content. He stated, “the diagnosis (of Non-specific Occupational Overuse Syndrome) rests on only one criterion, the complete absence of objective clinical signs of abnormality”. “Treatment is by splintage, compression bandaging and magnetic wave therapy and rest, rest and more rest. This is the stuff of genius - nay Nobel laureateship”. He claimed that the problem is “perpetuated with the approbation of the medical profession, either through ignorance or avarice or both.” Although on the surface it appeared that he was denying the existence of Non-specific Occupational Overuse Syndrome, the full implication is that he was suggesting that the problem existed only in the minds of the sufferers.

Ackland was more direct in pointing out his disbelief in the existence of “RSI”. He indicated this with the statement that “RSI” could not be a “valid diagnosis” for it “has no surgical meaning”. [Ackland, 1985, June] Sinclair stated that “Pain that lasts for months or years, which is unremitting and refractory to all forms of treatment and is unaccompanied by any sign whatever, simply cannot be arising from muscles or tendons.” Doctors “must not be afraid to state that no diagnosis is possible if the signs and symptoms do not make normal medical sense.” [Sinclair, 1985, November]

These opinions seemed to be implying that “RSI” does not exist and this was often the interpretation placed on them made by the lay public. This interpretation was unfortunate as these doctors did not doubt the presence of symptoms but only that there was any physical pathology causing those symptoms.

A more complete denial of the existence of pathology came from those writers who implied that a significant proportion of cases of Non-specific Occupational Overuse Syndrome were in fact cases of malingering. Cowan suggested, “Might it just be the case that, if not going to work meant going hungry, all these sufferers would somehow overcome their disabilities?” [Cowan, 1984, May]

Scarf and Wilcox described the following features which they found to be common to most cases of Non-specific Occupational Overuse Syndrome. “They often do not seem to have a strong work ethic, the pain gradually increases despite treatments and rest and the extent of the pain does not fit with any sensible clinicopathological entity. They frequently have well-developed musculature despite alleged non-use. We often see ingrained dirt on the palms of these “functionless” limbs. There is commonly a disparity of function observed between undressing and formal examination. Sometimes glove hypoaesthesia is found.” Although not spelled out, these are the features which all clinicians are taught to associate with feigned disease.

The final mechanism proposed to explain a source for symptoms without pathology was that of fatigue. Sinclair complained that the “Current plague of hearsay and media hype serves only to frighten honest, well-meaning workers into believing that they will be permanently crippled from what is nothing more than fatigue pain or postural neuralgia.” [Sinclair, 1985, November]

2.4.2.4. Missed Pathology

Writers from both the physical [Lowy, 1984, September][Schultz, 1985, April][Davies, 1985, July] and the psychological [Carey and Moran, 1985, April] pathology ideologies have raised the point that making the diagnosis of Non-specific Occupational Overuse Syndrome too readily would have the effect of leading to missed specific diagnoses. Patients with specific conditions where the pathology is well understood and recognised treatment known to be effective, might fail to be adequately assessed.

2.4.2.5. Personal criticisms and comments

An interesting feature of this “scientific” debate was the strength of personal feeling expressed by many of the writers. This was shown with a number of examples. Awerbuch’s made mocking comments of other’s work, “This is the stuff of genius - nay Nobel laureateship”. Magdulski’s replied, “Dr. Awerbuch’s comments on non-specific RSI are grossly inaccurate.

In the interests of sufferers and the families ...” Pervan, although he agreed with Awerbuch in principle, reproached him for “indulgence in pungent wit”. The best example of invective was perhaps Smith’s retort to Cowan’s proposition that RSI was malingering. Smith suggested that Cowan’s proposition was “probably less of an insult to the intelligence of others than it is a reflection of his own”.

2.4.3. Peer reviewed articles published after the commencement of this study.

A number of peer reviewed articles were published soon after the commencement of this study. Although it was not possible to utilize in this study the detail of the ideas and theories presented in these papers, previous discussion of the work of these authors in the mass media and the “letters to the editor” of various journals, allowed aspects of their theories to be considered.

At a seminar in Sydney in 1981 Lucire first presented her theory of RSI as a psychiatric condition due to conversion neurosis, but it was not until October 1986 that her work was first published in a peer review journal [Lucire, 1986]. She contended that the epidemic of RSI could be explained by psycho-social phenomena due to epidemic hysteria on the part of patients and the result of altered medical perception of endemic symptoms within the community. The reasons she used to justify this were the absence of pathological data and the failure of predictable recovery with appropriate rest and treatment. She suggested that the majority of patients with RSI had a pattern of symptoms consistent with psychogenic illness which could not be explained by any known pathophysiological mechanism. She referred back to previous epidemics of occupational related upper limb problems and suggested that these also were due to conversion neurosis.

Valencia [Valencia, 1986] extended the proposition put forward by Hadler into a formal hypothesis suggesting that the patho-physiological basis of Non-specific Occupational Overuse Syndrome may be local muscle fatigue. “The excessive loading of muscles under these conditions may lead to local muscle fatigue which may, in turn, manifest as pain, localized discomfort or kinaesthetic dysfunction.” “The physiological and biochemical changes in the muscle under contraction, which initiate the events of local muscle fatigue, may provoke the failure of individual muscle fibres or motor units. Chemoreceptors and mechanoreceptors in the muscle will convey this information to the central nervous system. Physical discomfort is the first symptom of muscle fatigue that is perceived.” This article does not make it clear whether the author believed that all the symptoms of Non-specific Occupational Overuse Syndrome could be attributed to fatigue or that fatigue was simply a precursor to the pathological changes which are its basis. The title of the article “Local muscle fatigue, a precursor to RSI?” would seem to suggest the later.

Research by Quintner and Elvey in Perth, Australia, led them to propose the hypothesis that the pathology underlying this condition was that of tethering of the peripheral nerves in the upper limbs and neck. They proposed that this occurred as a result of irritation due to repetitive movements, due to both movement of the nerves within the soft tissue and to release of metabolites from the surrounding muscles [Elvey and Quintner, 1986].

In the Australian and New Zealand Journal of Surgery, January 1986, Fry described the presence of consistent patterns of tenderness which, he argued, could be used to identify cases of overuse syndrome. He stated that "Such signs in the intrinsic muscles of the hand and ligaments of the wrist joint and carpometacarpal joint of the thumb are virtually diagnostic of overuse injury." [Fry, 1986a] He presented the findings of his survey of musicians in the February edition of the Medical Journal of Australia 1986 [Fry, 1986b]. In this he proposed his own method of staging the severity of the condition according to 5 "arbitrary" grades as follows.

- | | |
|---------|--|
| Grade 1 | Pain in one site brought on by playing the instrument |
| Grade 2 | Pain in two or more sites, difficulty with high workload, possibly some loss of co-ordination, possibly difficulty with 'top' of performance, physical signs present but minor, or no interference with other uses of the hand. |
| Grade 3 | Pain persists away from the instrument, early involvement of other uses of the hand, possible loss of co-ordination or strength, physical signs with persistent tenderness of upper limb structures. Student under performs, orchestral player or concert artist has difficulty with high load. |
| Grade 4 | Pain at rest, pain at night, or both. Pain on most uses of the hand, writing, driving, housework, dressing, hair grooming, but capacity is retained so long as the pain is tolerated. Student is under performing, orchestral player is in difficulty with normal load.. Established physical signs and disablement. |
| Grade 5 | As for grade 4 but with loss of capacity for those hand-uses described in Grade 4. Gross physical signs, career stops or is seriously threatened.. |

The implications inherent in this system were the same as those of the staging system of Browne et al listed above (section 2.4.1) i.e. those with problems classified by this system were all affected by the one condition at varying degrees of severity and progression from less to more severe grades was to be expected if appropriate intervention were not taken.

2.5. Early History Of Occupational Upper-limb Pain In Australia

Awareness of occupationally associated upper-limb pain in Australia first appeared in an article by Perrott in 1961 [Perrott, 1961]. He described the problem with an approach to prevention based on a biomechanical model. He commented that his approach to management had not previously been applied to the occupational setting although it had been used in sports medicine. The same year Peres published an article, "Process Work Without Strain", [Peres, 1961], where he suggested that the problem resulted from chronic fatigue due to intense effort, monotony and lack of variety of work. He mentioned that static muscle loading and poor posture were of significant importance. Prevention based on

redesign of work practices, early reporting of symptoms and task alteration were recommended. The first population survey was published by Ferguson in 1971 when he studied musculo tendinous injuries in process workers. He found that the minority of cases were due to distinct clinical entities and that the majority consisted of poorly defined symptom complexes involving large areas of the upper limb. It is interesting to note that Welch, in 1972, stated that tenosynovitis was a problem in factories where light assembly work was carried out. He went on to say "the peak of the disease was reached in 1963-64. Since then remedial steps have been taken in most factories and the instance has decreased". Thus, it can be said that clearly this problem was not new to Australia in the '80s but that it was seen and recognised and dealt with, at least to some degree, in the past.

2.6. Historical Recognition Of Occupational Upper-limb Pain

Authors reviewing the past history of this condition have referred to literature over the past 300 years and equated descriptions of occupationally related upper limb pain with the phenomenon of RSI in Australia [Fry , 1986h][Quintner, 1989a].

The first reported cases were those of Ramazzini in the 18th century [Ramazzini, 1713]. Velpeau in 1825 again described this condition in association with writing [Velpeau, 1825]. Towards the end of the 19th century there was a considerable amount of literature by authors such as Poore and Gowers [Poore, 1873][Poore, 1878][Poore, 1887][Poore, 1890][Gowers, 1904] discussing limb pain associated with a number of occupations including telegraphy, writing and also with performance of music. Poore classified these types of occupationally related problems as "Fatigue diseases, in which the constant repetition of some muscular movement which constitutes the very essence of the trade or handicraft produces an inability to perform such movement."

During the 20th century, literature describing the condition of peritendinitis crepitans has also been equated with RSI. Peritendinitis crepitans was first labelled as such by Troell in 1918, again described by Howard in 1937 [Howard, 1937] and later by Thompson in 1951 [Thompson et al, 1951].

Although the descriptions of the problems in this historical literature certainly referred to upper limb discomfort associated with occupational activity, care should be taken before equating these problems with the phenomenon of "RSI" as it was described in Australia in the early 1980's. All that can really be said is that discomfort associated with repetitive or continuous occupational activity has been recognised for centuries.

2.7. International Recognition Of Occupational Upper-limb Pain

In the more recent international literature there are also a large number of articles describing activity-related upper limb discomfort. In this literature a variety of different terms have been used and this would seem to have confused the issue concerning whether the phenomenon which has occurred in Australia is in fact present in other countries. The Scandinavian [Waris, 1979a][Kvarnstrom & Hallden, 1983] and Japanese [Maeda, 1977][Aoyama, 1979][Matsumoto & Murakami, 1981][Miyame, 1982] literature has tended to use the term occupational cervico brachial disorder. The United States literature has used the term cumulative trauma disorder. [Armstrong et al, 1982][Fine et al, 1986][Silverstein et al, 1986]

In the late 1960s Japanese researchers began to investigate occupational cervico brachial disorder in a number of occupational groups including cash register operators, industrial workers, film rollers, creche attendants, nurses, keyboard operators, telephone operators and clerks. Their research was essentially descriptive and did not lead to the identification of a specific pathology. Management of the condition focused on prevention, with the identification of ergonomic improvements required in the work place. During the 1970s there was considerable work done in Sweden investigating what appears to be a similar condition. A survey of cashiers in supermarkets, done in Canada and reported in 1985, indicated that 62% of cashiers experienced general fatigue and 14.9% reported severe fatigue. An article in The Guardian of March 1985 reported a study of British Telecom workers in Cardiff where over 50% of workers suffered with discomfort associated with their activity.

Consideration of this international literature makes it clear that the phenomenon of occupational upper-limb pain associated with activity was not uniquely Australian as had been claimed by Awerbuch [Awerbuch, 1985].

2.8. Summary

In summary, the literature indicates that the problem of activity related pain is a major problem in Australia and overseas, and that the understanding of the condition is confused due to the variety and disparity of terminology used by different researchers. There is general consensus, however, that only a small percentage of cases are due to specific identifiable pathological conditions and that the majority of cases are due to a non-specific condition in which no previously understood pathology has been identified. Although this condition has been given a large number of different labels, the term Non-specific Occupational Overuse Syndrome has been chosen for the purposes of this thesis. There is evidence that epidemics of this condition (temporary marked increases in incidence) have occurred at different times in different countries but that these have occurred in the context of an endemic base line incidence. It is also accepted that established cases provide a major management problem and that recovery from the condition in such cases is slow.

Authors from all points of view have recognised that, when assessing apparent non-specific upper-limb pain the differential diagnosis, including all forms of Specific Occupational Overuse Syndrome, should be carefully considered and adequate history and examination with appropriate investigations should be performed. If the diagnosis of Non-specific Occupational Overuse Syndrome (by any name) were made too readily, no matter which pathology was assumed, there would be potential for delay in making the correct diagnosis and consequently delay in providing the most appropriate treatment.

Beyond this basic level of agreement there is, however, a marked disparity of opinion regarding the nature of the Non-specific Occupational Overuse Syndrome. As is to be expected, where there are a number of conflicting theories, there are also a number of features of the condition which are accepted by some and rejected by others. Although some researchers suggest that the non-specific form of Occupational Overuse Syndrome is simply a collection of yet to be defined different conditions, the majority of those who have investigated the condition accept that it is, in fact, a single process but debate the nature of this process. There is considerable disagreement about whether or not there are, in fact, consistent physical features on the basis of clinical examination. Some writers say that the clinical findings are inconsistent [Lucire, 1986], others however, express the opinion that, although the clinical features do not suggest previously described pathology, they are internally consistent and demonstrate the presence of the physical process [Fry, 1986a]. Yet another group argues that the condition occurs in association with work dissatisfaction and boring jobs and that this is in fact a consistent feature [Ireland, 1986a]. There is also considerable debate over whether the condition can truly be said to be associated with excessive activity.

The range of theories proposed cover the range of possible anatomical sites which might cause pain anywhere from the peripheral tissues involved in activity to the psyche. Heated debate has been generated between the advocates of the various theories. The fiercest controversy can be seen between what appears to be two factions. These two factions can be identified according to the focus of their theories and amount to those arguing for and those arguing against the presence of physical pathology. The site of pathology for the physical pathology theories is in either the muscle tendon unit, in the neural structures of the upper limb or was due to an aberration of the central pain control mechanisms (fibromyalgia theory). The “no physical pathology” faction includes those favouring the psychological theories of conversion hysteria, compensation neurosis, and malingering (conscious pretence of pain) and those advocating various expressions of normal physiological fatigue as the source of symptoms. It is interesting to note that this battle line also equates with the medico-legal battle line of employer versus employee. Can this diversity of opinion and strength of feeling expressed in the context of a logical scientific debate be ascribed solely to the conflict between employer and employee? Consideration of Kuhn’s treatise on “The Structure of Scientific Revolutions” [Kuhn, 1970] allows an alternative or perhaps additional explanation for this.

Kuhn studied historical examples of scientific periods where a particular phenomenon did not have a widely accepted explanation. He described such a period as one where there was no accepted paradigm within which to operate. Research during such a period he has called “pre-paradigm” research. He found that the social interactions between researchers and the methods they could employ at those times were significantly different from later periods when some specific explanation has been accepted by the scientific community as a whole. The research is then being undertaken from within a defined paradigm.

The state of scientific knowledge in relation to Non-specific Occupational Overuse Syndrome remains at the stage which Kuhn would label as “pre-paradigm”, where no single theory has yet been widely accepted to explain the condition. He states that in assessing pre-paradigm research “though the field’s practitioners were scientists, the net result of their activity was something less than science. Being able to take no common body of belief for granted, each writer felt forced to build his field anew from its foundations.” “Early fact gathering is a far more nearly random activity than the one that subsequent scientific development makes familiar”, that is, after a single paradigm has become established.

Kuhn’s description of pre-paradigm research seems to closely describe current research into occupational overuse syndrome. This is not at all a criticism of the work involved in the current research into Non-specific Occupational Overuse Syndrome but more an attempt to understand the process which is taking place. When no individual theory proposed to explain a phenomenon has been accepted by the body of scientists working in that area, then research must be exploratory rather than focused. History has shown that in this situation numerous contradictory hypotheses will be developed with associated heated scientific debate. This is, thus, an explanation for the diversity of ideas as well as for the heated discussion observed in the medical literature.

From this perspective it can be seen that research into Non-specific Occupational Overuse Syndrome continues in a context of what might be termed epistemological indeterminacy - the precise nature of the phenomena involved, the conceptual categories required and what constitutes evidence and adequate explanation all remain as yet uncertain and contested. In this context, it is often necessary to weigh up the epistemological status of the presuppositions, claims and conclusions of other researchers, to attempt to determine how they are derived, by what evidence they are supported and how thoroughly they have been tested.

The notion of “normality” or the “normal” is one of the concepts put at issue by the Occupational Overuse Syndrome literature. The point at which the line should be drawn between “normal” populations and those seen as “affected” by the overuse syndrome is one area of controversy and theoretical contestation. Care should be taken, therefore, to define specifically what is meant by “normal” in each instance and to refer the usage back to the basic

sense of conformity with, or non deviation from the regular, the usual or the standard.¹ A number of authors use assumptions about what constitutes normality in order to draw conclusions about the nature of the pathology, as it is seen to affect those who are taken to be suffering from non-specific upper-limb pain.

Sinclair stated that “Pain that lasts for months or years, which is unremitting and refractory to all forms of treatment and is unaccompanied by any sign whatever, simply cannot be arising from muscles or tendons.” Doctors “must not be afraid to state that no diagnosis is possible if the signs and symptoms do not make normal medical sense.” [Sinclair, 1985, November]

In their book “Myofascial Pain and Dysfunction”, Travell and Simons make a direct statement of their basic assumption of the nature of “normal” muscles. “Normal muscles do not contain Trigger Points. Normal muscles have no taut bands of muscle fibers, are not tender to firm palpation”. [Travell and Simons, 1983a]

Fry in his description of “Physical signs in the hand and wrist seen in the overuse injury syndrome of the upper limb” states “In the normal uninjured hand, squeezing the intrinsic muscles between the surgeon’s thumb and fingers is not painful.” [Fry, 1986a]

The next chapter describes the development of a study designed to collect data with which a set of hypotheses selected from those implied by the current theories of pathology can be tested, using systematic collection of clinical information from a set of patients and controls.

1. Normal : constituting, conforming to, not deviating or differing from a type or standard, regular, usual.

(Shorter Oxford English Dictionary)

Chapter 3. Methods

3.1. Study type.

At the time when this study was being designed, the nature of the Non-specific Occupational Overuse Syndrome had for a number of years been the subject of a wide ranging debate. The theories regarding its pathophysiology were, and still remain, as different as the proposed methods of treatment. The state of scientific knowledge in relation to Non-specific Occupational Overuse Syndrome was, and indeed remains, at the stage which Kuhn [Kuhn, 1970] would label as “pre-paradigm” where no single theory has yet been widely accepted to explain the condition. Kuhn argues that when no individual theory proposed to explain a phenomenon has succeeded adequately to convince the body of scientists working in that area, then research must be exploratory rather than focused.

In 1986, when this study was set up to investigate Non-specific Occupational Overuse Syndrome, none of the current theories seemed particularly compelling, as would be expected from an analysis of the literature in a “pre-paradigm” period. The aspects of the debate which did seem to be worth investigating were:

1. The diversity of theories.
2. The absence of studies looking at features which were considered to be characteristic of Non-specific Occupational Overuse Syndrome in normal / unaffected individuals.
3. The absence of prospective studies.

Consequently, this study was designed to allow a wide ranging exploration of the evidence available from routine clinical assessment and to discover from this whether the basic methods of medical practice, when applied systematically, would provide any evidence for or against the various theories which have been proposed. It was decided that a suitable group of normal individuals should be included to permit the evaluation of the normal characteristics of such features as tenderness and grip strength, and to establish a prospective component to the study. This would allow some assessment of the risk associated with features considered to be aetiologically important.

3.2. Aims

3.2.1. Primary Aim.

The aim of the study was to investigate the previous theories , to either confirm the validity of one or more of them or, by means of this testing process, develop an alternative theory.

3.2.2. Secondary Aim.

The secondary objectives were to discover whether or not any of the clinical variables collected as part of the study had any value in predicting who was likely to develop the Non-specific Occupational Overuse Syndrome and to develop a systematic approach to its management.

3.3. Hypotheses to be tested.

(These were tested individually and were not necessarily considered to be mutually compatible)

From the various assumptions and theories concerning the Non-specific Occupational Overuse Syndrome which were extracted from the literature, the following set of hypotheses was selected for the investigation.

1. That the musculo-skeletal structures of normal individuals are non-tender.
2. That the musculo-skeletal structures of normal individuals are not painful.
3. That activities associated with higher levels of upper-limb activity are associated with a higher prevalence of symptoms consistent with a diagnosis of Non-specific Occupational Overuse Syndrome .
4. Change in activity levels increases risk of Non-specific Occupational Overuse Syndrome.
5. That routine clinical examination is able to discriminate, on the basis of the presence of tender sites, between those with Non-specific Occupational Overuse Syndrome and those not affected.
6. That the majority of cases of Non-specific Occupational Overuse Syndrome are caused by a single process.
7. Presence of tender sites increases the risk of later development of Non-specific Occupational Overuse Syndrome.

8. "Inconsequential" symptoms increase the risk of developing "Consequential" symptoms of Non-specific Occupational Overuse Syndrome.
9. Increased physical fitness decreases risk of Non-specific Occupational Overuse Syndrome.
10. Certain physical attributes increase risk of Non-specific Occupational Overuse Syndrome.
11. High Activity levels increase risk of Non-specific Occupational Overuse Syndrome.
12. Psychological tendencies increase risk of Non-specific Occupational Overuse Syndrome.

3.4. Study setting.

This study was undertaken within the Royal Adelaide Hospital Orthopaedic Hand and Upper-limb Clinic in association with the University Health Service at the University of Adelaide. Subjects for study were taken from the normal working activity of these two clinical areas. Prior to 1988, at the time that the initial data was collected, the University of Adelaide provided a free health check at its University Health Service for all first year students. The aim of the health check was twofold.

- Firstly, educational to inform students of the different medical, counselling and sporting facilities available in the university and to discuss the possible health hazards associated with their specific disciplines.
- Secondly, as a screening process directed towards the preventive goal of identifying students with either pre-existing conditions or risk factors, who might have benefited from early intervention to prevent later problems.

The health check consisted of detailed history and physical examination according to a set protocol and a series of standardised psychological screening questionnaires. For the purposes of this study, a systematic examination for sites of upper limb tenderness was added to the routine physical examination.

Those students who presented for this routine health check and who could be considered "normal individuals" in that they did not have symptoms associated with activity of any consequence, were allocated to the control group for the purposes of the Cross-sectional and Case-control parts of the study. The music students from this control group were utilized for the longitudinal prospective part of the study. Subjects classified as having Non-specific Occupational Overuse Syndrome were taken from patients who presented with activity related upper-limb problems to either the Orthopaedic Hand and Upper-limb Clinic or the University Health Service. The patients who presented to the University Health Service and whose problems were not solved within the first 2 to 3 visits were referred to the Orthopaedic Hand and Upper-limb Clinic for continued treatment under specialist supervision.

3.5. Populations studied

All Instrumental Music Students and a random selection of students from other disciplines, presenting over a two year period for the routine health check, were assessed. The Music Students were followed up during the subsequent two years. This provided a suitable sample from which the prevalence of the various sub-classifications of Occupational Overuse Syndrome could be estimated for a population of first year university students, comparing musicians with non-musicians.

The prevalence of “Consequential” symptoms was not expected to be high in the first year university student population and so all individuals who presented at either the University Health Service or the Royal Adelaide Hospital Orthopaedic Hand and Upper limb Clinic, seeking treatment for symptoms consistent with the Occupational Overuse Syndrome, were assessed for the study over a four year period. This increased the number of subjects with “Consequential” symptoms which were included in the study, enabling statistical comparisons to be made between individuals with different sub-classifications of Non-specific Occupational Overuse Syndrome .

Three populations of subjects could be identified:

1. A cohort sample consisting of two years of undergraduate instrumental music students

Population 1 = Normal Music Students (NMS n=95)

An attempt was made to collect data over a two year period from every undergraduate music student who was enrolled for the first time at the University. All health checks for music students were used in the study. Seven (7) students chose not to have the health check.

2. A sequential sample of 53 students who did not play musical instruments.

Population 2 = Normal students from Other Faculties (NOF n=53)

The author also worked as one of the doctors in the University Health Service and took his share of routine health checks for students from faculties other than music. A sequential sample from these health checks provided “normal” individuals which were used as a second control group for comparison.

3. Patients who sought treatment for upper-limb discomfort related to activity during the study period.

Population 3 = Patients classified as suffering from isolated Non-specific Occupational Overuse Syndrome (Non-specific Occupational Overuse Syndrome with “Consequential” symptoms n=29)

During the study period the author had a clinical commitment to both the University Health Service and the Orthopaedic Hand and Upper-limb Clinic, with a share of the clinical work load under the supervision of the director of the University Health Service and the senior consultant

at the Orthopaedic Hand and Upper-limb Clinic respectively. It was the author's specific responsibility to assess and treat those patients who presented with activity related pain. Those who were classified as suffering from isolated Non-specific Occupational Overuse Syndrome were included in this population group. Twenty seven (27) patients with activity related upper-limb pain were found to have specific diagnoses.

3.6. Time constraints

This setting imposed certain constraints on the study.

As the data was collected during the normal clinical sessions of both the University Health Service and the Orthopaedic Hand and Upper-limb Clinic there was a time constraint limiting both the range of information and the detail that could be collected during the assessment of subjects.

Time constraint occurred for the following reasons.

1. The University Health Service "health check" was based on a standardised form in routine use by the University Health Service for several years {see appendix A}, and was expected to take approximately 20 minutes to administer. This was the health check which had to be used for Population 2. The only additional information relevant to this study which could be collected within this frame-work was a systematic examination for tender sites in the upper-limbs.
2. The standard health check had been modified in the past for other faculties such as medicine and dentistry and so specific modifications particularly relevant to the music faculty were considered appropriate {see Appendix B and C}. A time constraint of 40 minutes was set to permit these added modifications. This was the maximum length of time considered reasonable for both the students expectations and the normal flow of clinical work within the University Health Service. This was instrumental in limiting the choice of the information which could be collected for Population 2.
3. The patients who made up Population 3 were assessed as part of the normal clinical proceedings of the University Health Service and the Orthopaedic Hand and Upper-limb Clinic. Both areas had a high clinical work load and so assessment was limited to that appropriate for accepted clinical practise of these areas. The only addition to this was a systematic examination for tender sites in the upper-limbs.

3.7. Research designs.

The study was undertaken in three phases.

1. A cross-sectional analysis of first year university students.

The study selection procedure meant that among the subjects were a large number of Instrumental Musicians. Consequently, prevalence comparisons between the occupational categories of Musician and Non-musician, as well as between the three categories of Non-specific Occupational Overuse Syndrome, was possible. It also allowed the distribution of values for the parameters which were considered to be of importance in the assessment of the Non-specific Occupational Overuse Syndrome to be defined for an unaffected or "normal" population.

The hypotheses 1 to 3 above were tested by this part of the study.

2. A case-control analysis including all subjects assessed.

By including a "patient" group, comparison of the differences between cases and appropriate controls was possible.

The hypotheses 4 to 6 above were tested by this part of the study.

3. A Cohort prospective analysis of a subgroup of those initially assessed.

The Instrumental Music Students who did not have "Consequential" symptoms at initial assessment were followed prospectively for three years to identify differences between those who subsequently developed "Consequential" Symptoms and those who did not.

Previous research suggested that the incidence of Non-specific Occupational Overuse Syndrome among music students was between 10 and 30% (Fry, 1985, personal communications). It was for this reason that this select population of students was chosen for a longitudinal study. They were assessed at the beginning of the course and again at the end of the third and final year of their undergraduate studies.

Because all of the students in the music faculty were offered a special health check specifically modified to be relevant to musicians, it was possible to include the detail necessary for this longitudinal part of the study. All the music instrumental students in the study were also asked to attend an interview and examination at the end of their third year. The goal of the Followup assessment was to determine which of the students had developed Non-specific Occupational Overuse Syndrome during their three years of study.

The hypotheses 7 to 12 above were tested by this part of the study.

3.8. Research procedures.

3.8.1. The standard University Health Service health check

The standard University Health Service health check was used as the basis for all students in this study. See Appendix A

Preliminary Introduction and Familiarisation

When students arrived at the University Health Service for the health check they were given literature containing information concerning the university facilities and various topics of preventive health such smoking, diet, exercise, breast self examination etc.

Nursing Assessment:

They were next seen by the nurse who took an immunisation history and took some basic physical measurements: Weight, height, Visual acuity, Colour vision, Urinalysis.

Medical Assessment:

The interview and physical examination by the doctor followed. This included:

- History: A general discussion of the services provided by the university was followed by a structured medical, family, social and occupational/educational history.
- Physical Examination: A standard screening physical examination was performed.
- Investigations: Chest X-ray/ECG etc were performed if indicated.
- Debriefing: A discussion of general health matters of particular relevance to each student followed. Topics included, diet, smoking, drugs, contraception, cervical smear tests, self breast examination, physical exercise.

If any abnormality was found during the health check it was discussed with the student. Follow-up was offered either by further review at the University Health Service, by referral to the student's GP or to a specialist as selected by the student. A consultant physician/cardiologist and a consultant psychiatrist attended the University Health Service each week for referral as appropriate.

3.8.2. Modification of the Health Check Non-Music Students

The following modifications to the standard University Health Service health check were made as part of the medical assessment.

- History: Details of activity related problems were avoided at this time.

- Physical Examination: A number of specific details were added to this: Assessment of posture and hypermobility. A systematic examination for sites of tenderness of the upper-limbs.
- Debriefing: Following the physical examination a structured questionnaire was given to discover any history of activity related problems.

3.8.3. Modification of the Health Check for Music Students

The standard University Health Service health check was redesigned specifically for the purposes of this study and also to cover other aspects of preventive medicine of particular relevance to musicians. See Appendix B and C The assessment followed this altered format.

Additions to the standard University Health Service health check used for this group.

Preliminary Introduction and Familiarisation

On arrival the student was also asked to fill in a set of psychological questionnaires.

The General Health Questionnaire (GHQ) assessing neurotic tendencies [Goldberg, 1972]

The "Trait" section from the Spielberger State Trait Anxiety Inventory (STAI) assessing tendency to be anxious [Spielberger et al, 1970]

The Illness Behaviour Questionnaire (IBQ) assessing aspects of illness behaviour [Pilowsky & Spence, 1983]

Nursing Assessment:

When seen by the nurse an Audio-gram was also performed.

Medical assessment:

The interview and physical examination by the doctor was modified as follows:

- History: Details of activity related problems were avoided at this time.
- Physical Examination: A number of measurements were added to the standard examination: Assessment of posture and hypermobility. A systematic examination for sites of tenderness of the upper-limbs. Hand Span, thumb tip to little finger tip. Hand grip strength (Jamar dynamometer) and pinch strength (Martin Vigorimeter). Dexterity Test (Maximal tap rate). Aerobic fitness V O₂ max. (McArdle Step Test) [McArdle, 1973]
- Investigations: Chest X-ray/ECG etc were performed if indicated.
- Debriefing: Following the physical examination a structured questionnaire to discover any history of activity related problems was then given.

3.8.4. Assessment of patients.

Routine medical history and physical examination appropriate to the patient's problem was taken. In addition, a systematic examination for sites of tenderness of the upper-limbs was performed. Investigations performed as appropriate included: CBP, ESR, Rh Factor, ANF/Anti DNA, Plain X-ray, Fluoroscopic Controlled Arthrogram, EMG/NCS.

3.8.5. Followup assessment of Music Students.

An attempt to reassess all the music students by personal interview at the end of three years of study was made. Only 2 who had moved interstate were unable to be contacted. If it was not possible to see them in person an attempt to interview them by phone was made. The primary goal was to discover which of the students had developed activity related symptoms during their studies. The secondary goal was to collect additional data which might throw further light on the hypotheses being tested.

Students were contacted by phone. They were told that the University Health Service were conducting a survey investigating the types of medical problems that affect music students and asked to attend an interview. If they were living too far from the University ie. inter-state/overseas or if they were unwilling to attend in person they were asked if they would mind answering a few questions by phone.

3.9. Measures taken to reduce assessor bias.

1. Initial assessment of populations one and two (the control subjects):

- A standard history was taken, however, questions relating to activity related symptoms were initially avoided.
- Complete physical examination, including examination for tender sites, was performed, after which a series of standard questions relating to activity related symptoms was asked.

The modification of the routine process of history and examination was made to minimise potential assessor bias during physical examination. It was considered possible that knowledge of activity related symptoms may have affected the assessor's expectations during physical examination.

2. Followup assessment of music students:

The data collected during the initial assessment was not collated or analysed until after the followup assessment was made. In this way the potential effect of knowledge of the initial values of variables on decisions of followup classification would be minimised.

3.10. Definition of Outcome Variable

At each assessment, subjects were allocated to one of the diagnostic categories of Occupational Overuse Syndrome as defined in this section. Thus, the primary outcome variable of this study is this diagnostic classification of the Occupational Overuse Syndrome.

It is important to note the differences between the system of classification defined and used in this study and the systems found in the existing literature. The sub-categories formulated in previous research have all applied to subjects assumed to have Occupational Overuse Syndrome, they did not include asymptomatic individuals. The sub-categories organised subjects into different groupings according to severity of Non-specific Occupational Overuse Syndrome. Their classifications relied, at least in part, on some "semi-objective" assessment of pain, whether in terms of persistence, distribution (the number of sites) or severity. Such a pain-focussed system of classification was rejected for the purposes of this study as it was considered unnecessarily complicated and conceptually problematic to attempt to assess differences between individuals with varying degrees of severity of symptoms. Instead it was decided to simply investigate differences between patients and controls i.e. between sufferers and non sufferers. It was hoped thereby to avoid the problems associated with a system of classification which relied on largely subjective parameters. Consequently an attempt was made to define an outcome variable which was as precise as possible and which did not rely on an assessment of the severity of symptoms.

A diagnostic process consisting of a three-step system of sub-classification was developed.

- Firstly, the division was between those with symptoms and those without. That is, subjects were divided into those who reported experiencing discomfort in association with activity and those who reported no such discomfort. This made the division between unaffected individuals and those with Occupational Overuse Syndrome. This diagnostic step, although obvious, was a necessary part of the process, as sampling was taken from a population of individuals independent of the presence of symptoms.
- Secondly, the group reporting symptoms was divided into those for whom some specific diagnosis could be made - i.e. rheumatoid arthritis, triangular fibro cartilage tear at the wrist - and those for whom no diagnosis could be made. This diagnostic step produced the division of Occupational Overuse Syndrome into Specific Occupational Overuse Syndrome and Non-specific Occupational Overuse Syndrome. This part of the classificatory process reiterates the fact that the diagnosis of Non-specific Occupational Overuse Syndrome is a diagnosis of exclusion, that is, it is the diagnosis which is applied when all specific pathological causes of Occupational Overuse Syndrome have been excluded.

- Thirdly, those with Non-specific Occupational Overuse Syndrome were divided into two categories. The reasons for and the details of this final sub-division were as follows.

A number of previous researchers, attempting to clarify the features of the Non-specific Occupational Overuse Syndrome, have proposed systems for staging or grading severity [Browne et al, 1984][Fry, 1986b]. All of these systems have, however, proved to be in some way problematic or controversial, being criticized by other authors [Wright, 1987] because they were based on symptoms and signs rather than on objective measurements or tests, or because unproven prognostic value had been placed on the varying levels of staging.

The initial intention in this study was to avoid the use of any form of subdivision of subjects classified as having Non-specific Occupational Overuse Syndrome and to simply analyse differences between affected and unaffected individuals. However, when collecting data early in the research, it became apparent that there were compelling grounds for making at least a two-way subdivision of those subjects who were in some way affected.

The research subjects came from two sources: patients who presented to the authors seeking treatment for pain associated with activity and a control group made up of students who presented to the University Health Service for the free first year preventive health check-up. Obviously, the first group of “patients” would be classified as having Occupational Overuse Syndrome since the syndrome was defined on the basis of the presence of symptoms. But it was soon discovered that a subsection of the control group also reported having experienced discomfort or pain associated with activity. There was, however, an important feature separating the majority of these control-group individuals from the “patients”.

Careful questioning of the majority of those from the control group who did report symptoms, indicated a significant difference in the effect of those symptoms from the effect of the symptoms reported by the patient group. The critical feature of this difference was that the individuals in the control group who had symptoms found those symptoms to be of no consequence in that they had felt neither the need to alter their activities in any way nor to seek treatment from any third party such as a doctor or physiotherapist.

Since Non-specific Occupational Overuse Syndrome was defined simply by the presence of symptoms, both the “patients” and this section of the control-group of individuals fell under this broad heading. But there were clearly significant differences between the “patients”, those who had felt their symptoms to be of consequence and had accordingly altered their activities and sought medical intervention and the those members of the control group with symptoms but who had not been affected by them. The stark difference between the effect of symptoms on these two groups strongly suggested a difference between them which should not be ignored by the research design and indeed should be investigated. It seemed likely that the major difference between the two groups would be the severity of the symptoms, although it was also

considered possible that this might not be the only difference. The criteria which defined the two groups did not, however, depend on any assessment of severity. Certainly the group of individuals who had symptoms which were of no consequence would not have been observed by clinical studies in which patients are assessed. This group would only be assessed by studies involving a survey of the wider population in which subjects are assessed independently of any perceived need for treatment.

Accordingly, the final diagnostic step divides cases of Non-specific Occupational Overuse Syndrome into two categories, with the division being made on the basis of the effect of the symptoms on the lives and activities of subjects reporting some degree of activity-related pain. To make this step of the diagnostic classification of Occupational Overuse Syndrome explicit and systematic, symptoms associated with Non-specific Occupational Overuse Syndrome were defined as either "Inconsequential" or "Consequential". "Inconsequential" symptoms were those which were of no consequence in that they did not cause the individual to seek intervention from a third party such as a doctor or physiotherapist, nor did the symptoms affect their activities in any way. "Consequential" symptoms were defined as being of some consequence by either inducing the individual to seek help or by affecting the activity involved in some way. Thus, the third diagnostic step in the classification of Occupational Overuse Syndrome involved the division of individuals classified as Non-specific Occupational Overuse Syndrome into two further sub-categories according to whether their symptoms were "Inconsequential" or "Consequential".

To avoid any ambiguity, a precise definition of "Inconsequential" and "Consequential" symptoms was established as follows:

Non-specific Occupational Overuse Syndrome with "Inconsequential" Symptoms

Individuals who reported symptoms of pain or discomfort associated with activity for whom no diagnosis had been made and for whom the symptoms were of **no consequence** were defined as having "Inconsequential Symptoms".

"Symptoms of no consequence" was defined to mean that both of the following occurred.

- The experience of symptoms did not result in a change of the normal routine of activities - the symptoms did not interfere with the continuation of activity.
- As a result of experiencing the symptoms the individuals did not seek intervention from any third party such as a doctor or physiotherapist.

Non-specific Occupational Overuse Syndrome with “Consequential” Symptoms

Individuals who reported symptoms of pain or discomfort associated with activity for whom no diagnosis had been made and for whom the symptoms were of **some consequence** were defined as having “Consequential Symptoms”.

“Symptoms of some consequence” was defined to mean that at least one of the following occurred

- The experience of symptoms did result in a change of normal routine of activities - the symptoms did interfere with the continuation of activity.

or

- As a result of experiencing the symptoms the individuals did seek intervention from a third party, such as a doctor or physiotherapist.

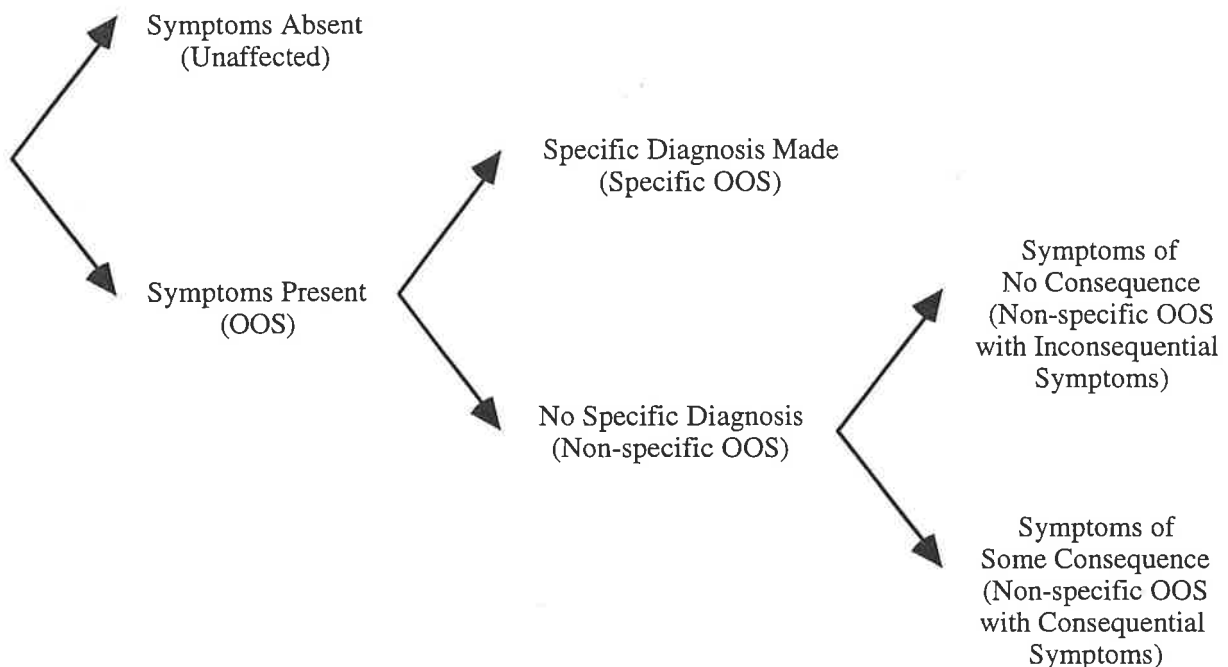
This final category, Non-specific Occupational Overuse Syndrome with “Consequential” Symptoms, roughly equated to all the subjects considered and sub-classified in previous research, since these researchers had assumed they were classifying people with disease. That is, they were sub-classifying people who had presented as patients or had been forced to modify their normal activity in some consequential way and hence would meet the criteria for inclusion in the “Consequential Symptoms” category in this study.

The study departs from, or at least extends, the previous research by including the “Inconsequential Symptoms” category. Members of this group reported symptoms typically associated with Non-specific Occupational Overuse Syndrome but had not been consequentially affected, in the sense defined above, by those symptoms. It is not clear whether previous researchers would have classed this group with “Inconsequential” symptoms as having Non-specific Occupational Overuse Syndrome. They have either failed to mention this group or remained ambiguous as to whether their categories included such subjects.

Both the “Inconsequential” and “Consequential” diagnostic categories were non specific - by definition there were no objective investigations for determining whether or not the subjects had a disease. Similarly, no investigations were available for determining degrees of severity of the disease which might have provided a basis for distinguishing the two categories. However, the different ways in which they responded to their symptoms, set out above, did provide a clear basis for establishing a subdivision and these categories operated accordingly. Such a difference should be important in terms of medical assessment because it would necessarily influence approaches to management. In particular, it would not seem appropriate to undertake invasive investigations for those subjects who were not affected by their symptoms. Consequently, it appeared necessary to explore systematic differences between these two categories, and the decision was made to do so by means of clinical assessment. The primary focus of the study was, however, an investigation of the differences between asymptomatic individuals and symptomatic individuals, that is, the differences between those in diagnostic

category "No symptoms" and those with the diagnosis of Non-specific Occupational Overuse Syndrome. With this subdivision of the diagnosis Non-specific Occupational Overuse Syndrome the study became an investigation comparing the three diagnostic categories, No symptoms, "Inconsequential" symptoms and "Consequential" symptoms. The study thus became defined as an investigation as to whether or not there were patterns of variation between these three categories in terms of symptoms, physical findings and psychological factors. It was not assumed, however, that there was an ordinal relationship between these three categories as it was considered important for the analysis to allow for the possibility that there were complex interactions occurring between factors, leading to the inclusion of an individual into one or other category. The fourth diagnostic category, Specific Occupational Overuse Syndrome, was used as the principle exclusion criterion.

The diagnostic algorithm developed for the classification of Occupational Overuse Syndrome (OOS) is thus presented in graphical form.



This produced four possible diagnostic categories as follows:

Diagnostic categories for Occupational Overuse Syndrome:

0. No symptoms / Unaffected individuals: those who denied ever having pain/discomfort associated with upper-limb activity.
1. Non-specific Occupational Overuse Syndrome with "Inconsequential" symptoms: {These individuals could be considered "normal" in that the symptoms they experienced did not cause any functional limitation, nor was there psychological perception of a physical problem causing them to change their activities. On the other hand they could be considered "abnormal" in that they did experience symptoms associated with activity.}
2. Non-specific Occupational Overuse Syndrome with "Consequential" symptoms:
3. Specific Occupational Overuse Syndrome: those who reported pain/discomfort associated with upper-limb activity in whom a specific diagnosis of the cause of symptoms could be made.

It can be seen that this diagnostic classification did not completely overcome the problem of differentiating between Specific Occupational Overuse Syndrome and Non-specific Occupational Overuse Syndrome in an epidemiological survey. It was likely that in some of the individuals in categories 1 and 2 a specific diagnosis could have been made if appropriate investigations were performed. Use of investigations for individuals in category 1, whether invasive or not, was not justifiable and so some potential loss of specificity in classification had to be accepted.

The aim of this study was to investigate the nature of the non-specific form of Occupational Overuse Syndrome. Only in category 3, Specific Occupational Overuse Syndrome, was a specific diagnosis present and so individuals classified as diagnostic category 3 were excluded from the analyses. The other three categories (0 to 2) provided the basis for comparative analyses of different categories of Non-specific Occupational Overuse Syndrome, that is, "No Symptoms", "Inconsequential Symptoms" and "Consequential Symptoms". Thus the outcome measure for this study was the classification into one of these diagnostic categories. Those classified as diagnostic category 3 (Specific Occupational Overuse Syndrome) were excluded.

3.11. Inclusion and exclusion criteria.

3.11.1. Population 1, "Music Students".

All undergraduate music students enrolling for the first time at the University over the specified two year period were eligible for inclusion. The students from this group who were excluded from this population for all parts of the study were :

- Those who chose not to have the University Health Service "health check" n=7
- Administrative problems caused the failure to collect some study variable for a few individuals. Missing data due to this reason is detailed in Appendix G along with the relevant analysis.
- Those who presented with a previous medical history of upper-limb dysfunction which consequentially interfered with activity and where a specific diagnosis had been made, that is individuals classified as Consequence of Problem = 3. The presence of a specific diagnosis excluded them from consideration in the spectrum of individuals who could be considered as having isolated Non-specific Occupational Overuse Syndrome. Differences from the control group of individuals would be likely to be due to their particular specific diagnosis. Excluded n=2

Total excluded from analysis = 9 out of 102 instrumental music students.

For the purposes of the prospective arm of the study, 2 further groups of students were excluded.

- Those who would have been included but could not be contacted at the end of the course to enable a followup assessment to be made. None of the students who were able to be contacted declined to be assessed. Excluded n=2.
- Those who presented with a previous medical history of upper-limb dysfunction which consequentially interfered with activity and with no specific diagnosis, that is individuals initially classified as Consequence of Problem = 2. These could not be considered “normal” subjects at the commencement of the study. Excluded n=6

Total additional music students excluded from the prospective analysis = 8 instrumental music students.

3.11.2. Population 2. "Non-Music Students "

During the second year of the study the administration of the University of Adelaide decided to stop providing the free “health check” for first year students. Students from faculties other than music who were seen by the author for their health check up till this time were eligible for inclusion. If there had been students from this group with Consequence of Problem = 3 they would have been excluded.

Excluded n=0 out of 53

3.11.3. Population 3, patients with activity related upper-limb pain.

Patients who presented to either the University Health Service or the Orthopaedic Hand and Upper-limb Clinic with upper-limb discomfort related to activity were assessed for the study and were included in this group. Individuals in this population were necessarily classified as Consequence of Problem = 2 or 3. Those classified as Consequence of Problem = 3, that is those who were given a specific diagnosis as the cause of their problem, were excluded from the analyses. Excluded n=27 out of 56

3.12. Measures of study variables.

The standard clinical history and examination, as described in Macleod’s Clinical Examination [Munro & Edwards, 1990], was used as the basis of the clinical assessment. A standard form was used during the assessment to ensure collection of certain specific information. Appendix B and C

Self assessment questionnaires were used for assessment of psychological profiles. Appendix D, E and F.

Assessment of sites of tenderness was based on standard techniques for the digital palpation of tenderness. Standard clinical examination only recommends that symptomatic areas, and not

asymptomatic areas, be examined for tenderness. As the presence of sites of tenderness was considered by some authors to be of major significance, it was decided to develop a standardised protocol for its assessment. Thus routine assessment of sites of tenderness in symptomatic areas was extended to a systematic assessment of tender areas of the upper-limbs, irrespective of the presence and distribution of symptoms. The protocol developed for this study was based on the system of examination for tenderness of the upper-limb developed by Fry (Fry, 1985, Personal communications) and presented in part in the Australian New Zealand Journal of Surgery [Fry, 1986a].

3.12.1. The protocol for assessment of tenderness

The protocol involved a list of sites which were systematically examined in all patients at a standard pressure which was applied to all sites by digital palpation to illicit tenderness.

The list of areas and associated sites with sequence of examination was as follows:-

Right & Left.

Hand & Wrist

4th Inter-digital Cleft
 3rd Inter-digital Cleft
 2nd Inter-digital Cleft
 1st Inter-digital Cleft
 Thenar Eminence
 Hypo-thenar Eminence
 Carpo- metacarpal jnt thumb
 Radial Wrist Ligament
 Ulna Wrist Ligament
 Dorsal Wrist Ligament
 Palmar Wrist Ligament.

Forearm & Elbow

Common Extensor Muscle Belly
 Common Flexor Muscle Belly
 Common Extensor Muscle Origin
 Common Flexor Muscle Origin

Upper-arm

Biceps Muscle Belly
 Triceps Insertion

Shoulder

Coracoid Process

Neck

Superior Trapezius

A total of 19 sites in 5 areas on each side, giving a total of 38 possible tender sites in 10 areas for each individual.

The protocol included initial training of the medical assessors with a pneumatic pressure gauge, the "Martin Vigorimeter", so that they were able to consistently exert pressure to a maximum of 30-40 KPa on digital palpation. Examination for tenderness at each site was performed with digital palpation in an area including the whole structure in question. The site was palpated initially with minimal pressure which was rapidly increased until either the patient showed signs of discomfort or to a maximum of 30-40KPa which ever occurred first. Subjects were asked to indicate if they experienced discomfort during any of the examination and in particular if one

side were more sensitive than the other. If tenderness were found at any pressure from the lightest of touch to the full 30KPa-40KPa that site was recorded as a tender site. With experience, this examination took less than 5 minutes.

Having defined the protocol to permit the assessment of this variable (presence and number of upper-limb tender-sites) it was necessary to determine whether or not the variables so measured had adequate reliability and reproducibility to permit statistical analysis. Subsequent to the commencement of this study, other authors have confirmed that assessment of sites of tenderness can be shown to demonstrate acceptable inter and intra rater reliability [Reeves et al, 1986][Fischer, 1988][Stockstill, 1989a].

3.12.1.1. The reliability of the assessment of tenderness.

After the protocol had been developed by Dr. John White (JW) specifically for the study, it was taught to a colleague at the University Health Service (Dr. TT). It was then used for several months as part of their routine physical examination of patients, in order to gain familiarity with the technique.

The study of reliability was performed on a group of 25 volunteers. Each subject was examined during a morning, once by Dr. White and once by Dr. TT in random order. Each subject was examined again that afternoon by Dr. White. Inter-observer reliability was tested comparing Dr. White's observations with those of Dr. TT and intra-observer reliability was tested comparing Dr. White's morning and afternoon observations.

Results

Agreement as to presence or absence of tenderness at a site was compared with the KAPPA statistic [Wright & Feinstein, 1992]. A total of 950 sites (25 patients with 38 sites in each) were examined during each assessment. The 2x2 tables for inter and intra observer agreement as to presence of tenderness at a site are:-

Inter-observer comparison

		JW_am	
		Non Tender	Tender
TT	non Tender	696	101
	Tender	46	107

KAPPA = 0.50 (considered to be moderate agreement)

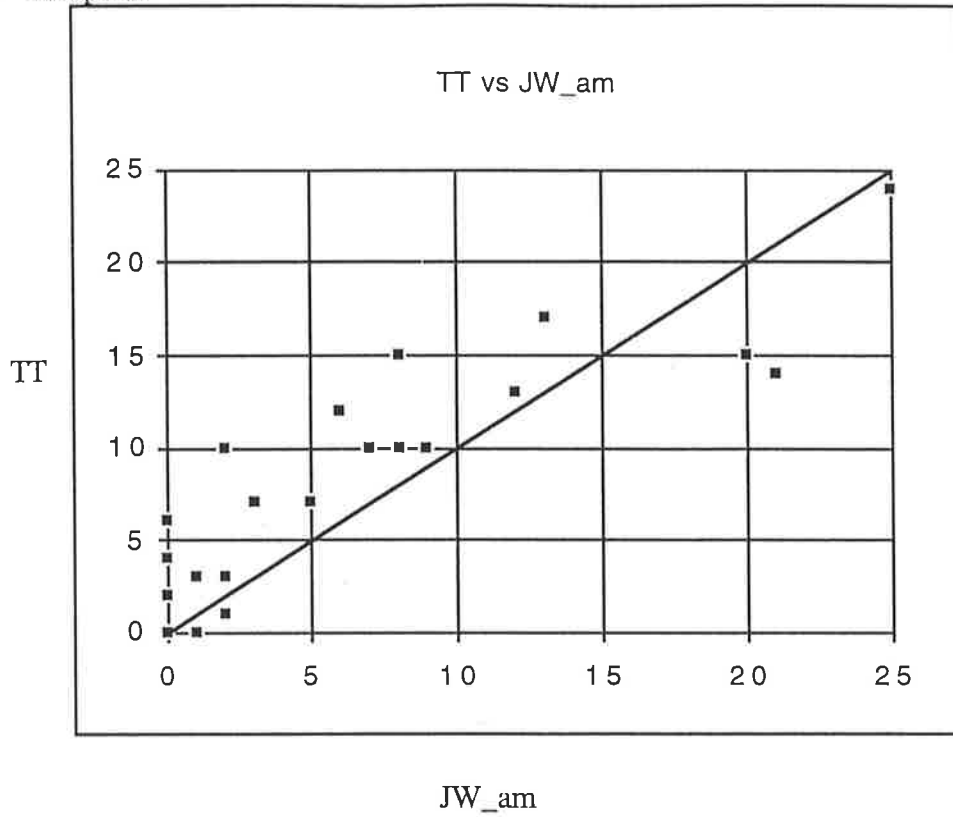
Intra-observer comparison

		JW_am	
		Non Tender	Tender
JW_pm	non Tender	674	26
	Tender	68	182

KAPPA = 0.73 (considered to be good agreement)

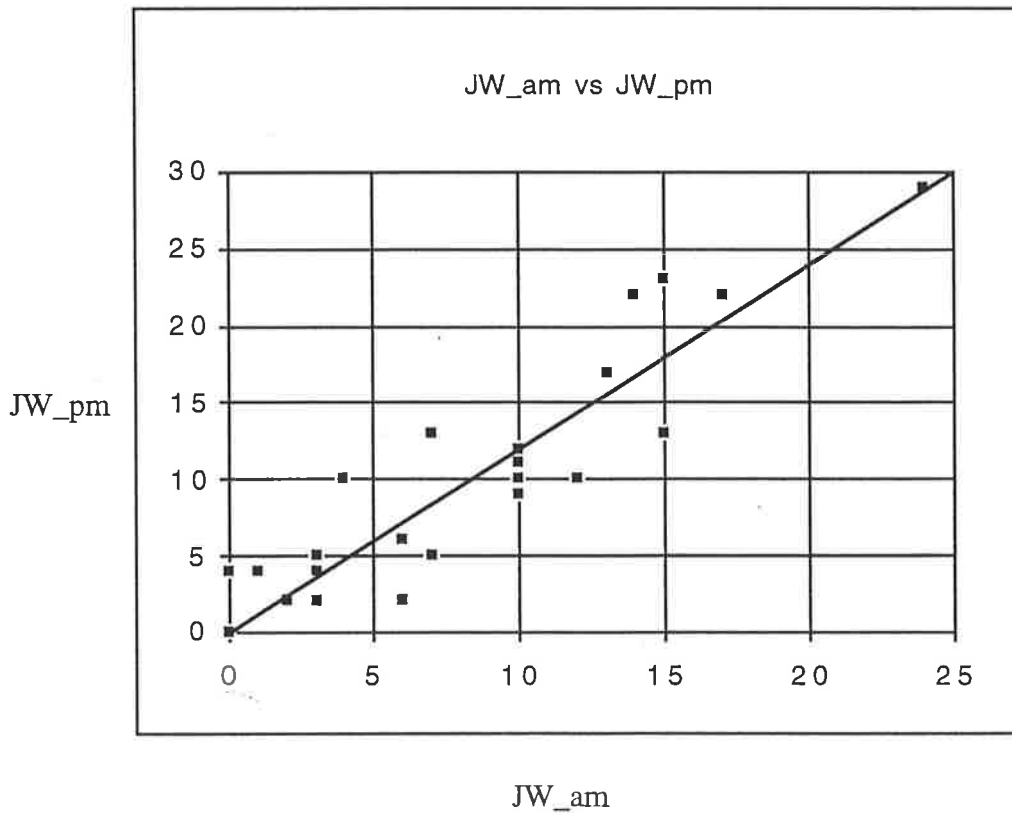
Agreement as to the number of sites of tenderness in each subject was tested using the Intra-class Correlation Coefficient (ICC)[Wright & Feinstein, 1992]. The scatter-plot charts and the ICC for inter and intra observer agreement as to number of tender sites in an individual are:-

Inter-observer comparison



Intra-class Correlation Coefficient =0.814

Intra-observer comparison



Intra-class Correlation Coefficient =0.860

This demonstrates a high degree of agreement between the number of sites of tenderness found by the same assessor on 2 occasions.

Summary.

The inter-observer reliability results indicated that similar findings could be expected when different assessors look for sites of upper-limb tenderness, given appropriate training and experience. The intra-observer reliability results indicated an adequate level of reproducibility to permit statistical analysis of this variable collected from different subjects by one assessor.

3.12.2. Study variables from the clinical history.

The methods of measurement of the study variables are detailed within the frame work of the assessment process during which they were collected.

A series of specific questions provided the following variables

- Regular aerobic physical activity (hours/week). Questions about sport, aerobics etc permitted calculation of this variable.
- Hours per week spent at potential problem-causing activity. (ie music practice). Questions about music practice, writing, typing etc permitted calculation of this variable.
- Upper-limb symptoms associated with activity. (The Outcome Variable). After physical examination, a series of questions were asked to permit coding according the system of classification of the Occupational Overuse Syndrome as developed above (Section 3.10). Consideration of possible specific diagnoses was made at all phases of the assessment.
- History of change in activity at time of symptom onset. If any symptoms were described, questions concerning the history of onset permitted a decision to be made whether or not there were any preceding change in the level or type of activity which was associated with those symptoms. This provided a dichotomous (yes/no) variable.
- Details of which activities were associated with symptoms. Specific questioning regarding the relationship of activities to symptoms was made, to establish whether one or more activities were involved.

3.12.3. Study variables from clinical examination.

- Posture . During the clinical examination music students were asked to stand upright with their normal relaxed posture. Posture was assessed by observation of the position of the neck and shoulders into satisfactory and sub optimal. This provided a dichotomous variable.

- Body Mass Index: Calculated as the Weight (in Kilograms) divided by the Height (in meters) squared
- Upper-limb tenderness . The presence of sites of tenderness was defined using a set examination protocol which used a specified list of sites irrespective of reported symptoms as described above.

3.12.4. Study variables from special tests.

- Aerobic Fitness. The standardised McArdle Step test was used to estimate V O₂ max. [McArdle, 1973] This uses the average heart rate 15 seconds after a three minute stepping exercise to estimate this variable. During the exercise subjects step up and down from a standard height step at a fixed rate. In terms of accuracy of prediction, one could be 95% confident that the predicted V O₂ max. would be within +/- 16% of the true V O₂ max.
- Hand dexterity. Maximal tap rate of the right and left index finger, ring finger and wrist. Each maximal tap rate was measured by asking the subject to keep time with a quartz controlled metronome and then gradually increasing the metronome rate until the subject was not able to keep in time. The highest number of beats per minute, where the subject was able to keep time, was recorded as the maximal tap rate for that appendage.
- Hand Span. The subjects were asked to stretch each hand along a line on a sheet of paper. The position of the tip of the thumb and the tip of the little finger was marked and the distance between measured in millimetres.
- Grip Strength. The "Jamar" Grip Strength Meter was used to measure grip strength. The maximum force applied at each of the five settings of the Jamar Grip Strength Meter was recorded for both the right and left hand.
- Pinch Strength. The "Martin Vigorimeter" was used to measure pinch strength. The maximum thumb to index finger pinch force was recorded for the right and left hand.
- Peak Expiratory Flow Rate. (Wright PEFR meter) .The "Wright" Peak Expiratory Flow Rate meter was used to measure this simple parameter of respiratory function. The best of three attempts was recorded as this variable.

3.12.5. Study variables from self assessment questionnaires.

Subjects were asked to complete three standardised psychological self assessment questionnaires while waiting for the medical assessment.

- Illness Behaviour Questionnaire. “The Illness Behaviour Questionnaire (IBQ) was developed as a self-report instrument to record aspects of illness behaviour, particularly those attitudes which suggest inappropriate or maladaptive modes of responding to one’s state of health.” [Pilowsky and Spence, 1983] The concept of Abnormal Illness Behaviour encompasses a number of syndromes where there is a fundamental discrepancy between objective pathology and the patient’s response to it, ie hypochondriasis, conversion reaction, neurasthenia. The modified form of this questionnaire adapted for use with all individuals irrespective of the presence or absence of symptoms was utilised.

This questionnaire provided a measure of seven aspects of illness behaviour. These 7 scales were:

- A. General hypochondriasis (GH)
- B. Disease conviction (DC)
- C. Psychological vs somatic concern (PvSC)
- D. Affective inhibition (AI)
- E. Affective disturbance or dysphoria (AD)
- F. Denial (D)
- G. Irritability (I)

Extensive testing of reliability and validity has been done by Pilowsky and Spence.

The three scales which were used to characterise conversion reaction were Disease Conviction (DC), Denial (D) and Psychological vs Somatic Concern (PvSC) [Spence et al, 1985]. The theory that Non-specific Occupational Overuse Syndrome is a conversion reaction would be supported if those who developed Non-specific Occupational Overuse Syndrome had scores on these three scales significantly different from those who did not develop the problem. Appendix D

- Trait Anxiety. The State-Trait Anxiety Inventory, as developed by Spielberger, Gorsuch and Lushene, consists of two separate 20 item self-report questionnaires designed to measure two distinct anxiety concepts. “State-Anxiety” is a measure of the level of anxiety at a particular moment in time and “Trait-Anxiety” is a measure of an individual’s tendency to respond to psychological stress with different levels of State-Anxiety. Trait-Anxiety refers to relatively stable individual differences in anxiety proneness. The Trait Anxiety Inventory was chosen to test the hypothesis that an increased tendency to anxiety is associated with a higher incidence of Non-specific Occupational Overuse Syndrome. Appendix E
- General Health Questionnaire. The General Health Questionnaire is a self-report questionnaire which measures a tendency towards neurosis as well as the current level of psychological stress. This questionnaire was chosen to test the hypothesis that an increased neurotic tendency is associated with a higher incidence of Non-specific Occupational Overuse Syndrome. Appendix F

3.12.6. The study variables VS hypotheses tested.

The individual study variables are listed against the hypotheses to be tested.

Research Design used	Hypothesis to be tested	Study Variable
<u>Cross-sectional Analysis</u> (Sample from "normal" population)	1. That the musculo-skeletal structures of normal individuals are non-tender.	• Presence and number of sites of tenderness
	2. That the musculo-skeletal structures of normal individuals are not painful.	• Presence and severity of symptoms
	3. That activities associated with higher levels of upper-limb activity are associated with a higher prevalence of symptoms consistent with a diagnosis of Non-specific Occupational Overuse Syndrome .	• Details of which activities were associated with symptoms and who undertook those activities
<u>Case-control Analysis</u> (Differences between asymptomatic and symptomatic individuals)	4. Change in activity levels increases risk of Non-specific Occupational Overuse Syndrome	• Presence of change in activity levels preceding onset of symptoms
	5. That routine clinical examination is able to discriminate on the basis of the presence of tender sites between those with Non-specific Occupational Overuse Syndrome and those not affected.	• Number of sites of tenderness
	6. That the majority of cases of Non-specific Occupational Overuse Syndrome are caused by a single process.	• Diagnoses apparent in those with symptoms
<u>Cohort Prospective Analysis</u> (Three year followup of initially unaffected music students, looking at the differences in pre-morbid study variables, comparing subsequently affected individuals with those not affected)	7. Presence of tender sites increases the risk of later development of Non-specific Occupational Overuse Syndrome .	• Presence and number of sites of tenderness
	8. The presence of "Inconsequential Symptoms" increases the risk of developing "Consequential Symptoms" of Non-specific Occupational Overuse Syndrome.	• Presence and severity of symptoms at initial assessment of followup assessment
	9. Increased physical fitness decreases risk of Non-specific Occupational Overuse Syndrome :	• Aerobic fitness assessed by estimation of V O ₂ max • Involvement in physical activity (Hours / week) • Dexterity assessed by maximal coordinated digital tap rate. • Grip strength assessed with Jamar dynamometer • Pinch strength assessed with Martin Vigorimeter • Peak Expiratory Flow Rate • Posture
	10. Certain physical attributes increase risk of Non-specific Occupational Overuse Syndrome	• Hand span • Body Mass Index Wt (kg)/Ht (m) Squared
	11. High Activity levels increase risk of Non-specific Occupational Overuse Syndrome	• Number of hours music practice per week
	12. Psychological factors are associated with increased risk of Non-specific Occupational Overuse Syndrome	• Tendency to neurosis assessed by GHQ score • Tendency to be anxious assessed by Trait component of STAI • Factors of Illness Behaviour assessed by the IBQ

3.13. Sample size.

Population 1 was expected to be approximately 100 subjects as the yearly intake of music students was 50. Previously reported incidence of Non-specific Occupational Overuse Syndrome among music students was between 10 and 30% (Fry, personal communications 1985) providing an expected group of 10 to 30 subjects affected by the Non-specific Occupational Overuse Syndrome in the longitudinal arm of the study.

Population 2 contained all the students from faculties other than music who were appropriately assessed. The number of 53 students provided a satisfactory sample size for comparison.

Population 3 contained all the patients presenting with activity related symptoms. The number of 56 patients, 29 with no specific diagnosis, provided a satisfactory sample size for comparison.

3.14. Analysis.

For the purposes of statistical testing a p value of 0.05 was taken as significant. The computer programme "StatView 4" was used to perform all statistical analyses except for the test of normal distribution used for the Psychological Questionnaire Scores which was done with MiniTab.

Hypotheses 1 and 2 were tested by looking for sites of tenderness and presence of symptoms amongst members of a normal population. If sites of tenderness or activity related symptoms were found to occur in members of such a population then either these hypotheses should be rejected or the nature of normality would have to be reconsidered.

Hypothesis 3 was tested by comparing the prevalence of symptoms between instrumental musicians and non-musicians with the contingency table Chi Squared statistic.

Hypotheses 4 and 6 were not amenable to formulation of a null hypothesis allowing for statistical testing. Hypothesis 4 was investigated by calculating the proportion of cases of Non-specific Occupational Overuse Syndrome in which changes in the intensity of the nature of the activity immediately preceded the onset of symptoms. Hypothesis 6 required a general overview of the data to consider whether or not there were similarities between the majority of the cases of Non-specific Occupational Overuse Syndrome which would be consistent with a single process.

Hypothesis 5 was tested:

- by determining whether the presence of tenderness was present in the "unaffected" individuals.
- by testing the null hypothesis that there would be no difference in the number of tender sites between the different diagnostic and occupational categories with 2 way Analysis of Variance.

Hypotheses 7 to 12 were investigated by testing the null hypothesis of no association between the variable in question and the eventual development of Non-specific Occupational Overuse Syndrome. The Chi Square/Fisher's Exact Test was used for categorical variables and the appropriate parametric/non-parametric Analysis of Variance was used for continuous variables.

Chapter 4. Results

4.1. Introduction

The fact that this study was designed to explore a wide range of factors of potential relevance to Non-specific Occupational Overuse Syndrome meant that a large amount of data was collected. Consideration of the preceding medical literature allowed the definition of a series of specific hypotheses whose testing might allow the resolution of some of the questions regarding its pathophysiology. Study variables were selected from the overall data-set on the basis of their ability to provide a test for one of the hypotheses selected for investigation. (see section 3.12.6).

Data was collected from three different populations, using different sampling techniques and for different reasons. Sample 1, "Instrumental Music Students", was chosen as previous studies have found a high incidence of Non-specific Occupational Overuse Syndrome in instrumental musicians. With this high incidence, it was likely that a prospective study on members of this group would include a significant number of individuals who developed the Non-specific Occupational Overuse Syndrome and so permit the search for pre-morbid risk factors. Sample 2, "Other Students" was included in order to be able to compare the base line findings of musicians with a more normal group of individuals. These two groups included individuals from all categories within the diagnostic classification of Occupational Overuse Syndrome (as defined in section 3.10), however, the prevalence of "Consequential Symptoms" was small (8 cases). Sample 3, "Patients presenting for treatment", was included in order to increase the number of individuals with "Consequential Symptoms", to allow statistical comparisons with the use of a case-control methodology. Individuals from Sample 3 were, by definition, classified as having "Consequential Symptoms". This sample contained both instrumental musicians and non-musicians. In this way, it was possible to collect sufficient numbers to be able to analyse the study variables by comparing their values in the occupational categories "Instrumental musician" of "Non-musician" and the diagnostic categories defined by the primary study outcome variable given by the diagnostic classification of Occupational Overuse Syndrome .

Pertinent aspects of the results and statistical analyses are included in this chapter. Appendix G contains supplementary statistical tables and is referenced at the appropriate point in this chapter.

4.2. Demographic characteristics and differences between Study Samples.

4.2.1. Sample 1, "Instrumental Music Students",

All instrumental music students enrolled over a two year period at the University of Adelaide were eligible for inclusion in this Sample. Those who presented for the "Health Check" provided by the University Health Service were included. The sampling method for this Sample could be described as a cohort sample. These students were all included in the occupational category "Instrumental Musician".

<u>Instrumental Music Students assessed at the beginning of course</u>	<u>95</u>
<u>Instrumental Music Students enrolled during the 2 year period of data collection who choose not to present for the "Health Check"</u>	<u>7</u>

4.2.2. Sample 2, "Other Students".

Students, other than instrumental music students, were randomly allocated for their "Health Check" to the different medical officers at the University Health Service. Students who were assessed by Dr. White in this way were included in this group. The sampling method for this Sample could be described as a random sample. It might be argued that this sample was not random in the truly statistical sense, however, it did form an unbiased sample of non-music students. These were all included in the occupational category "Non Musician".

<u>Other Students</u>	<u>53</u>
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4.2.3. Sample 3, "Patients"

Details were collected from individuals who sought treatment and in whom initial symptoms were consistent with the diagnosis of Occupational Overuse Syndrome during the course of the study. The primary goal in assessment of these patients was the search for a specific diagnosis to explain their symptoms. The sampling method for this Sample could be described as a "patient sample".

Instrumental Musicians	47
<u>Non-Musicians</u>	<u>9</u>
<u>Total</u>	<u>56</u>

All subjects in these Samples were allocated to one of the categories within the diagnostic classification defined for the Occupational Overuse Syndrome (section 3.10). By definition all individuals in Sample 3 (patients) were classified as either category 2 (Non-specific

Occupational Overuse Syndrome with “Consequential Symptoms”) or category 3 (Specific Occupational Overuse Syndrome)

Samples 1 and 2 were chosen so that data on a control group of individuals who did not suffer from any symptoms associated with activity could be collected. In addition data from these Samples could also be used to give an indication as to the prevalence of symptoms consistent with Occupational Overuse Syndrome in those populations.

The contribution from the three Samples to the categories defined by the diagnostic classification of Occupational Overuse Syndrome are presented. Percentages are included for Samples 1 and 2. to indicate the associated prevalence estimate.

	Sample 1. Instrumental Music Students	Sample 2 Other Students	Sample 3 Patients	Total
No Symptoms (Unaffected)	52 (55%)	43 (81%)	0	95
Non-specific Occupational Overuse Syndrome with “Inconsequential Symptoms”	35 (37%)	8 (15%)	0	43
Non-specific Occupational Overuse Syndrome with “Consequential Symptoms”	6 (6%)	2 (4%)	29	37
Specific Occupational Overuse Syndrome	2 (2%)	0 (0%)	27	29
Total	95 (100%)	53 (100%)	56	204

Sample 3 can be divided into Instrumental Musicians and Non-musicians.

	Patients Instrumental Musicians	Patients Non-Musicians
Non-specific Occupational Overuse Syndrome with “Consequential Symptoms”	25	4
Specific Occupational Overuse Syndrome	22	5

This provided a collection of individuals from the populations of both instrumental musicians and non-musicians each containing subjects from each of the diagnostic categories.

The students classified as either “No Symptoms” or “Inconsequential Symptoms” were used subsequently as “control” groups. Details of the assessment of these groups were compared with the group in which the diagnosis of Non-specific Occupational Overuse Syndrome with “Consequential Symptoms” (Occupational Overuse Syndrome category 2) had been made.

There were 29 individuals for whom a specific diagnosis was made, 2 from the music student sample and 27 from the patient sample. Details of the specific diagnoses were as follows:

Site of pathology	Diagnosis	No. of cases
Neck and Shoulder	Chronic cervical soft tissue injury secondary to Motor Vehicle Accident	3
	Calcific supraspinatus tendonitis	1
	Rotator cuff capsulitis	1
Elbow	Medial epicondylitis	1
Wrist	Dorsal wrist ganglion	3
	Scapho-lunate instability	2
	Triangular fibro-cartilage complex tear	2
	deQuervains tenosynovitis	1
	Stress fracture of pisiform bone	1
	Traumatic wrist sprain	1
Hand	OA 1st CMCJ thumb	2
	Chronic sprain ulnar collateral ligament MCPJ thumb	1
	Trigger finger	1
Nerve Compression Syndromes	Ulna neuritis	3
	Anterior interosseous nerve syndrome	1
	Digital nerve compression index finger	1
	Radial tunnel syndrome	1
Neurological Conditions	Focal dystonia	2
	Brachial neuritis	1

These 29 individuals in whom a specific diagnosis had been made were not included in the analysis of the features of Non-specific Occupational Overuse Syndrome.

Before any analysis of the differences between the subgroups of Non-specific Occupational Overuse Syndrome could be made, any effects caused by the differences in content and sampling for the original three different Samples had to be considered.

4.2.4. Comparison of the demographic details .

4.2.4.1. Occupation.

The occupations of the individuals in the three groups are listed in Appendix G, Table 1.

Features of importance are:

1. Sample 2 contains no-Instrumental Musicians.
2. Sample 3 contains 19 Instrumental Music Students, 10 other instrumental musicians and only 4 non musicians. Both the high school student and the university lecturer (Table 1) also had symptoms directly associated with the performance of musical instruments.

Within the Instrumental Music Students' group (Sample 1) Appendix G Table 2. lists the principal instrument played.

Of note, out of the 93 Instrumental Music Students, 45 played a second instrument. Details of the second instrument are listed in Appendix G Table 3.

Within the Patient group, Sample 3, 25 played musical instruments and 4 did not. Appendix G, Table 4. lists the principal instrument played.

Of these, 10 played a second instrument. (Details Appendix G, Table 5)

Clearly the activities undertaken by the musicians and the non-musicians differed. Sample 1 and Sample 3 were very similar because of the number of Instrumental Musicians included in Sample 3. The reason for this is likely to be that musicians at large and the Instrumental Music Students in particular were made aware of Dr. White's interest in the potential problems of musicians through the special University Health Service health check designed specifically for instrumental musicians. When they developed problems associated with music performance they therefore sought his opinion.

4.2.4.2. Age

Age at time of Assessment. (statistics in detail. Appendix G, Table 6)

	Sample 1	Sample 2	Sample 3
mean	19.4	20.6	24.6
Std.Dev.	3.2	5.4	7.6

The difference between the means was compared with Analysis of Variance (Details Appendix G, Table 7) which showed:-

1. that there was no statistically significant difference between the mean age of Sample 1 and Sample 2
2. that the difference between the mean age of Sample 3 and Sample 1 and 2 was statistically significant.

This was to be expected as Sample 3 was taken from the community at large whereas Sample 1 and 2 was taken from 1st year University students. Although the difference was statistically significant the magnitude of the difference in both the absolute value of the means and magnitude of the range, was not large and was unlikely to affect other comparisons.

4.2.4.3. Sex.

Sex ratio within the different study samples.

Observed Frequencies for Sex, Study Sample

	Sample1	Sample2	Sample3	Totals
F	63	25	20	108
M	30	28	9	67
Totals	93	53	29	175

Percents of Column Totals for Sex, Study Sample

	Sample1	Sample2	Sample3	Totals
F	67.742	47.170	68.966	61.714
M	32.258	52.830	31.034	38.286
Totals	100.000	100.000	100.000	100.000

The sex ratio of Sample 1 (Instrumental Music Students) was 63:30 Female to Male. This was consistent with the preponderance of females enrolled in the music faculty.

The sex ratio of Sample 2 was 25:28. This sex ratio was consistent with that which could be expected from enrolment figures in the university at large. This was statistically different from Sample 1. (Chi Sq=5.14: p=0.02, details Table 8)

The sex ratio of Sample 3 was 20:9 F:M. It was not significantly different statistically (at the 95% level) from Sample 1 or Sample 2. (details Appendix G, Table 9 and 10)

To understand these sex ratio differences Sample 3 was divided into 3 groups, Instrumental Music Students (n=19), other instrumental musicians (n=10) and non musicians (n=4). The sex ratio for each group in this division is shown in the following table:-

	Instrumental Music Students	Other Instrumental Musicians	Non Musicians
Female	13 (68.4%)	7 (70%)	2 (50%)
Male	6 (31.6%)	3 (30%)	2 (50%)

It can be seen that the sex ratio for the instrumental musicians (both Instrumental Music Students and Other Instrumental Musicians) in Sample 3 was similar to Sample 1. For the non musicians, although there were small numbers, the sex ratio was similar to Sample 2 which also contained no musicians. Thus, Sample 3 was a collection of both musicians and non musicians, weighted by numbers towards more musicians. The sex ratio for this group lay between that for Sample 1 and Sample 2.

Thus the differences in sex ratio across the three Samples would seem to be due to the difference in sex ratio between musicians and non-musicians in the population at large. Where appropriate, sex was controlled for in the analysis of study variables.

4.2.5. Comparison of demographic details between the analysis subgroups

The three sampling methods were used to collect adequate numbers in each of the subgroups created by both of the classifications which were to be used for comparisons of the study variables. These were the Occupational and Diagnostic classifications. Variation in the demographic details were assessed between the subgroups produced by the categories from these classifications.

Age:

Occupational Classification

	Instrumental musicians	Non-musicians
mean	20.4	21.2
Std.Dev.	4.5	6.4

There was no significant difference in the age of musicians of non-musicians. (Details Appendix G, Table 11)

Diagnostic classification

	No Symptoms	Inconsequential Symptoms	Consequential Symptoms
mean	19.90	19.98	23.38
Std.Dev.	4.04	4.73	7.14

There was a significant difference in the age between diagnostic categories. (Appendix G, Table 12) (ANOVA $p=0.0013$). Fisher's post hoc test showed this to be due to the higher mean age of the "Consequential Symptoms" category. This was to be expected as the majority of subjects for this category were derived from Sample 3 which showed the same slightly higher mean age.

Sex:

Occupational Classification

As expected from the analysis of the sex ratios in the original samples there was a significant difference in the sex ratio (M:F) of musicians (37:81) of non-musicians (30:27) (Chi Sq. 6.49 $p=0.01$; Details Appendix G, Table 13)

Diagnostic classification

There was no significant difference in the sex ratio between diagnostic categories. (Details Appendix G, Table 14)

4.3. Cross-sectional Analysis

The first set of analyses related to the data collected at the initial assessment of individuals from Sample 1 and 2. The sampling method was that of unbiased selection from a homogeneous population, that is, university students, allowing a cross-sectional analysis to be undertaken

Sample 1 was drawn from the population of first year university students involved in performance of instrumental music. Sample 2 was drawn from the population of first year university students who did not play musical instruments. Sampling was independent of the presence of musculo-skeletal symptoms associated with upper-limb activity. The prevalence of such a problem in these two samples was, thus, likely to be representative of the populations from which they were drawn. Sample 3 (patients) was drawn on the basis of the presence of "Consequential Symptoms" and so was not useful in determining the prevalence of such problems.

4.3.1. The prevalence of individuals reporting symptoms consistent with Occupational Overuse Syndrome.

The prevalence of musculo-skeletal problems within Sample 1 and Sample 2 gave an indication of the prevalence in the wider population. The following table lists the number in each subgroup. This value is expressed as the percentage of the total number included in that Sample. For the symptomatic subgroups this is an estimate of the prevalence of that diagnostic category in that occupational category.

	Sample 1. Instrumental Music Students	Sample 2 Other Students
No Symptoms (Unaffected)	52 (55%)	43 (81%)
Non-specific Occupational Overuse Syndrome with "Inconsequential Symptoms"	35 (37%)	8 (15%)
Non-specific Occupational Overuse Syndrome with "Consequential Symptoms"	6 (6%)	2 (4%)
Specific Occupational Overuse Syndrome	2 (2%)	0 (0%)
Total	95 (100%)	53 (100%)

Of particular note was the prevalence of non-specific musculo-skeletal symptoms among individuals whose activities were not affected by those symptoms and who did not consider themselves to have an illness. The prevalence of such "Inconsequential Symptoms" among Instrumental Music Students was 37% and among Other Students was 15%.

Instrumental Music Students (Sample 1) had a higher prevalence of symptoms consistent with the Non-specific Occupational Overuse Syndrome than Other Students (Sample 2).

As noted above there was a sex ratio difference, but no age difference, between Sample 1 and Sample 2.

Analysis for an effect of sex on the prevalence of upper-limb activity symptoms showed no significant effect.

(Chi Sq=3.70; $p=0.16$; Details Appendix G, Table 15).

Analysis of the prevalence of symptoms comparing Instrumental Music Students with Other Students showed that the Instrumental Music Students did have a higher prevalence of "Inconsequential Symptoms" which was statistically significant (37.6% cf 15.1%, Chi Sq.= 7.9, $p=0.005$). Because of small numbers, analysis including individuals with "Consequential Symptoms" was not valid. There did, however, seem to be a trend for higher prevalence of "Consequential Symptoms". (6.5% cf 3.8%) (Details Appendix G, Table 16)

The difference in prevalence of symptoms between Sample 1 and Sample 2 could be explained by the differences in the activities involved.

The activities associated with symptoms in Sample 1 included writing and performance on various musical instruments. The activities associated with symptoms in Sample 2 included writing and racket sports. All individuals in Sample 1 and Sample 2 were involved with writing as part of their studies. The total numbers involved in racket sports was unknown. Of the 93 Instrumental Music Students 45 played two instruments. The total number of individuals involved with each instrument was therefore known. Of the 93 Instrumental Music Students 41 reported symptoms, of these 10 associated the symptoms with more than one activity.

The number of individuals involved in each specified activity and the number of these who reported symptoms associated with that activity is tabulated in the following table. Because of the small number involved in the more unusual instruments, prevalence figures were not calculated in relation to them.

Sample 1 (Musicians) Activity	Total number involved in activity	Number reporting “ Inconsequential ” symptoms associated with that Activity (Prevalence)	Number reporting “ Consequential ” symptoms associated with that Activity (Prevalence)
Writing	93	12 (13%)	1 (1%)
Piano	53	16 (30%)	4 (8%)
Violin	17	4 (24%)	2 (12%)
Flute	12	3 (25%)	0 (0%)
Clarinet	8	2 (25%)	0 (0%)
Guitar	8	3 (38%)	0 (0%)
Cello	4	0	0
Percussion	4	1	0
Trumpet	4	1	0
Oboe	3	1	0
Trombone	3	0	0
Double Bass	2	1	0
Organ	2	0	0
Piccolo	2	0	0
Recorder	2	0	0
Viola	2	0	0
Bassoon	1	0	0
Lute	1	0	0
Saxophone	1	0	0

In Sample 2 the symptoms which occurred were associated with writing in 8 out of a total of 10 occasions.

Sample 2 (Non- musicians) Activity	Total number involved in activity	Number reporting “ Inconsequential ” Symptoms associated with that Activity (Prevalence)	Number reporting “ Consequential ” Symptoms associated with that Activity (Prevalence)
Writing	53	6 (11%)	2 (4%)
Racket Sports	Unknown	2 (N/A)	0 (N/A)

The prevalence of “Inconsequential Symptoms” associated with writing was the same in both Sample 1 and Sample 2 (Chi Sq.=0.000, $p>0.9999$). (this analysis excludes the group with “Consequential Symptoms” because of small numbers) (Details see Appendix G, Table 17)

The upper-limb intensive activity common to both Sample 1 and Sample 2 (writing) was associated with the same prevalence of symptoms. The activities with which the subjects in Sample 1 were involved and with which those in Sample 2 were not (performance of musical instruments) explained the excess of symptoms noted in Sample 1 as compared with Sample 2.

4.3.2. The prevalence of Tenderness in the different diagnostic categories .

The question as to whether or not tenderness may be elicited during the examination of upper-limb structures of “normal” individuals was investigated by calculating the prevalence of the presence of tenderness in the control groups derived from Sample 1 and 2. The following table lists the ratio of the number of individuals with tenderness to the total number in each subgroup; this ratio is expressed as a percentage (in brackets) giving an estimate of the prevalence of tenderness in that subgroup.

	Sample 1. Instrumental Music Students	Sample 2 Other Students
No Symptoms (Unaffected)	29/52 (56%)	24/43 (56%)
Non-specific Occupational Overuse Syndrome with “Inconsequential Symptoms”	28/35 (80%)	8/8 (100%)
Non-specific Occupational Overuse Syndrome with “Consequential Symptoms”	6/6 (100%)	2/2 (100%)

Of particular note was the prevalence of tenderness in the “No Symptoms” category. This was 56% for both the Instrumental Music Students and the Other Students who had never experienced symptoms associated with activity. Further analysis of the phenomenon of tenderness in these “normal” subgroups is included in the Case-control analysis.

4.4. Case-control Analysis

The addition of sample 3 increased the numbers of subjects in the “Consequential Symptoms” category. This meant that the overall sample was not unbiased. In particular, the additional subjects had been selected on the basis that they were seeking help for the presence of symptoms that were of some consequence to them (they were patients). Consequently, the analysis suitable for this combined sample was that of a case-control analysis. In this study, for reasons discussed above the division between cases and controls remained somewhat blurred because of the category in the classification of Non-specific Occupational Overuse Syndrome labelled “Inconsequential Symptoms”. Nevertheless, the aim of this part of the study was to explore the differences between the three diagnostic categories, “No Symptoms”, “Inconsequential Symptoms” and “Consequential Symptoms”, while simultaneously considering the differences between the two occupational categories “Instrumental Musician” of “Non-Musician”. The issue as to whether the individuals in the Inconsequential Symptoms category should be considered as cases or controls, will be discussed in subsequent chapters.

Thus, the distribution of the numbers of subjects in each of the subgroups which were used for the Case-Control analysis was:

	Instrumental Musicians	Non-musicians	Total
No Symptoms (Unaffected)	52	43	95
Non-specific Occupational Overuse Syndrome with "Inconsequential Symptoms"	35	8	43
Non-specific Occupational Overuse Syndrome with "Consequential Symptoms"	31	6	37
Total	118	57	175

4.4.1. Features of the history

The features of the history which were considered to be of interest were:

1. The relationship between activity and symptoms.
2. The site and extent of symptoms.

4.4.1.1. Relationship between activity and symptoms.

The onset of symptoms was associated with a recent modification to or change in intensity of the associated activity.

The history involved specific questioning concerning the onset of symptoms.

Number (percentage) reporting recent modification to or increase in intensity of the associated activity is given in the following table:-

	Musicians	Non-musicians	Total
Inconsequential Symptoms	14/35 (40%)	1/8 (12.5%)	15/43 (35%)
Consequential Symptoms	18/31 (58%)	2/6 (33%)	20/37 (54%)
Total	32/66 (48%)	3/14 (21%)	35/80 (44%)

There were 43 individuals classified as having "Inconsequential Symptoms", 15 of these recalled a recent modification to, or change in intensity of the associated activity (35%). There were 37 individuals classified as having "Consequential Symptoms", 20 of these recalled a recent modification to, or change in intensity of the associated activity (54%). The difference between diagnostic categories, however, was not statistically significant. (Fisher's Exact p-Value = 0.11) (Detail Appendix G, Table 18)

Analysis of individuals with symptoms comparing musicians with non-musicians indicated that more musicians were aware of a recent change in their activity (48% cf 21%), however, this difference was not statistically significant (Fisher's Exact p-Value = 0.08) (Detail Appendix G, Table 19)

4.4.1.2. The areas in which symptoms occurred.

Symptoms were usually somewhat diffuse and poorly localised. When asked where discomfort was felt, subjects would indicate a region. Consequently, the 5 areas defined in section 3.12.1 were used to identify the site of symptoms. These areas were as follows.

1. Hand/Wrist
2. Forearm/Elbow
3. Arm
4. Shoulder
5. Neck

4.4.1.3. The distribution of symptoms

The distribution of symptoms within the four symptomatic subgroups is tabulated as follows:

	Inconsequential Symptoms (Musicians) (N=35)				Consequential Symptoms (Musicians) (N=31)			
	Right		Left		Right		Left	
	Percent	Number	Percent	Number	Percent	Number	Percent	Number
Hand/Wrist	46%	16	34%	12	65%	20	55%	17
Forearm/Elbow	17%	6	11%	4	35%	11	23%	7
Arm	6%	2	3%	1	10%	3	10%	3
Shoulder	17%	6	11%	4	23%	7	13%	4
Neck	34%	12	29%	10	19%	6	19%	6

	Inconsequential Symptoms (Non-musicians) (N=8)				Consequential Symptoms (Non-musicians) (N=6)			
	Right		Left		Right		Left	
	Percent	Number	Percent	Number	Percent	Number	Percent	Number
Hand/Wrist	38%	3	13%	1	100%	6	17%	1
Forearm/Elbow	38%	3	13%	1	17%	1	0%	0
Arm	13%	1	13%	1	0%	0	0%	0
Shoulder	0%	0	0%	0	17%	1	17%	1
Neck	13%	1	13%	1	0%	0	0%	0

4.4.1.4. The extent of symptoms.

The extent of symptoms of an individual was expressed as the number of areas affected out of the 10 possible areas. The number of areas where symptoms were bilateral as opposed to unilateral is also presented. Only those individuals with symptoms are included in this analysis.

Comparing the extent of symptoms for Occupational Classification (Musicians of Non-musicians with symptoms), the average number of areas with symptoms out of a possible 10 (and the range) was as follows.

	Musicians	Non-musicians
Total	2.38 (1-10)	1.57 (1-4)
Bilateral	1.69 (0-10)	0.71 (0-4)
Unilateral	0.68 (0-3)	0.86 (0-2)

Comparing the mean extent of symptoms for Diagnostic Classification (“Inconsequential Symptoms” of “Consequential Symptoms”) the average number of areas with symptoms out of a possible 10 (and the range) was as follows.

	Inconsequential Symptoms	Consequential Symptoms
Total	1.97 (1-10)	2.54 (1-10)
Bilateral	1.30 (0-10)	1.78 (0-10)
Unilateral	0.67 (0-3)	0.76 (0-2)

There appears to be a difference in the mean extent of symptoms between groups for both the factors “Occupational Classification” and “Diagnostic Classification”. These apparent trends were tested with a 2 way ANOVA which showed that neither were of statistical significance (ANOVA p-Value for Occupational Classification = 0.19, ANOVA p-Value for Diagnostic Classification = 0.52, Details Appendix G, Table 20)

4.4.2. Features of the examination (tenderness)

The primary feature to arise from examination of the subjects was the presence of tenderness. Tenderness was assessed with use of a standard protocol as described in chapter 3, section 3.12.1. The phenomenon of tenderness was present in all subgroups. The following analyses investigated this.

4.4.2.1. Percentage of subgroup with at least 1 tender site

The following table gives the percentage of each subgroup with at least 1 site of tenderness. The actual number with tenderness and the total number in each subgroup is given in brackets.

	No Symptoms	Inconsequential Symptoms	Consequential Symptoms
Musicians	56% (29/52)	80% (28/35)	100% (31/31)
Non-musicians	56% (24/43)	100% (8/8)	100% (6/6)

4.4.2.2. The Number of Tender Sites

The following table gives the mean number of tender sites (out of 38 possible sites) subdivided into Occupational and Diagnostic subgroups. Division into number of bilateral and number of unilateral tender sites is also presented. Range of number of tender sites for each cell is given in brackets.

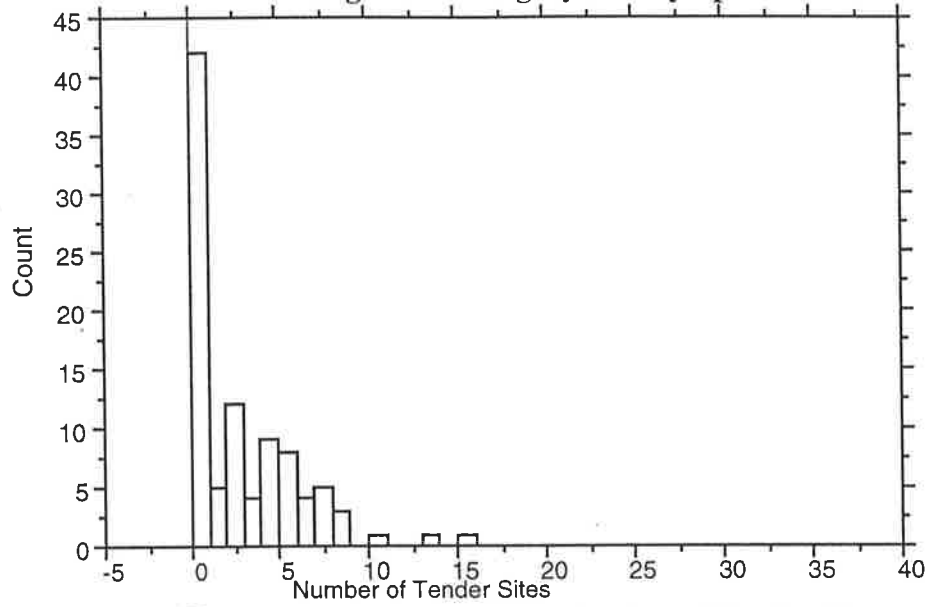
	No Symptoms	Inconsequential Symptoms	Consequential Symptoms	Total
Musicians	2.9 (0 to 15)	7.7 (0 to 17)	16.1 (5 to 31)	7.8 (0 to 31)
Bilateral	2.3 (0-14)	6.9 (0-16)	14.6 (2-30)	6.9 (0 to 30)
Unilateral	0.65 (0-3)	0.83 (0-4)	1.5 (0-4)	0.92 (0 to 4)
Non-musicians	2.0 (0 to 8)	7.8 (4 to 12)	16.7 (9 to 24)	4.4 (0 to 24)
Bilateral	1.6 (0-8)	6.8 (0-12)	15.7 (8-22)	3.8 (0 to 22)
Unilateral	0.37 (0-3)	1.0 (0-4)	1.0 (0-4)	0.53 (0 to 4)
Total	2.5 (0 to 15)	7.7 (0 to 17)	16.2 (5 to 31)	6.7 (0 to 31)
Bilateral	2.0 (0-14)	6.8 (0-16)	14.8 (2-30)	5.9 (0 to 30)
Unilateral	0.53 (0-3)	0.86 (0-4)	1.4 (0-4)	0.79 (0 to 4)

Simultaneous comparisons of the number of sites of tenderness found on examination were made between Occupational Categories (musicians cf non-musicians) and also between the different Diagnostic Categories, with a two way analysis of variance. There was not a significant difference in the number of tender sites between musicians and non-musicians (AVOVA p-Value for Occupational Classification = 0.91). There was a significant difference in the number of tender sites between the three Diagnostic Categories of Non-specific Occupational Overuse Syndrome (AVOVA p-Value for Diagnostic Classification < 0.0001). Fisher's PLSD post hoc test for between group differences showed that the mean number of sites of tenderness was significantly greater in the "Inconsequential Symptoms" category (7.8) than the "No Symptoms" category (2.5) ($p < 0.0001$) and there were significantly more tender sites in the "Consequential Symptoms" category (16.2) than the "Inconsequential Symptoms" category (7.8) ($p < 0.0001$). (Details Appendix G, Table 21)

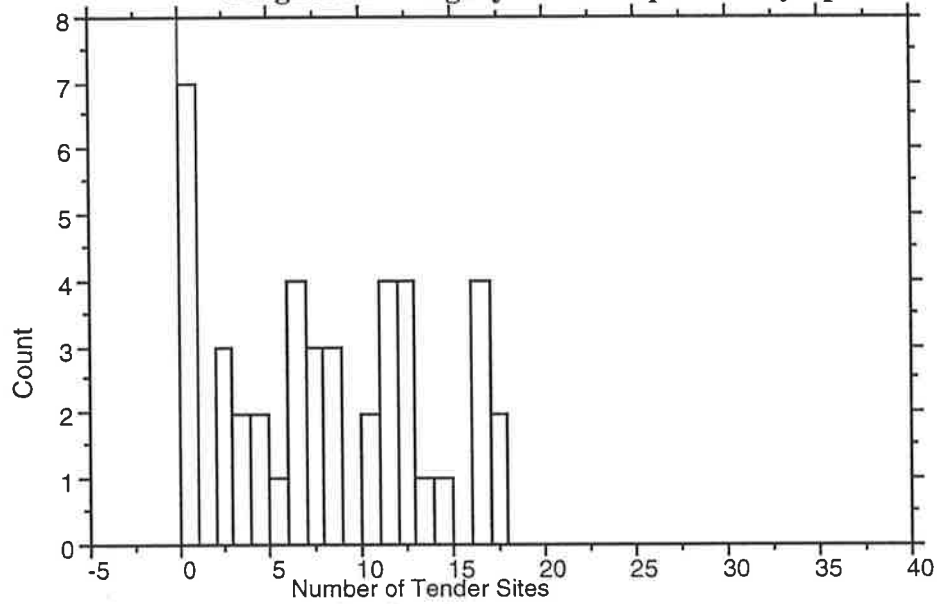
Thus it was shown that the average number of sites of tenderness was independent of occupational classification, but increased progressively across the range of symptoms. The average number of sites of tenderness in the category "No symptoms" was lower than that in the category "Inconsequential Symptoms" which in turn was lower than in the category "Consequential Symptoms" and the difference between categories was highly statistically significant.

The following frequency distribution for number of sites of tenderness graphically displays this difference.

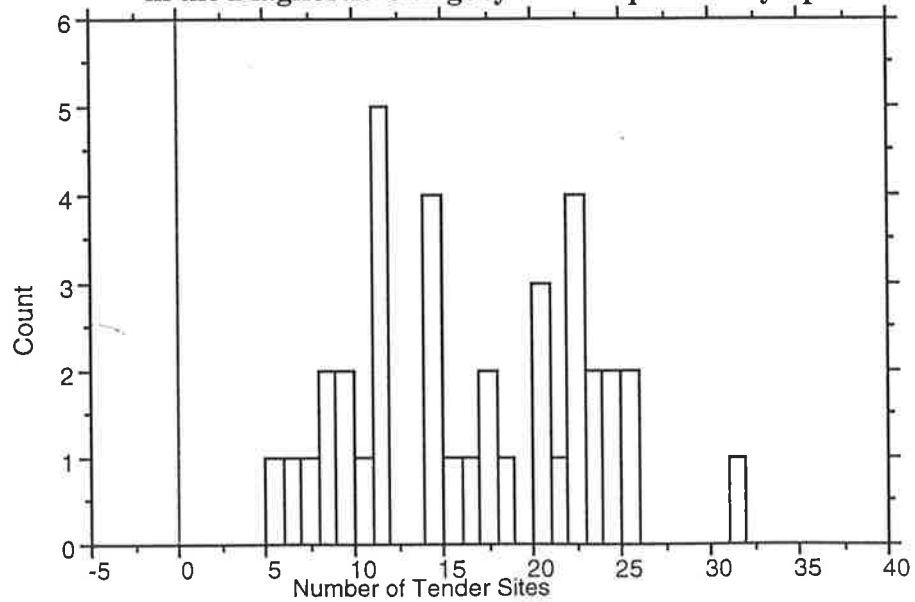
Frequency distribution for Number of Tender Sites in the Diagnostic Category "No Symptoms"



Frequency distribution for Number of Tender Sites in the Diagnostic Category "Inconsequential Symptoms"



Frequency distribution for Number of Tender Sites in the Diagnostic Category "Consequential Symptoms"



4.4.2.3. Anatomical Distribution of Tender Sites

The prevalence of tenderness at each anatomical site for each subgroup also demonstrates certain characteristics of this difference. In addition, this table give a guide as to what to expect when making a systematic examination for sites of tenderness in the upper-limbs.

	Musicians No Symptoms		Musicians Inconsequential Symptoms		Musicians Consequential Symptoms	
	right	left	right	left	right	left
	4th Interdigital Cleft	0.0%	0.0%	5.7%	8.6%	19.4%
3rd Interdigital Cleft	0.0%	1.9%	2.9%	5.7%	19.4%	22.6%
2nd Interdigital Cleft	1.9%	3.8%	8.6%	11.4%	32.3%	35.5%
1st Interdigital Cleft	21.2%	32.7%	42.9%	54.3%	80.6%	80.6%
Thenar Eminence	0.0%	0.0%	0.0%	0.0%	9.7%	9.7%
Hypo-thenar Eminence	0.0%	0.0%	0.0%	2.9%	6.5%	9.7%
CMCJ of Thumb	0.0%	3.8%	11.4%	11.4%	32.3%	35.5%
Radial Wrist Ligament	3.8%	7.7%	22.9%	25.7%	67.7%	54.8%
Ulna Wrist Ligament	0.0%	0.0%	0.0%	0.0%	3.2%	3.2%
Dorsal Wrist Ligament	0.0%	0.0%	0.0%	2.9%	32.3%	25.8%
Plantar Wrist Ligament	0.0%	0.0%	0.0%	2.9%	22.6%	19.4%
Extensor Muscle Belly	9.6%	21.2%	48.6%	45.7%	87.1%	90.3%
Flexor Muscle Belly	1.9%	0.0%	5.7%	2.9%	29.0%	22.6%
Extensor Muscle Origin	38.5%	42.3%	68.6%	71.4%	100.0%	100.0%
Flexor Muscle Origin	28.8%	30.8%	54.3%	65.7%	83.9%	90.3%
Biceps Muscle Belly	5.8%	5.8%	28.6%	28.6%	48.4%	45.2%
Triceps Insertion	0.0%	0.0%	5.7%	5.7%	35.5%	25.8%
Coracoid Process	3.8%	7.7%	37.1%	40.0%	54.8%	61.3%
Superior Trapezius	7.7%	11.5%	20.0%	20.0%	48.4%	48.4%

	Non-musicians No symptoms		Non-musicians Inconsequential Symptoms		Non-musicians Consequential Symptoms	
	right	left	right	left	right	left
	4th Interdigital Cleft	0.0%	0.0%	0.0%	0.0%	16.7%
3rd Interdigital Cleft	0.0%	0.0%	0.0%	0.0%	16.7%	16.7%
2nd Interdigital Cleft	0.0%	0.0%	12.5%	12.5%	33.3%	33.3%
1st Interdigital Cleft	7.0%	14.0%	25.0%	37.5%	100.0%	100.0%
Thenar Eminence	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Hypo-thenar Eminence	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
CMCJ of Thumb	0.0%	0.0%	0.0%	0.0%	33.3%	16.7%
Radial Wrist Ligament	0.0%	0.0%	37.5%	37.5%	50.0%	33.3%
Ulna Wrist Ligament	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Dorsal Wrist Ligament	0.0%	0.0%	12.5%	0.0%	33.3%	33.3%
Plantar Wrist Ligament	0.0%	0.0%	0.0%	0.0%	16.7%	0.0%
Extensor Muscle Belly	2.3%	4.7%	50.0%	50.0%	100.0%	83.3%
Flexor Muscle Belly	0.0%	2.3%	0.0%	12.5%	66.7%	33.3%
Extensor Muscle Origin	41.9%	53.5%	87.5%	100.0%	100.0%	100.0%
Flexor Muscle Origin	27.9%	30.2%	75.0%	87.5%	100.0%	100.0%
Biceps Muscle Belly	0.0%	0.0%	25.0%	25.0%	66.7%	66.7%
Triceps Insertion	0.0%	0.0%	0.0%	0.0%	33.3%	33.3%
Coracoid Process	4.7%	7.0%	12.5%	25.0%	66.7%	66.7%
Superior Trapezius	2.3%	2.3%	25.0%	25.0%	50.0%	50.0%

4.4.3. Relationship of Examination to History (tenderness to symptoms)

4.4.3.1. The prevalence of tenderness in symptomatic areas.

Conventional medical wisdom teaches that tenderness occurs in the area where pain or discomfort is reported. Thus, the most direct relationship to be expected between tenderness and symptoms is that tenderness is likely to be found in symptomatic areas.

Prevalence of tenderness in symptomatic areas was defined as the ratio of the number of symptomatic areas with tenderness to overall number of symptomatic areas for each subject with symptoms. If the expected relationship were true then this ratio should be close to 1. The following table presents this data comparing the results for the subgroups derived from both the Diagnostic and Occupational classifications.

The following table shows the mean value of the “Prevalence of tenderness in symptomatic areas” in each subgroup. Number of subjects involved in the calculation is included in brackets (), only those subjects with at least one symptomatic area are included.

	Inconsequential Symptoms	Consequential Symptoms	Total
Musicians	0.580 (35)	0.923 (31)	0.741 (66)
Non-musicians	0.875 (8)	0.917 (6)	0.893 (14)
Total	0.768 (43)	0.922 (37)	0.768 (80)

It can be seen that a value close to 1 occurred in the subjects with “Consequential Symptoms”. The subgroup in which symptoms without tenderness was most likely was musicians with “Inconsequential Symptoms”.

A two way analysis of variance was used to assess the statistical significance of differences observed. No significant differences were observed: (Details Appendix G, Table 22)

Two way ANOVA Occupational Classification $p=0.20$

Diagnostic Classification $p = 0.09$

Although there appears to be a trend for higher sensitivity in the symptomatic areas of the individuals in the “Consequential Symptoms” Category this did not reach statistical significance.

4.4.3.2. The prevalence of tenderness in asymptomatic areas.

Conventional medical wisdom leads to the assumption that the presence of tenderness suggests the presence of some pathological process and in consequence the presence of symptoms. Thus, tenderness would not commonly be expected in asymptomatic areas. For the purposes of

4.4.3.3. Prevalence of tenderness in unilateral asymptomatic areas.

There were 5 areas on each side of the body selected for the documentation of distribution of symptoms. In each of these 5 areas symptoms could be absent, unilateral or bilateral. The following table repeated from section 4.4.1.4 gives the mean number of bilateral and unilateral symptomatic areas (range given in brackets) for both the Musicians and Non-musicians categories.

	Musicians (N=66)	Non-musicians (N=14)
Total	2.38 (1-10)	1.57 (1-4)
Bilateral	1.69 (0-10)	0.71 (0-4)
Unilateral	0.68 (0-3)	0.86 (0-2)

The presence of unilateral symptoms allows some interesting observations to be made. This arises from consideration of the contra-lateral area which is asymptomatic. For the purpose of brevity, the term unilateral asymptomatic area was defined as that area contralateral to a unilateral symptomatic area. The special case in which tenderness occurs in a unilateral asymptomatic area provides the following observations. Tenderness in such an area would not conventionally be expected any more than tenderness in any other asymptomatic area. Analysis of the data showed quite the opposite. Such areas were significantly more likely to have tenderness than other asymptomatic areas.

“Prevalence of tenderness in unilateral asymptomatic areas” was defined as the ratio of the number of unilateral asymptomatic areas with tenderness to the overall number of unilateral asymptomatic areas for each subject. The following table shows the mean value for the “prevalence of tenderness in unilateral asymptomatic areas” in the various subgroups. Number of subjects involved in the calculation is included in brackets (), only those subjects with at least one area of unilateral symptoms are included.

This table answers the question that follows: If an individual has unilateral symptoms in one area how likely is the asymptomatic area on the other side of the body, to be tender?

	Inconsequential Symptoms	Consequential Symptoms	Total
Musicians	0.500 (18)	0.861 (18)	0.681 (36)
Non-musicians	1.000 (6)	1.000 (5)	1.000 (11)
Total	0.625 (24)	0.891 (23)	0.755 (47)

If this table is compared with the data for the “prevalence of tenderness in asymptomatic areas” in general it appears that the prevalence is substantially higher in unilateral asymptomatic areas. This table is repeated here from section 4.4.3.2 to facilitate direct comparison.

	No Symptoms	Inconsequential Symptoms	Consequential Symptoms	Total
Musicians	0.188 (52)	0.374 (34)	0.674 (28)	0.363 (114)
Non-musicians	0.140 (43)	0.401 (8)	0.759 (6)	0.241 (57)
Total	0.166 (95)	0.379 (42)	0.689 (34)	0.323 (171)

To test this apparent difference, the two parameters following:

- prevalence of tenderness in unilateral asymptomatic areas = ratio of number of unilateral asymptomatic areas with tenderness to the overall number of unilateral symptomatic areas
- prevalence of tenderness in asymptomatic areas = ratio of number of asymptomatic areas with tenderness to the overall number of asymptomatic areas

were compared, in each suitable individual, with the paired Student’s t-test. This confirmed that in any one individual a unilateral asymptomatic area was significantly more likely to be tender than other types of asymptomatic areas. (Paired Student's t-test, $p < 0.0001$, details Appendix G, Table 24)

Indeed comparison with the following table (repeated from section 4.4.3.1 to facilitate direct comparison), which gives the prevalence of tenderness in symptomatic areas, indicates that the sensitivity of unilateral asymptomatic areas approaches that of symptomatic areas.

	Inconsequential Symptoms	Consequential Symptoms	Total
Musicians	0.580 (35)	0.923 (31)	0.741 (66)
Non-musicians	0.875 (8)	0.917 (6)	0.893 (14)
Total	0.768 (43)	0.922 (37)	0.768 (80)

To test the apparent similarity the two parameters following,

- prevalence of tenderness in unilateral asymptomatic areas = ratio of number of unilateral symptomatic areas with tenderness to the overall number of unilateral symptomatic areas
- prevalence of tenderness in symptomatic areas = ratio of number of symptomatic areas with tenderness to the overall number of symptomatic areas

were compared, in each suitable individual, with the paired Student’s t-test. This analysis failed to show a significant difference in the sensitivity of the unilateral asymptomatic areas from

symptomatic areas in any one individual. (Paired Student's t-test, $p=0.20$, details Appendix G, Table 25)

To ensure that significant variation within one of the subgroups was not missed this test was repeated stratifying for both Occupational and Diagnostic Classification. No significant difference in the sensitivity of the unilateral asymptomatic areas from symptomatic areas in any one individual was found irrespective of stratification. (Details Appendix G, Table 26)

4.5. Cohort Prospective-Analysis

This set of analyses related to the Instrumental Music Students, followed prospectively during their undergraduate studies. The aim of this part of the analysis was to explore the possibility that differences in certain factors might be associated with differences in risk of developing Non-specific Occupational Overuse Syndrome with "Consequential Symptoms".

4.5.1. Demographic Details

There were 95 Instrumental Music Students initially assessed. Of these, 6 were classified as having Non-specific Occupational Overuse Syndrome with "Consequential Symptoms" and 2 were classified as having Specific Occupational Overuse Syndrome. These 8 subjects were excluded from the prospective arm of the study. At the end of three years an attempt to followup the 87 selected Instrumental Music Students was made, 2 students could not be traced, one each from the initial "No Symptoms" and "Inconsequential Symptoms" categories. This left 85 students initially assessed and followed up for the 3 years of undergraduate music study.

At the three year followup review the students were again questioned concerning the presence of symptoms associated with activity, occurring any time during their course. On the basis of this information, classification into the various diagnostic categories was made.

The principle aim of the prospective study was to discover which of the subjects initially with "No Symptoms", or "Inconsequential Symptoms", developed "Consequential Symptoms" during their studies.

Tabulation of Initial against Followup Diagnostic Category.

		Initial Diagnostic Category		Total
		None	Inconsequential	
Followup Diagnostic Category	None	32	6	38
	Inconsequential	12	16	28
	Consequential	7	9	16
	Specific Diagnosis	0	3	3
	Total	51	34	85

This allowed an estimate of the three year incidence of Non-specific Occupational Overuse Syndrome with “Consequential Symptoms” in undergraduate music students to be calculated. Out of 85 students initially without “Consequential Symptoms”, 16 developed such symptoms during a three year period, giving a three year incidence estimate of 19%. This would seem to indicate that, contrary to the suggestion of Ireland [Ireland, 1986a][Ireland, 1988], this condition can not principally be ascribed to the monotony of the tasks involved.

Three subjects developed symptoms associated with a specific diagnosis during the three years of their undergraduate studies. These were consequently excluded from the analysis, leaving 82 subjects in the final comparative analysis of the prospective study.

4.5.2. Analysis of the significance of Diagnostic Classification

The presence of “Inconsequential Symptoms” at the initial assessment did not increase the risk of later development of “Consequential Symptoms”.

To investigate the hypothesis that individuals initially with “Inconsequential Symptoms” are more likely to subsequently develop “Consequential Symptoms” than individuals with “No Symptoms”, a suitable null hypothesis needs to be tested. A simple contingency table Chi Square analysis of the above table implies the null hypothesis of even distribution between cells. Because of the expected effect of initial symptoms on the presence of either “No Symptoms” or “Inconsequential Symptoms” during the music course, this is not a suitable null hypothesis. In order to formulate a more appropriate null hypothesis, it is necessary to consider reasonable expectations based on allocation to the initial categories of “No Symptoms” or “Inconsequential Symptoms” which do not impact on the hypothesis to be investigated. A reasonable expectation is that the majority of those initially with “No Symptoms” would continue to have “No Symptoms” and those initially with “Inconsequential Symptoms” would continue to have “Inconsequential Symptoms”. This is to say that the initial diagnostic classification would be likely to remain unchanged at the followup assessment. The hypothesis to be investigated adds to this base-line expectation the additional expectation that, of those who do develop “Consequential” symptoms, a higher proportion would come from the initial “Inconsequential Symptoms” Category. The null hypothesis suitable to test the validity of this

addition expectation would add to the base-line expectation that the same proportion of individuals would come from both the initial “No symptoms” and the initial “Inconsequential Symptoms” diagnostic categories.

To set up a contingency table suitable to test this null hypothesis, adjustment of the cells in the above table needs to be made. A suitable method to make this adjustment is to reclassify the Followup diagnostic classification as follows:

- A. Consequence of Followup symptoms unchanged from Initial. (either Initial and Followup classification = “No symptoms” or Initial and Followup classification = “Inconsequential Symptoms”)
- B. Consequence of Followup symptoms changed to other lesser degree diagnostic category. (either those initially with No symptoms changed to Followup classification “Inconsequential Symptoms” or those initially with “Inconsequential Symptoms” changed to “No Symptoms”)
- C. Followup symptoms were of "Some Consequence"

This produced the following contingency table:

		Initial diagnostic category		Total
		No Symptoms	Inconsequential Symptoms	
Followup diagnostic category	No change in diagnostic category	32	16	48
	Change to other lesser severity diagnostic category	12	6	18
	Development of Consequential Symptoms	7	9	16
	Total	51	31	82

On the basis of Chi-Squared analysis according to this scheme there was no association between the Initial Diagnostic Classification and the Followup Diagnostic Classification. That is to say, those who initially had “No Symptoms” were as likely to continue to have “No Symptoms”, as those who initially had “Inconsequential Symptoms” would continue to have “Inconsequential Symptoms”. Conversely, those who had “No Symptoms” initially were as likely to develop “Inconsequential Symptoms”, as those who initially had previously had “Inconsequential Symptoms” would subsequently have “No Symptoms”. Those who initially had “Inconsequential Symptoms” were no more likely to develop “Consequential Symptoms” than those who initially had “No Symptoms”. In other words the results did not support the hypothesis that the presence of “Inconsequential Symptoms” increased the risk of developing “Consequential Symptoms”. Chi-square (2 degrees of freedom) = 2.8764 p= 0.2374. In addition this analysis shows that those initially with “Inconsequential Symptoms” had a 19%(6/31) chance of having no further symptoms.

4.5.3. Analysis of tender site data

Neither presence of tender sites nor high number of tender sites nor high prevalence of tenderness in asymptomatic areas, were associated with an increased risk of development of “Consequential Symptoms”.

If the presence of tenderness was taken to be an indicator of the presence of Non-specific Occupational Overuse Syndrome, then it might be expected that those with one or more sites of tenderness would be more likely to develop “Consequential Symptoms” than those without. Contingency table analysis did not confirm this expectation.

(Chi Sq. = 1.43, $p = 0.49$, Details Appendix G, Table 27)

Two features of the initial physical examination were strongly associated with the Diagnostic classification made at the initial assessment. These were the number of sites of tenderness and the prevalence of tenderness in asymptomatic areas. One-way ANOVA was used to test each of these as a possible risk factors for subsequent development of “Consequential Symptoms” during the music course.

Those who subsequently developed “Consequential Symptoms” did not initially have a higher number of sites of tenderness than those who did not develop “Consequential Symptoms”. There was no association between the initial number of sites of tenderness and the Followup diagnostic classification. (ANOVA $p=0.18$) (Details Appendix G, Table 28)

Those who subsequently developed “Consequential Symptoms” did not initially have higher prevalence of tenderness in asymptomatic areas than those who did not develop “Consequential Symptoms”. There was no association between the “Prevalence of tenderness in asymptomatic areas” at the initial assessment and the Followup Diagnostic Classification. (ANOVA $p=0.53$) (Details Appendix G, Table 29)

4.5.4. Analysis of physical fitness

Significant differences in physical fitness parameters were found between males and females, in the study variables:- Aerobic Fitness as given by an estimate of $\dot{V} O_2 \text{ max.}$, Grip Strength, Pinch Strength and Peak Expiratory Flow Rate (PEFR) (Details Appendix G, Table 30). The analysis of the risk associated with these study variables were split so that males and females were analysed independently.

The following table lists a summary of the results of the analyses for the study variables investigating physical fitness, subdivided into male and female where appropriate. Details of these analyses are given in the indicated table in Appendix G.

Study Variable	Sex	Mean Value No Symptoms	Mean Value Inconsequential Symptoms	Mean Value Consequential Symptoms	ANOVA p Value	Details in Table
Aerobic Fitness V O ₂ max.	M	54.9	54.2	55.2	0.97	31
	F	37.6	38.3	37.3	0.68	32
Hours Physical Activity per week		4.1	3.2	4.2	0.72	33
Dexterity		131.6	130.4	132.2	0.85	34
Grip Strength (Max. Kg force)	M	54.5	52.6	54.7	0.89	35
	F	32.5	34.0	29.0	0.03	36
Pinch Strength (Max. KPa)	M	51.5	52.0	52.5	0.95	37
	F	42.7	46.0	42.8	0.26	38
PEFR	M	547.9	595.5	523.0	0.16	39
	F	457.8	442.5	426.0	0.43	40

Posture was assessed as a dichotomous variable, there was no significant difference found between diagnostic categories (Chi Sq= 2.67, p=0.26, Details Appendix G, Table 41)

In females those with lower grip strength had a higher risk of development of “Consequential Symptoms”

The only statistically significant result from the analysis of the physical fitness parameters was with respect to grip strength in females. In females a lower grip strength was associated with an increased risk of developing “Consequential Symptoms” (ANOVA p=0.028) (Details Appendix G, Table 36). This was not found to be an association in males (ANOVA p=0.89) (Details Appendix G, Table 35). Fisher’s PLSD post-hoc analysis showed that in females, those who developed “Consequential Symptoms” had significantly lower grip strength (29.0 Kg) than either those with No Symptoms (32.5Kg) (p= 0.045) or those with “Inconsequential Symptoms” (34.0 Kg) (p = 0.0084). There was not a significant difference between those with No Symptoms cf “Inconsequential Symptoms” (p = 0.36) (Details Appendix G, Table 36). This finding was consistent with the clinical impression made during the study that females who sought treatment for Non-specific Occupational Overuse Syndrome frequently had poorly developed upper-limb musculature.

4.5.5. Analysis of physical attributes

A significant difference between males and females was found in relation to the study variable “Hand Span”, no significant difference was found in relation to the study variable “Body Mass Index” (Details Appendix G, Table 42). The analysis of variance assessing association between the study variable Hand Span and the outcome variable “Followup Diagnostic Classification” was split so that males and females were analysed independently .

The following table lists the results of the analyses for the study variables investigating physical attributes, subdivided into male and female where appropriate. Details of these analyses are given in the indicated table in Appendix G.

Study Variable	Sex	Mean Value No Symptoms	Mean Value Inconsequential Symptoms	Mean Value Consequential Symptoms	ANOVA p Value	Details in Table
Hand Span (mm)	M	229.8	227.3	229.2	0.89	43
	F	202.8	204.0	200.8	0.78	44
Body Mass Index		22.1	22.1	21.9	0.97	45

There were no significant results in these analyses.

4.5.6. Analysis of activity levels at beginning of course.

There was no significant difference between the number of hours of musical instrument performance per week undertaken by males compared with females at the beginning of their course. (Details Appendix G, Table, 46) A significant difference in the mean value of this study variable was found between Followup Diagnostic Categories.

Study Variable	Mean Value No Symptoms	Mean Value Inconsequential Symptoms	Mean Value Consequential Symptoms	ANOVA p Value	Details (Table)
Hours music performance per week	16.6	22.6	17.1	0.017	47

Fisher's PLSD post-hoc analysis showed:-

a significant difference between the "No Symptoms" and "Inconsequential Symptoms" categories ($p=0.0063$).

no significant difference between the "No Symptoms" and "Consequential Symptoms" categories ($p=0.82$).

no significant difference between the "Inconsequential Symptoms" and "Consequential Symptoms" categories ($p=0.051$).

4.5.7. Analysis of Psychological data.

Average psychological profiles for all the diagnostic categories were all within the normal range.

Analysis of the scores from the psychological questionnaires found no significant sex differences (Details Appendix G, Table 48). Analysis of the distribution of the scores from the psychological questionnaires found that all psychological factors assessed except the General Health Questionnaire score, the Illness Behaviour Questionnaire factor "General Hypochondriasis" and the Illness Behaviour Questionnaire factor "Disease Conviction" conformed to the conditions required of "normal" distribution for the purposes of statistical analysis to allow the use of parametric Analysis of Variance (ANOVA). Kruskal-Wallis non-parametric ANOVA was consequently used for these three factors which were not normally distributed (Test of normal distribution as set out in MiniTab Manual, reference [Shapiro and Wilk, 1970] Details Appendix G, Table 49).

The means of the diagnostic classification categories for all scores were within the range of the normal population as defined by the normative data set out in each questionnaire's instruction manual. Although the means of all the study variable assessing psychological profiles were within the normal range, Analysis of Variance demonstrated that there were some significant differences between diagnostic categories. The following tables give details of the differences in the mean values between diagnostic categories for the study variables investigating psychological profiles as well as the relevant ANOVA p-value and the "Normative Values" taken from the individual Questionnaire Instruction Manuals.

General Health Questionnaire (GHQ) (Standard Error of the Mean in brackets adjacent to mean values). Higher values indicate increasing neurotic tendencies.

Study Variable	Mean Value No Symptoms	Mean Value Inconsequential Symptoms	Mean Value Consequential Symptoms	ANOVA p Value (Details)	Normative Values (London 15 to 24 year olds)
GHQ	3.18 (0.69)	5.79 (1.36)	4.88 (1.57)	0.28	9.64

Details of Analysis of Variance given in Appendix G, Table 50.

Speilberger Anxiety Questionnaire (SAQ) (Standard Error of the Mean in brackets adjacent to mean values). Higher values indicate increasing tendency towards anxiety.

Study Variable	Mean Value No Symptoms	Mean Value Inconsequential Symptoms	Mean Value Consequential Symptoms	ANOVA p Value	Normative Values (American College Freshmen)
SAQ	35.1 (1.29)	38.8 (1.68)	37.6 (2.21)	0.21	38.17

Details of Analysis of Variance given in Appendix G, Table 51

Illness Behaviour Questionnaire (IBQ) (Standard Error of the Mean in brackets adjacent to mean values).

IBQ Factor	Mean Value No Symptoms	Mean Value Inconsequential Symptoms	Mean Value Consequential Symptoms	ANOVA p Value	Normative Values (General Practice Patients)	Normative Values (Pain Clinic Patients)
GH	0.87 (0.27)	1.89 (0.35)	1.36 (0.39)	0.009	1.42	1.79
DC	0.89 (0.18)	1.11 (0.15)	1.21 (0.26)	0.20	1.58	3.72
PvSC	2.00 (0.10)	2.36 (0.18)	1.64 (0.17)	0.013	2.01	0.62
AI	2.49 (0.26)	1.89 (0.33)	2.50 (0.49)	0.33	2.46	2.51
AD	1.00 (0.21)	1.93 (0.33)	1.86 (0.47)	0.038	2.33	2.61
D	3.03 (0.18)	3.18 (0.23)	3.21 (0.30)	0.81	2.91	3.88
I	0.89 (0.21)	1.00 (0.21)	1.50 (0.27)	0.26	2.81	3.11

Details of Analysis of Variance given in Appendix G, GH Table 52, DC Table 53, PvSC Table 54, AI Table 55, AD Table 56, D Table 57, I Table 58)

Three factors in the "Illness Behaviour Questionnaire" showed significant variation between different diagnostic categories.

Using the appropriate analysis of variance to test for differences between the means for the three diagnostic classifications of Non-specific Occupational Overuse Syndrome, found three factors in the "Illness Behaviour Questionnaire" with which there were significant differences.

A. There were significant differences between diagnostic categories on the psychological profile "General Hypochondriasis" (Phobic concern about one's state of health), measured with the Illness Behaviour Questionnaire. The Kruskal-Wallis non-parametric one-way ANOVA test was used, $p=0.009$. The Mann-Whitney non-parametric U-test was used to assess post hoc between group differences. The "Inconsequential Symptoms" category had a higher score (1.89) than the "Consequential Symptoms" category (1.36) but this was not significant $p=0.39$. The "Consequential Symptoms" category had a higher score than the "No Symptoms" category (0.87) but this also was not significant $p=0.10$. The subsequent difference between the "Inconsequential Symptoms" category and the "No symptoms" category was significant however, $p=0.003$. (Details Appendix G, Table 51)

Thus the order of difference between mean value of this study variable for the diagnostic categories was "Inconsequential Symptoms" > "Consequential Symptoms" > "No Symptoms" with a significant difference only shown between the extremes.

B. There were significant differences between groups on the psychological profile "Psychological vs Somatic Concern" (PvSC), measured with the Illness Behaviour Questionnaire. The standard parametric ANOVA test was used, $p=0.013$. Fisher's PLSD post-hoc test was used to assess between-group differences. The "Inconsequential Symptoms" category had a higher score (2.36) than the "No Symptoms" category (2.00) but this was not significant $p=0.058$. The "No Symptom" category had a higher score than the "Consequential Symptoms" category (1.64) but this also was not significant, $p=0.13$. The subsequent difference between the "Inconsequential Symptoms" category and the "Consequential Symptoms" category was significant however, $p=0.004$. (Details Appendix G, Table 53)

Thus the order of difference between categories was "Inconsequential Symptoms" > "No Symptoms" > "Consequential Symptoms" with a significant difference only shown between the extremes.

A high score in the factor PvSC indicates that the individual felt somehow responsible for his illness and felt in need of psychiatric, rather than medical, treatment. A low score indicates the contrary attitude with a tendency to somatise concerns. The mean value of the score for the "Inconsequential Symptoms" category was high, the mean value of the score for the

“Consequential Symptoms” category was low and the mean score for the “No Symptoms” category was intermediate. There was a statistically significant difference between the “Inconsequential Symptoms” and the “Consequential Symptoms” diagnostic categories.

C. There were significant differences between categories on the psychological profile "Affective Disturbance" (Tendency towards anxiety and / or sadness) measured with the Illness Behaviour Questionnaire. The standard parametric ANOVA test was used, $p= 0.038$.

Fisher's PLSD post-hoc test was used to assess between-group differences. The “Inconsequential Symptoms” category had a higher score (1.93) than the “Consequential Symptoms” category (1.86) but this was not significant $p= 0.89$. The “Consequential Symptoms” category had a higher score than the “No Symptoms” category (1.00) but this also was not significant, $p= 0.080$. The subsequent difference between the “Inconsequential Symptoms” category and the “No symptoms” category was significant however, $p= 0.018$. (Details Appendix G, Table 55) Thus, the order of difference between groups was “Inconsequential Symptoms” \geq “Consequential Symptoms” $>$ “No Symptoms” with a significant difference only shown between the extremes.

4.6. Summary

The failure to detect significant effects from a number of factors investigated, should not be taken as necessarily indicating that there was no effect from any of these factors. This study was exploratory in nature and as such, can be seen in many ways to be taking the form of a pilot study. That is to say, there was not pre-existing data to show the expected variance in the factors measured and so the power of the study to detect an effect in the study variables, could not be determined in advance. In statistical terms, this is to say that the risk of type 2 errors could not be calculated for the study variables in advance. Consequently, it is quite possible that alternative choice of study variables, better precision in their measurement, or greater number of subjects, may well have demonstrated effects not detected in this study.

There were significant differences found, however, between the out-come variable categories in a range of study variables. These results do demonstrate a number of significant features with regard to parameters considered to be of importance with respect to the assessment of Non-specific Occupational Overuse Syndrome. The implications of these findings are discussed in the next chapter.

Chapter 5. Discussion.

5.1. Introduction

Before turning to a detailed discussion of the research results, it is useful to observe how, in general terms, these findings bear on what might be seen as the key hypotheses or conclusions in the literature.

5.1.1. Critical conclusions of previous hypotheses

Dividing the hypotheses into those which propose physical pathology, those which propose psychological pathology and those who suggest that there is no pathology, it can be said in overview that previous workers have concluded:

(physical pathology)

- there is a clear, objective difference between the “normal”, unaffected population and Non-specific Occupational Overuse Syndrome “patients” in terms of the presence of pain and/or tenderness. This “normal” population is assumed to be free of pain and/or tenderness and consequently clinical examination should be able to discriminate between those with non-specific occupational overuse syndrome and this “normal”, unaffected population.
- following from the previous point, the presence of tenderness is an indicator of the presence of a local pathology which is likely to progress to a more severe state. Thus, to have discovered tender sites is to have discovered an early stage of a pathology which is likely to intensify.

(psychological pathology)

- the symptoms of non-specific occupational overuse syndrome do not have physical causes but are the result of a psychological disorder such as conversion hysteria, compensation neurosis or a neurotic exaggeration of normal sensations.

(no pathology)

- the symptoms of non-specific occupational overuse syndrome are either a pretence or an exaggeration of the minor aches and pains of normal fatigue.

5.1.2. Key Findings of this study

With reference to these general conclusions, the key findings of the study were as follows:-

- a significant proportion of the “normal” population - those who reported no symptoms - had sites of tenderness.

- a significant group of subjects had not changed their behaviour in any way nor sought medical intervention but had, nevertheless, experienced pain and discomfort associated with occupational activity and did have sites of tenderness upon examination.
- the data did not show qualitative differences between diagnostic categories but demonstrated a continuous variation in features as the study moved from the “unaffected” group, through those with “Inconsequential” to those with “Consequential” symptoms.
- the magnitude of the variation within the data was such that there were significant differences apparent between categories.
- those with “Inconsequential” symptoms were no more likely to go on to experience “Consequential” symptoms than those without symptoms.

5.1.3. Implications of the findings of this study on previous hypotheses

Clearly the discovery of the sub-group of asymptomatic subjects with tender sites (56% of asymptomatic subjects) and the identification of the “Inconsequential” symptoms category created a problem, as much of the previous research assumed or concluded that there is an objective, qualitative distinction between the unaffected population and “patients”. These two groupings suggested that reconsideration should be given to the notion of “normality” as it had previously operated in the literature. Presumably, the previous research would still classify the first sub-group - asymptomatic subjects with tender sites - as “normal”, as belonging to the category of “non patient”. And yet the presence of tender sites suggested that there were some grounds for seeing this grouping as “affected” in some way, as sharing a key feature with subjects who were viewed unproblematically as “patients”. The second grouping - those classified as belonging to the “Inconsequential” symptoms category was ambiguous in terms of classification. Although the individuals in this group appeared closer to “patients” than to unaffected individuals in that they reported symptoms, they had not altered their routine or sought medical intervention. They appeared to be “normal” from one perspective and “patients” from another.

The discovery of the first grouping clearly challenged the assumption or conclusion that the “normal” population is free from sites of tenderness. It would be possible to accommodate these findings by reclassifying the members of this asymptomatic sub-group as “abnormal” in some way, by suggesting they represented a preclinical degree of Non-specific Occupational Overuse Syndrome where the pathological process had commenced but was not yet clinically apparent. The problem with this approach is that it requires the classification of over 50 percent of the “normal” population which was sampled in this study as “abnormal” or diseased. The conclusion that must be reached by making this classification is that over 50% of percent of the student population is suffering the preliminary stages of Non-specific Occupational Overuse Syndrome . For reasons explored in more detail below, this was rejected and these results were construed as supporting the contrasting conclusion that there is a need to abandon, or at least

doubt, the assumption or conclusion that the presence of tender sites is necessarily “abnormal”, necessarily indicative of pathology. That is, the findings suggest that it is possible for “normal” individuals to share a feature with “patients” and yet not be thereby classified as diseased.

It must be allowed that there is another possible explanation of these findings. The “normality” of the asymptomatic sub-group with sites of tenderness might be maintained by seeking to differentiate their sites from those found in “patients”- that is, by contending that their sites were in some way qualitatively different and hence did not indicate pathology.

The experience gained from systematic examination for tender sites in all subjects did not provide any reason to think that the process causing tenderness in some of the asymptomatic group was any different from that causing the tenderness found in those with Non-specific Occupational Overuse Syndrome in either the “Inconsequential” or “Consequential” categories. The finding of tenderness in asymptomatic areas was initially surprising and always surprised the subjects no matter which diagnostic category they came from. As a consequence, the explanation that these sites are in some way qualitatively different was rejected.

The second grouping - subjects with sites of tenderness classified as belonging to the “Inconsequential” symptoms category - posed a similar problem of classification. Depending on how much weight was attached firstly to the presence of tenderness and secondly to the reporting of symptoms, this grouping could be classified either as “normal” or as “patient”. Once again, this tenderness could be accounted for in either of two ways: (1) by classifying the group with “Inconsequential” symptoms as “abnormal” on the basis of this tenderness or, alternatively (2) by abandoning the assumption that “tenderness” is necessarily incompatible with “normality”. There could be a similar response to this group’s reporting of symptoms. It could be interpreted as an indicator of “abnormality”, possibly suggesting that these individuals represented a less advanced stage of the syndrome’s development. Alternatively, the assumption that symptoms necessarily indicate “abnormality”, or the presence of pathology, could be abandoned or, at least, put at issue. This is the explanation adopted and argued for at length below.

Once again it must be allowed that there is another possible explanation. It could be argued that this second group’s symptoms were qualitatively different from those experienced by “patients” and hence maintain their “normality” or “non-patient” status. Once again this was rejected. Just as no difference was found between the sites of tenderness of all subjects, similarly the characteristics of the symptoms and the relationship to activity did not appear to differ between the groups with “Inconsequential” and the “Consequential” Symptoms. The only difference seemed to be the severity and persistence of the symptoms.

Another key finding was that those with more tender sites were no more likely to develop “Consequential” symptoms than those with fewer or no tender sites. This provided compelling

support for the conclusion that the presence of tender sites is not indicative of the early stages of a pathology which is likely to increase in severity. It also lends support to the conclusion that the presence of tender sites need not be seen as indicating "abnormality" or pathology. If tenderness were abnormal and pathological then it could be expected that its presence would be associated with an increased risk for the development of "Consequential" symptoms - whereas the lack of this is more indicative of stability or "normality".

This study's findings provide a similarly strong challenge to the psychological hypotheses. The discovery of the tender sites in the asymptomatic subjects is directly damaging to these explanations. The psychological hypothesis argues for a diagnosis of conversion hysteria. It contends that the subject "converts" or expresses some psychological problem or neurosis as physical symptoms. But since there were no symptoms for this grouping, conversion hysteria could not, by definition, be used to explain the presence of the tender sites. The subjects typically had no "experience" of the tender sites until subjected to clinical examination.

The second grouping - those with "Inconsequential" symptoms - poses a somewhat similar problem for the psychological hypothesis. Conversion hysteria is held to operate because the affected subject benefits in some way from the "converted" symptoms. The presence of the symptoms enables them to avoid or diminish some psychological crisis. But members of this second group, those with "Inconsequential" symptoms, did not change their behaviour in any way. Their experiencing of symptoms did not enable them to escape psychological conflict or need. As a consequence the notion of "conversion hysteria" was robbed of any explanatory power in this instance.

The conclusions reached so far suggest that it is normal to be able to elicit tenderness with or without the presence of symptoms and for some degree of aches and pains to occur as a normal part of life. In addition, the results suggest that the nature of both the discomfort and tenderness associated with Non-specific Occupational Overuse Syndrome be qualitatively the same in all diagnostic categories ("No Symptoms", "Inconsequential Symptoms" and "Consequential Symptoms"). These conclusions would seem to support the hypothesis that no pathology is present. Authors who previously supported this hypothesis maintained, however, that either symptoms were feigned (malingering) or that the symptoms were due to exaggerated importance placed on minor aches and pains associated with fatigue.

In musicians and in music students in particular it is difficult to imagine reasons for malingering to occur more than very occasionally. Pretended exaggeration of minor discomforts would be likely to hinder students chances of advancement in their chosen careers. Musicians as a group most commonly thrive on any opportunity to perform. Any suggestion that they might not be able to continue to play as much as they wish is seen as a severe penalty. Among the musicians treated in this study, although the possibility was always considered, there was not one instance

where a secondary gain could be uncovered which might lead to such behaviour. Certainly the data collected in this study could not be ascribed to the effects of malingering.

More recently Parker and Denzin [Parker and Denzin, 1990a] re-stated the fatigue hypothesis, "Repetition Strain Injury is a musculo-skeletal fatigue syndrome". They offered an example of pain obviously caused by muscle fatigue to justify this hypothesis. "If one were to perform an experiment and to stand up and to raise the arms sideways with the hands level with the shoulders and to maintain that position after a short while, the shoulders will begin to ache and the arms feel heavy. Then (if continued) the pain would increase". They claim that in their experience it is "readily reversible... If appropriate attention is paid to the ergonomic, fatigue and postural features and to the restoration of normal muscular balance." They suggested that if these measures are not effective then a psychological cause should be considered. "In persisting cases, features of secondary gain should be resolved." Can the symptoms of Non-specific Occupational Overuse Syndrome be attributed to the pain associated with the physiological process of muscle fatigue?

What then is muscle fatigue and how can it cause pain? Muscle fatigue has been defined as "failure to sustain force or power output" [Edwards, 1986]. Edwards discussed some of the theories as to what physiological changes could be occurring to cause this failure. In summary, it could either be due to central causes, central inhibition of motor neurone activity, or due to peripheral causes, failure of the muscle fibres to contract subsequent to a motor nerve impulse.

Which factors generate the pain which is associated with muscle fatigue and is this source of pain consistent with the signs and symptoms observed in the majority of cases Non-specific Occupational Overuse Syndrome? Awerbuch [Awerbuch, 1990] suggested that the cause of pain in the example of fatigue suggested by Parker and Denzin was that of local muscle ischaemia. He implied that the repetitive actions commonly associated with Non-specific Occupational Overuse Syndrome do not develop the tension required to produce the degree of muscle ischaemia required to produce pain and so the example of Parker and Denzin had no relevance to this problem. Valencia, however, argued that over prolonged periods of continuous sub-maximal activity intra-muscular blood flow may be compromised [Valencia, 1986]. The advocates of the fatigue theory expect the results of local muscle fatigue, that is, muscle failure and associated discomfort, to be directly related to the activity causing the muscle fatigue, to be temporary and to resolve with rest. "On ceasing the exercise and relaxing, the symptoms will be relieved" [Parker and Denzin, 1990a]. "Repetition is the essence of conditioning and recovery from fatigue is rapid and total." [Bloch, 1984]

These features of pain associated with muscle fatigue do not conform to the overall picture of the symptoms and signs associated with Non-specific Occupational Overuse Syndrome. Sites of tenderness which persist over time and can be observed both in the absence of recent activity which might cause fatigue and also in the absence of symptoms, seem unlikely to be caused by

a fatigue process. The presence of symptoms which frequently occur in the absence of any objective or subjective evidence of muscular failure is also evidence against physiological fatigue as being the primary factor in the development of the symptoms of Non-specific Occupational Overuse Syndrome.

The following is a discussion of the results of the study in detail with consideration of the hypotheses presented in Chapter 3, section 3.3.

5.2. Hypotheses tested with use of data from the Cross-sectional analysis.

5.2.1. Hypothesis 1.

The first hypothesis was that the musculo-skeletal structures of “normal” individuals are non-tender. The expectation prior to commencement of data collection was that asymptomatic individuals would be non tender. In this study, however, the presence of tenderness was found in 56% of the asymptomatic first year university students, both from the music and non-music student groups. For this hypothesis to be true and consistent with this finding it must be concluded that 56% of the asymptomatic first year university students were “abnormal”. If, however, this conclusion is not accepted and the presence of tenderness in 56% of the asymptomatic first year university students is taken to be inconsistent with the first hypothesis, then the implication is that the presence of tenderness on physical examination should not necessarily be considered an abnormal finding. It does, however, raise the question as to the significance of tenderness in “normal” individuals.

Confirmation of this high prevalence of tenderness in asymptomatic individuals was found in two previous studies. Sola et al reported a survey of 200 asymptomatic individuals and found that “54% of the women and 45% of the men had latent trigger points (sites of tenderness without symptoms) in the shoulder girdle muscles” [Sola et al, 1955]. A study by Yunus et al reported finding at least one tender point in 48% of the subjects in a group of 50 asymptomatic individuals [Yunus et al, 1981].

5.2.2. Hypothesis 2.

The second hypothesis was that the musculo-skeletal structures of “normal” individuals are not painful. This study found that 37% of first year Instrumental Music Students and 10% of the first year students from other courses experienced minor levels of pain associated with activity (classified as having “Inconsequential symptoms”). In order to accept this hypothesis one must therefore conclude that 37% of first year Instrumental Music Students and 10% of the first year students from other courses were abnormal. The alternative conclusion is to reject this hypothesis and to conclude that the experience of “Inconsequential” symptoms is a common

experience of “normal” individuals. The significance of such “Inconsequential” symptoms in “normal” individuals then becomes a further issue which remains to be explained. The fatigue theory is at least consistent with the minor aches and pains experienced in the “Inconsequential Symptoms” category but an additional factor must be evoked to explain the progression of minor pain to severe pain as experienced by those in the “Consequential Symptoms” category. It was not possible to conceive of a reason for malingering or conscious exaggeration in most of the music students who reported such symptoms. Quite the reverse, they were often very concerned that they might miss a concert, music camp or even a tour with one of the music groups. There was no reason for them to consciously wish for their problem to appear worse than it was. Nevertheless, it was apparent that while they continued with the activities they were engaged in without any alteration the symptoms were becoming progressively more severe.

The prevalence of “Inconsequential” Symptoms in this study is consistent with that found by other investigators. Hadler has stated that the overall prevalence of neck/arm pain in adults is 10% [Hadler, 1985]. Gow reported a survey of clerical and keyboard workers in New Zealand in which the prevalence of musculo-skeletal symptoms was 50% [Gow, 1987].

5.2.3. Hypothesis 3.

The Instrumental Music Students had a higher prevalence of symptoms than the Students from other courses (37% cf 10%) and this could be shown to be related to the activities of music making (Tables 20-25, chapter 4). This data is consistent with the third hypothesis, that those involved in activities associated with higher levels of upper-limb activity will have a higher prevalence of symptoms consistent with a diagnosis of Non-specific Occupational Overuse Syndrome.

5.3. Hypotheses tested with use of data from the Case-Control analysis.

5.3.1. Hypothesis 4.

It was not possible to develop a control group in which it would be possible to determine the frequency with which changes in activity were not associated with the onset of symptoms. Consequently it was not possible to directly determine the impact of changes in activity on the development of symptoms of Non-specific Occupational Overuse Syndrome. Nevertheless, the frequency with which the onset of symptoms was associated with changes in activity in the symptomatic categories was determined. This association was seen in 35% of those in the “Inconsequential” diagnostic category and 54% of the “Consequential” diagnostic category. The lack of controls means that the significance of this observed association remains unclear. There was a strong impression, however, that the change in activities was an aetiological factor

in the onset of symptoms in a large proportion of cases. This result is not inconsistent with hypothesis 4, that change in activity levels increases risk of Non-specific Occupational Overuse Syndrome.

5.3.2. Hypothesis 5.

Although there were significant differences between the mean number of sites of tenderness in the three diagnostic categories there was a considerable range of values in each category. This was demonstrated graphically in section 4.4.2.2, Figure xx (Page 66) with a graph showing the distribution of the number of tender sites in each diagnostic category. As an illustration of the overlap between the categories, there were 23% of the “No Symptoms” category, 53% of the “Inconsequential Symptoms” category and 46% of the “Consequential Symptoms” category, within the range 5 to 14 sites of tenderness. Consequently, neither the presence of tenderness nor the actual number of sites of tenderness could be used as a clear indicator as to which category an individual belonged. Thus, the data does not strongly support the fifth hypothesis, that clinical examination is able to discriminate between those with Non-specific Occupational Overuse Syndrome and those not affected on the basis of the presence of tender sites. Fry’s claim that the presence of tenderness in the muscles and ligaments around the hand and wrist “are virtually diagnostic of overuse injury” [Fry, 1986a] must be seriously questioned.

A similar finding was made when observing the prevalence of tenderness in asymptomatic areas. Although there were significant differences between the mean prevalence of tenderness in asymptomatic areas, between the three diagnostic categories there was a considerable range of values in each category. Consequently, the prevalence of tenderness in asymptomatic areas could not be used as a clear indicator as to which category an individual belonged.

One of the central criteria required to make a diagnosis of conversion hysteria is that psychological factors must be judged to be aetiologically related to the symptom. The group of asymptomatic individuals with tenderness had no symptoms and, prior to their assessment, had no awareness of any abnormality. It is not possible to propose a psychological mechanism as the origin of the phenomena observed in this group. For the data to be consistent with the theory of conversion hysteria, it must be concluded that there is a qualitative difference in the nature of the tender sites found in this group.

The group with “Inconsequential” symptoms had not considered those symptoms to be of any consequence. The symptoms had not been associated with a change in their activities, their interaction with others or indeed anything which could be interpreted as being associated with some psychological conflict or need. Consequently, the process causing the symptoms in this group could not be conversion neurosis. Thus, for the data to be consistent with the theory of conversion hysteria in the Non-specific Occupational Overuse Syndrome, as it affects those whose lives are affected by their symptoms (those with “Consequential” symptoms), the

conclusion must be that there is a qualitative difference in the nature of the process causing symptoms in the "Inconsequential Symptoms" category.

At this stage it seemed that the process observed in this study, which certainly seemed to be consistent with the Non-specific Occupational Overuse Syndrome as described by other researchers, could not be adequately explained by the Conversion Hysteria theory.

It is, also, difficult to accept the required conclusions necessary to make the data consistent with the theory of local pathology. It would have to be accepted that only the 42 students with no symptoms and no sites of tenderness out of a total of 148 first year university students initially assessed (28%), were "normal" or in other words unaffected by the pathological process which caused the symptoms of Non-specific Occupational Overuse Syndrome.

5.3.3. Hypothesis 6.

An important finding was that, in 48% (27 of 56) of the individuals presenting for treatment of symptoms consistent with the Occupational Overuse Syndrome, a specific diagnosis was found. Once the cases in which specific diagnoses had been identified were excluded, however, the remaining cases, where no specific diagnosis could be made (Non-specific Occupational Overuse Syndrome with "Consequential" symptoms), did seem to demonstrate consistent features. Contrary to previous expectations these features did not show discrete differences from the other diagnostic categories defined for Non-specific Occupational Overuse Syndrome but seemed to demonstrate a continuous but significant variation of features. The data seemed to suggest that the phenomena which were observed were due to a single process directly related to the activities with which it was associated and this process seemed to be present at varying levels of expression across all diagnostic categories of Non-specific Occupational Overuse Syndrome. Thus, the hypothesis that the majority of cases of Non-specific Occupational Overuse Syndrome are caused by a single process, was supported.

5.4. Hypotheses tested with use of data from the Prospective analysis.

5.4.1. Hypothesis 7.

Despite the fact that there was a significant difference in the mean number of sites of tenderness between diagnostic categories, the number of sites of tenderness present at the beginning of the prospective study was not shown to be useful in predicting who was more likely to develop symptoms at a later time. Thus, the data is inconsistent with hypothesis number 7, that the presence of tender sites increases the risk of later development of Non-specific Occupational Overuse Syndrome. The data does not support the proposition that tender sites indicate the

presence of, and are a measure of the severity of physical pathology which, if present, is likely to eventually progress to a more severe state.

5.4.2. Hypothesis 8.

The data has shown that the presence of "Inconsequential" symptoms does not increase the likelihood of subsequent development of "Consequential" symptoms. Thus the data does not support hypothesis 8, that "Inconsequential" symptoms increase the risk of developing "Consequential" symptoms of Non-specific Occupational Overuse Syndrome. The data can be said to be inconsistent with the proposition that the presence of "Inconsequential" symptoms is an indicator of an early stage in the pathological process which, if present, is likely to eventually progress to a more severe state.

5.4.3. Hypothesis 9.

The exploratory nature of this study is most apparent when considering the investigation of this hypothesis. Seven factors which were considered to be aspects of physical fitness were studied to discover whether or not low values were associated with higher incidence of Non-specific Occupational Overuse Syndrome.

Out of these 7 possible factors, one aspect of physical fitness was found to be associated with the development of Non-specific Occupational Overuse Syndrome, that is weakness of grip. There is, however, a complication regarding this. The association between weakness of grip and increased risk of developing Non-specific Occupational Overuse Syndrome was only demonstrated for females and not males. As expected males had a significantly higher mean grip strength than the females (53.7 vs 32.2 Kg force; Student's t-test $p < 0.0001$). This could be due to genetic factors or developmental factors or both. It could be that there is a threshold effect on whether grip strength is a risk factor or not and that all of the males in the study had grip strength above this threshold. Certainly the lowest male grip strength was 38 Kg force, which was greater than the mean value for the females. Thus, it might be that, for the males in the study, it was not possible to observe grip strength as a risk factor. Nevertheless, the fact that a physical variable has been shown to be a significant risk factor seems important. It suggests that, whatever process is operating, physical factors play a part. Thus, the data does support hypothesis 7. At least one aspect of increased physical fitness decreases risk of Non-specific Occupational Overuse Syndrome.

5.4.4. Hypothesis 10.

Two physical attributes were measured to investigate whether or not they were associated with an increased incidence of Non-specific Occupational Overuse Syndrome. These were the width of the subjects hand span and their Body Mass Index. No significant association was found.

The music students included in the study played a wide range of instruments. There were too few subjects, however, to analyse the study variables for each musical instrument individually, consequently the possibility that certain physical attributes are important for some instruments and not for others could not be assessed. This study only collected data for two such factors and there may well be others that are of importance.

Although the results failed to find evidence in favour of hypothesis 10, that certain physical attributes increase risk of Non-specific Occupational Overuse Syndrome, there is inadequate data with which to argue against it. Intuitively it would seem to warrant further investigation.

5.4.5. Hypothesis 11.

The study variable, "Number of hours of instrumental music performance per week" reported by the music students at the time of the University Health Service "Health Check", showed significant variation between diagnostic categories of Non-specific Occupational Overuse Syndrome. The mean value of this study variable was significantly greater for the "Inconsequential symptoms" category than either the "No symptoms" or "Consequential symptoms" categories. The implications of this finding are somewhat difficult to interpret. Unfortunately this parameter was not particularly appropriate for the investigation of hypothesis 11, that high activity levels increase risk of Non-specific Occupational Overuse Syndrome for the reasons outlined below.

The information for this study variable was collected during the first year of the music students' undergraduate studies at the University Health Service "Health Check", which occurred at the beginning of the course. Activity levels could change from day to day and week to week and would be affected by many factors. In order to determine whether or not high activity levels increase risk, it would be necessary to know the maximum levels of activity undertaken during the course. The value of the study variable actually measured (the activity level at the beginning of the course) need not bear a direct relationship to the maximum level of activity. Although direct correlation between the two parameters is unlikely, it is likely that there is some form of relationship. The nature of this relationship is not possible to determine precisely and so any conclusions made from the analysis of this study variable, which would depend on the nature of this relationship, must remain conjectural. One possible relationship is that those students who were already in the habit of high levels of activity might have been more likely to continue with high levels during a degree music course and that some of those who initially had lower levels of activity might have found that subsequent demands of the course led them to increase their activity levels. Based on this proposed relationship it is possible to suggest an explanation of the findings from the analysis of this study variable as follows. Continued high levels of activity might be associated with increased risk of "Inconsequential" symptoms and a change from low levels to high levels of activity might be associated with an increased risk of

“Consequential” symptoms. It must be reiterated that these suggested associations remain purely a matter of conjecture.

Although the null hypothesis of no association between the “No. of hours of instrumental music performance per week undertaken at the beginning of the course” and the “followup diagnostic classification” was rejected, these results can not be said to confirm hypothesis 11 because, in retrospect, the study variable used can not be said to be a direct indicator of either maximum activity levels or the level of activity occurring at the time at which symptoms developed. Nevertheless, it does seem to suggest that activity levels are an important factor in the development of symptoms.

5.4.6. Hypothesis 12.

The study has identified three psychological factors as possible risk factors.

1. General Hypochondriasis (fear of disease) would seem to act indirectly by making individuals more aware and anxious of “Inconsequential” symptoms as well as causing them to react more strongly to the same level of symptoms. It is interesting to note that the “Inconsequential” Symptoms category had the highest mean value for General Hypochondriasis. An explanation for this could be that, given a group of people who have experienced very minimal symptoms, those with a high score for General Hypochondriasis are more likely to recall those symptoms than those with a low score. Thus, at the border between the categories “No Symptoms” and “Inconsequential Symptoms”, whether or not an individual reports symptoms leading to a classification into one or other of the categories is, in part, determined by this tendency towards a high score of General Hypochondriasis. The mean score of the “Consequential Symptoms” category lies almost midway between that of the “Inconsequential Symptoms” category and the “No Symptoms” category. An explanation for this may be that those with severe symptoms will have their activities affected irrespective of their tendency towards General Hypochondriasis. Thus, this pattern of association would seem to suggest that this psychological factor does not have a direct effect on the presence or severity of symptoms but affects the way in which an individual responds to symptoms if they do occur.
2. The factor “Affective Disturbance”, is most characterised by a tendency towards dysphoria (anxiety and depression). This factor would seem to act by directly increasing the risk of development of symptoms, but not affecting whether the symptoms are expressed as “Consequential” or “Inconsequential”. There was minimal difference between the mean score for the “Inconsequential” and “Consequential” Symptoms categories but there was a significantly larger difference between these categories and the “No Symptoms” category. An explanation for this may be that the presence of psychological tension or anxiety is associated with increased physical tension [Wigley, 1990a][Salminen et al, 1991][McPhee, 1983]. Thus a tendency for increased psychological tension or anxiety is associated with an increased tendency

towards physical tension. The combination of increased physical tension and excessive activity could act synergistically to amplify the effect of the process which causes activity related pain.

Evidence was found in a recent study by Moulton and Spence [Moulton & Spence, 1992] that when psychological tension or anxiety is associated with increased physical tension, pain associated with activity is more likely. This study found that a group of 14 musicians who experienced pain related to activity, responded to a stressful physical task with increased muscle tension, whereas a control group of 14 musicians who did not have activity related pain did not develop increased muscle tension as a response to a similar stimulus.

3. The influence of the psychological tendency "Psychological vs Somatic Orientation" seems also to act indirectly by affecting an individual's response if symptoms do occur. The mean value of this parameter for the "No Symptoms" category lay mid-way between the means for the "Inconsequential" and the "Consequential Symptoms" categories. An explanation for this could be that if no symptoms are present then this psychological tendency has no effect. If however symptoms do occur then an individual's response to those symptoms will be in part determined by his/her perception of those symptoms. Those who perceive the symptoms to be the result of psychological causes (those who have a tendency towards a higher score on this factor) would be likely to ignore the symptoms and not seek medical help. Consequently, their symptoms would be classified as being of no consequence (Inconsequential). Those who tend to assume that the symptoms have a physical cause (those who have a tendency towards a lower score) would be likely to seek medical advice. Consequently, their symptoms would be classified as being of some consequence (Consequential).

How can the results from measurement of these psychological factors be reconciled with the previous theories?

Only one of the factors, shown by previous research to be indicative of conversion neurosis, was found to be of importance in relation to Non-specific Occupational Overuse Syndrome, ie. "Psychological vs Somatic Orientation". The expected result based on the theory of conversion neurosis is that higher levels of somatization would be associated with greater severity of symptoms. In this study, however, "Psychological vs Somatic Orientation" does not correlate with the severity of symptoms but conversely varies in a non-linear pattern indicating that there is variability in the way in which individuals with symptoms will react to those symptoms. Thus, the results of the assessment of psychological profiles do not strengthen the case for the theory of conversion neurosis.

It would seem that these psychological factors are neither evidence for nor against the theories of local pathology. They do, however, indicate that the diagnostic classification which has been identified for the purposes of this study, does not simply specify a degree of severity of symptoms. The sub-classification of Non-specific Occupational Overuse Syndrome into

“Inconsequential” and “Consequential” Symptoms is defined on the basis of the way in which an individual responds to symptoms. These categories are distinguished by whether or not the symptoms are of any consequence to that individual. (See section 3.10). The study has demonstrated that this response is not only affected by the severity of symptoms but that it is also affected in a non linear fashion by various psychological tendencies.

The means of these various psychological factors for all the diagnostic categories were similar to the standardised normal population of general practice patients and differed significantly from the standardised abnormal populations, Chapter 4, Table 80. Thus, although significant trends were found for three factors, the magnitude of the difference between diagnostic categories, even for the three factors with significant variation between diagnostic categories, could not be taken to indicate the presence of significant trends towards psychological pathology in any category. Nevertheless, it is true that the results did support hypothesis 12, that psychological factors are associated with increased risk of Non-specific Occupational Overuse Syndrome.

5.5. Summary

The effect of the factors which have been identified in the study does not consistently bear a linear relationship to what might be considered the severity of symptoms. Conversely, the determinants of what causes individuals to develop symptoms and to react in certain ways to those symptoms are shown to be a complex combination of a number of physical and psychological factors.

A similar result was found in a study by Helliwell et al who performed a cross-sectional analysis of ergonomic and psychological factors in 63 factory workers in an industrial company in Britain and compared these with the presence of upper-limb symptoms. They also found that the presence of such symptoms could be attributed to a combination of the psychological and physical factors which they assessed. [Helliwell et al, 1992]

The findings are inconsistent with two of the hypotheses which form the basis of certain assumptions implied by advocates of both the theories of psychological and physical pathology. The hypothesis that the musculo-skeletal structures of normal individuals are non-tender was rejected, as was the hypothesis that the musculo-skeletal structures of normal individuals are not painful. The alternative proposition required by the rejection of these hypotheses is that tenderness is a common finding in the musculo-skeletal structures of normal individuals and that the experience of minor degrees of pain in musculo-skeletal structures in the association with activity is a common occurrence in normal individuals.

As a consequence of finding both tenderness and minor degrees of activity-related pain commonly in normal individuals it can be said that none of the associations which have been

found in this study with regard to these factors permit a clear distinction to be made between the diagnostic categories. Indeed in contra distinction to this there appears to be considerable overlap between categories. In other words, there did not appear to be any qualitative difference between either the tenderness elicited in nor the discomfort experienced by normal individuals and individuals qualifying as "patients". Thus, the proposition that there was a pathological change occurring, either physical or psychological, which generated the difference between affected and unaffected individuals, was not supported by the analysis of the data pertaining to these factors.

The finding that the prevalence of symptoms was directly related to the activities with which individuals were engaged and that changes in activity levels seemed to be associated with the onset of symptoms suggested a causal relationship between activity and symptoms. This and the finding that low grip strength was a risk factor for the development of "Consequential Symptoms" in women seems to weight in favour of a physical aetiology rather than a psychological one.

The psychological factors which did show significant variation between diagnostic categories were not the ones which supported either general neurotic tendencies or the tendency toward conversion hysteria as aetiological factors. In addition the range of values for the psychological factors which did show significant variation was such that none of the categories could be regarded as differing from the normal range to a clinically significant extent and the overlap between categories meant that these factors could not be used to predict into which category an individual would be likely to fall, with any degree of certainty.

On the other hand neither the presence of tender sites nor the presence of "Inconsequential" symptoms appeared to increase the risk of developing "Consequential" symptoms. The proposition that these factors were indicative of early stages in the development of a physical pathology was consequently not supported.

Nevertheless, the data has shown a number of highly statistically significant trends across the various diagnostic categories of Non-specific Occupational Overuse Syndrome. The musculo-skeletal tissues of those individuals with apparently more severe symptoms were significantly more sensitive to digital palpation than those individuals with symptoms of lesser degree. Importantly this was most clearly demonstrable in the areas of the upper-limbs which were asymptomatic. This would seem to support the suggestion that there was a physical rather than psychological difference leading to this alteration in sensitivity. Thus, this is evidence against the theory of psychological pathology. Although the finding of increased risk for those with lower grip strength would seem to favour fatigue as a source of symptoms, the presence of persistent significant features occurring at times when physiological muscle fatigue would not actually be present, argues against a direct relationship between fatigue per se and the presence of symptoms. In addition, the presence of an objectifiable physical difference between

diagnostic categories also argues against the no pathology theories as previously constructed. These theories rely on the proposition that the cases of more severe symptoms are the result of exaggerated claims about the severity of symptoms, rather than any real physical difference.

The analysis of the data in this study demonstrated findings pertinent to 11 out of the 12 hypotheses selected for testing. These findings as a whole do not, however, seem to support any of the theories upon which the hypotheses were based. Significant differences were found between diagnostic categories but these differences were quantitative rather than qualitative. The observations do, however, show consistent patterns present across the diagnostic categories, suggestive of a single process as the source of the symptoms studied. If this process does not seem to be attributable to either psychological pathology or physical pathology and the effects of fatigue do not provide an adequate explanation, then an effort must be made to find an alternative explanation. A theory which provides such an alternative explanation is developed in the next chapter.

Chapter 6. Conclusions

6.1. Introducing an alternative theory

The discussion to this point has identified a number of fundamental flaws in the previous attempts to explain the pathophysiology of Non-specific Occupational Overuse Syndrome. The findings of this study, derived from carefully controlled clinical observations, are clearly inconsistent with all previous theories. Consequently, it is necessary to attempt to develop a new theory capable of accommodating and explaining both what was previously known about the syndrome and what has been discovered by the current research project.

Perhaps the most important findings, in this respect, are those which indicate

- that tenderness is widely distributed in “normal”, unaffected populations.
- that a significant proportion of the general population experience “Inconsequential” symptoms which do not disrupt normal activity.
- that the experience of such “Inconsequential” symptoms does not increase the risk of the subsequent development of “Consequential” symptoms. That is, in a large proportion of cases, these “Inconsequential” symptoms continue as part of “normal” routine without increase in severity and may not infrequently resolve spontaneously.

It is these findings in particular which put at issue the assumption, relied on by most of the previous research, that the presence of tenderness and/or symptoms is necessarily indicative of the presence of some pathology. To continue to accept this assumption, it is necessary to classify the majority of the “normal” population as “diseased” (on the basis of tender sites) and to see 37 percent of music students and 10 percent of other students as experiencing a more advanced pathology (they reported “Inconsequential” symptoms). Thus, the validity of this assumption is placed in doubt. To conclude that some activity-based pathology is so widely distributed in the general population is difficult to accept, but clearly, to argue that pathology is not necessarily the cause of tenderness and symptoms, makes it necessary to offer an alternative explanation. This is a key issue which must be resolved before a new, alternative theory is set out which can provide a more comprehensive and consistent explanation of the pathophysiology of Non-specific Occupational Overuse Syndrome.

Just how reasonable is the claim that it is possible to have a source of tenderness and pain which does not have a pathological basis? It is a common clinical observation that where there is disease there is frequently pain and that physical examination for tenderness is often helpful in its assessment. It does not necessarily follow, however, that the presence of pain or discomfort need indicate the presence of pathology, either physical or psychological.

Can the concept of fatigue, as previously proposed in the literature, be utilized as the source of pain in the absence of pathology? As discussed in the previous chapter (Section 5.1.3.), the attempt to equate the pain associated with physiological fatigue and the pain described as occurring in the Non-specific Occupational Overuse Syndrome has major problems. If this physiological process must be rejected as an explanation, could there be some other physiological process occurring which would be able to generate the symptoms observed? One of the principle features of Non-specific Occupational Overuse Syndrome is the observation that controlled non-violent activity when performed for extensive periods on a regular basis, can cause pain in some people and not in others. If there is a physiological process which is the source of the symptoms of Non-specific Occupational Overuse Syndrome, it must have a function directly involved with such activities.

There is an example of a physiological process which generates pain and tenderness in the context of activity which bears close similarities to the problem of Non-specific Occupational Overuse Syndrome. If people who are "unfit" run even a short distance they will experience pain and tenderness in their muscles some time later, this phenomenon has been labelled as "delayed muscle soreness". It has been shown that this phenomenon is not associated with pathological change in the muscles (an injury) but certain physiological changes can be demonstrated. Even with this easily reproduced and recognised phenomenon the details of the actual physiological process involved in generating the experience of discomfort are not understood in detail [Lancet Editorial, 1987b]. Nevertheless, the association of acutely increased unaccustomed activity (acute overuse) with pain and tenderness is an accepted phenomenon which neither causes undue concern for the sufferer nor leads a medical professional to expect to find pathology [Armstrong, 1984a][Owen, 1985b]. Certainly this discomfort will incline affected individuals not to repeat that unaccustomed level of activity immediately but does not induce fear that they will be unable to perform that activity in the future.

Conversely, it has been shown that even well trained athletes who run a marathon do suffer pathological changes in the muscles with demonstrable muscle necrosis [Hikida, 1983][Warhol et al, 1985]. Thus, there is a situation where pain and tenderness will occur as part of a physiological response to minor levels of excessive, acute activity, presumably to induce an individual not to continue and so to reduce the risk of that activity being continued to the point where damage does occur. The level of acute activity which leads to discomfort could be called the tolerance limit for that activity. It is reasonable to expect that this bears some relationship to that level of acute activity which would cause damage. Presumably the tolerance limit is significantly lower than the level at which damage occurs so that the response anticipated from the experience of discomfort when this limit is exceeded, that is, reduction of activity, will prevent damage occurring.

It is well recognised that the discomfort associated with acute activity is frequently delayed by hours to days [Armstrong, 1984a]. A rationale for the characteristics of any physiological process can be deduced from the expected evolutionary advantage that it would provide. A situation which might lead to the need for unaccustomed acute activity is likely to be associated with danger. The immediate experience of pain would interfere with the ability to escape from such a situation. The delayed experience of pain would induce a subsequent period of inactivity, permitting a period of recuperation.

If an appropriate training regime is undertaken then physiological changes will occur so that the level of acute activity which causes discomfort will be raised. Thus, an individual's tolerance to that activity will have increased. If there is a consistent relationship between tolerance limit and the level at which damage occurs then it is reasonable to think that training also raises the level of activity that would be required to cause damage. It can be seen that the level of acute activity which is both comfortable and safe for an individual is not fixed, it can be modified by appropriate intervention such as physical training. Thus similar levels of activity will cause pain in some and not in others.

Careful consideration of the patterns observable from clinical assessment in the course of the study, has led to the development of a new theory to explain the majority of cases which fall within the classification Non-specific Occupational Overuse Syndrome. Non-specific Occupational Overuse Syndrome is associated with regular continuous or repeated activity over a long time span, that is over days, weeks, months or even years. Thus, the context of Non-specific Occupational Overuse Syndrome is that of chronic overuse rather than acute overuse.

The key hypothesis postulated here is that there is a process acting in this context of chronic overuse akin to the process which is more readily observed for acute overuse. This process is not expected to operate via the same mechanism but would have an analogous protective function in terms of controlling activity which might, if continued, cause damage. The rationale for proposing a very different mechanism can be explained by considering the situations leading to an evolutionary advantage for the presence of such a process. Regular repeated activity is not likely to be associated with a dangerous situation but rather with the routine regular activities involved in day to day living. Thus, if there is to be an inbuilt physiological process acting to limit this kind of activity, it could not be the kind of process activated by some sudden unexpected event. It must rather be functioning continuously, in some way monitoring day to day activity so that, if the level of this kind of activity becomes too great, then it will produce the appropriate stimulus to control that activity. The expectation would be to find evidence for this process in a wide range of individuals involved in regular continuous activity of various intensities, not only those who had performed some new unaccustomed activity.

Just such evidence is provided by the findings of the study. For example 56% of asymptomatic individuals have sites of tenderness and 37% of music students and 15% of other

students have “inconsequential symptoms”. It is these findings which provide one of the key supports for the formulation of an alternative theory to explain the origin of symptoms in Non-specific Occupational Overuse Syndrome.

6.2. The alternative theory.

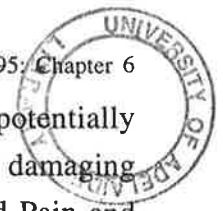
There is a process which occurs in the structures involved in continuous or repeated musculo-skeletal activity of every individual, which acts to vary the sensitivity of some part of those structures proportional to the intensity and duration and inversely proportional to the efficiency of that activity. The level of sensitivity is also modulated by other factors such as the individual's level of physical fitness, physical tension and genetic predisposition. The proposed function of this process is to limit the level of activity when it exceeds that which is appropriate for an individual's current level of fitness. When the sensitivity of the structures reaches a certain threshold, then tenderness can be detected and when it exceeds a higher threshold, then symptoms are experienced. The lowest activity level at which symptoms are experienced is the tolerance limit for that activity. That is to say, an individual's tolerance for an activity has been exceeded at the level of that activity which causes symptoms. This process, thus, becomes functional at activity levels above the tolerance limit, although the fluctuations of sensitivity which it induces within the structures involved can be observed at activity levels below this by the examination for sites of tenderness.

The process functions via the expression of discomfort or pain, with the anticipated effect of limiting activity. As it is proposed that this process functions continuously in all people, that it responds to normal physical activity and that its function is to set a limit to the amount of regular normal activity, it should be considered a normal physiological control process. The term “Physiological Activity Limitation Process” is proposed to describe it and the term “Physiological Activity-related Pain” is proposed to describe the symptom whereby the Physiological Activity Limitation Process exerts its controlling influence. An individual's actual response to Physiological Activity-related Pain will be affected by various psychological factors.

6.3. Justification of the “alternative theory”

How does this theory differ from previous theories which have proposed an alternative to pathological changes as the source for the symptoms of Non-specific Occupational Overuse Syndrome? These “no pathology” theories are represented by three different approaches. The first proposes that there is no real problem present at all, “RSI does not exist”, the second, that the cause is malingering and the third, that the symptoms are due to physiological fatigue. It is obvious that the first two seek to deny the existence of any real physical process, where as this “alternative theory” seeks to explain what appears to be clear evidence that some real physical process is present.

The differences between the proposed Physiological Activity Limitation Process and physiological fatigue is described as follows. The role of the physiological process which generates fatigue-related pain is essentially passive in that it has no function in the absence of true fatigue. In contra-distinction it is proposed that the Physiological Activity Limitation Process is an active process functioning at all times. The function of fatigue-related pain is to induce immediate rest and so allow the fatigued muscles to recover. The function of the



Physiological Activity Limitation Process is to protect the body from all the potentially damaging effects of truly excessive, continuous, controlled activity, not just the damaging effects on muscles. It is suggested that, although Physiological Activity-related Pain and fatigue pain may coexist, they are not directly related. The stimuli which act as input to the Physiological Activity Limitation Process are not limited to the stimuli generated by the fatigue of muscles, although it would see reasonable that these form part of the process.

Although authors advocating the fatigue theory have acknowledged that minor discomfort or “aching” may be “physiological”, they have also implied that when severe pain occurs some pathological process either physical or psychological must have supervened. Valencia wrote about fatigue as a “precursor” to RSI implying that RSI is the pathological result [Valencia, 1986]. Grundy made the proposition, “Let us recognise that strenuous repetitive work can cause physical damage with conditions ranging from tenosynovitis to simple muscular aching” [Grundy, 1987]. Parker and Denzin [Parker and Denzin, 1990a] stated that “Repetition Strain Injury is a musculo-skeletal fatigue syndrome”. “If appropriate attention is paid to the ergonomic, fatigue and postural features and to the restoration of normal muscular balance then the condition is always reversible.” They suggested that, if application of these measures are not effective, then “some secondary gain is present”.

In contrast, this theory proposes that the experiences involved in the Non-specific Occupational Overuse Syndrome, ranging from minor discomfort to severe pain, may all be due to the same physiological process and do not require the development of disease, injury or neurosis for the progression to severe pain. It proposes that it is possible for a physiological process to exist which can induce this full range of sensations.

This aspect of the theory conforms to the concept of “physiological pain” as developed by Woolf [Woolf, 1989]. Woolf developed the concept of “physiological pain” in an article in the British Journal of Anaesthesia. In this article he pointed out that “the same word, *pain* is used to label a wide variety of sensations as soon as they become uncomfortable, unpleasant, irritating, disturbing, severe, intense, distressing, intolerable or disabling.” He proposed that “pain can be divided into two distinct and qualitatively different categories” and suggested the terms “physiological pain” and “pathological pain” to represent these. The distinction that he makes “between the two depends on the argument that physiological pain is a ‘normal’ sensation, while pathological pain is the consequence of an ‘abnormal’ state.”

“Physiological Pain is used to define the range of transient sensations we experience in response to stimuli that are of sufficient intensity to threaten to damage tissue or produce small localized areas of injury”. It “can be elicited by mechanical, thermal or chemical stimuli and clearly defined thresholds can be established at which the sensation stops being one of pressure, hot or cold and becomes painful”. “Physiological pain differs from the sensations produced by innocuous stimuli in being particularly susceptible to interference from psychological factors

such as anxiety or suggestion and by being accompanied both by autonomic and affective responses.” He offered two justifications for using the words “physiological pain” to describe the sensations generated by “non-tissue damaging noxious stimuli”. Firstly the “sensory apparatus involved and the nature of the stimulus-response relationship” are similar to other physiological sensations. Secondly, “from a teleological perspective, physiological pain has a protective role”. He explained that there are two mechanisms whereby this protective function is achieved. “Because of the unpleasant nature of the sensations involved, we learn to avoid certain stimuli and, because of the simultaneous activation of the flexion withdrawal reflex there is an automatic removal of the body from the source of the stimulus.” He points out that sensations which can be classified as “Physiological pain” are “something we all experience frequently in our daily lives”.

“In contrast, inflammatory and neuropathic pain (pathological pain) appear to be the consequences of adaptive and maladaptive disturbances which occur within the somatosensory system. Inflammatory pain retains some teleological resemblance to physiological pain, in that a protective role can be appreciated”. “However, neuropathic pain appears to offer no such benefit and is the pathological product of a disturbed nervous system.”

The primary symptom associated with Non-specific Occupational Overuse Syndrome is that of pain. However, when an individual experiences symptoms associated with intense occupational activity the word used to describe those symptoms will, as often as not, be discomfort rather than pain. This is especially so for those individuals classified as having “Inconsequential” symptoms. From Woolf’s definition it can be seen that the distinction between discomfort and pain in this context is a matter of semantics and depends upon a quantitative rather than qualitative difference. These terms indicate a matter of degree rather than any real functional or anatomical difference. The simple act of lying in bed provides a demonstration of this. A healthy person lying in one position for an extended period will initially experience a minor level of discomfort and will respond by changing position long before the sensation experienced, increases to the level of pain. If, however, an individual consciously chooses to actively ignore an initial experience of mild discomfort, it would be possible to cause the level of discomfort to progressively increase until it reaches the stage of severe pain. Further more, if that individual were able to continue to remain in the one place, either by conscious decision or by inability to move, despite the experience of pain, then eventually tissue damage due to localised ischaemia would occur. Pressure sores caused by lying in one position for too long do occur in patients who can not respond in the normal way to such physiological pain, either because they do not feel it, or because they are unable to move. Thus, it is reasonable to think of discomfort as simply the lower end of a spectrum of sensations, all of which can be classified as physiological pain as defined by Woolf and hence to consider the mechanisms which control discomfort and severe physiological pain as one and the same.

There are a number of important aspects of physiological pain as defined by Woolf, which have a direct bearing on an understanding of the implications of the theory of the Physiological Activity Limitation Process developed in this thesis. If the discomfort or pain which is observed in the context of Non-specific Occupational Overuse Syndrome is to be labelled as physiological pain it should be consistent with the characteristics of physiological pain, as described by Woolf.

Woolf stated that “physiological pain differs from sensations produced by innocuous stimuli in being particularly susceptible to interference from psychological factors such as anxiety or suggestion”. The impact of psychological factors on the phenomenon of Non-specific Occupational Overuse Syndrome has been recognised for a long time. This can be seen from both the development of the psychological theories of causality and the Australian National Occupational Health and Safety Commission’s report on “RSI”. The definition of “RSI” in this report included the statement “Psycho-social factors, including stress in the working environment, may be important in the development of Repetition Strain Injury.” [Australian National Occupational Health and Safety Commission, 1986] Helliwell et al reported an occupational survey in which “Subjects reporting pain were distinguished by higher BSI, anxiety and depression scores.” [Helliwell et al, 1992] The results of this present study have shown clear evidence of significant effects from psychological factors on the expression of the symptoms of Non-specific Occupational Overuse Syndrome (section 4.5.7, Analysis of Psychological data). Of the three factors found in this study, anxiety is a major component of “Affective Disturbance” and suggestion can be seen to influence the factors “General hypochondriasis” and “Psychological versus Somatic Concern”.

Woolf also indicated that physiological pain is likely to be “accompanied by both autonomic and affective responses.” The presence of autonomic responses in cases of Non-specific Occupational Overuse Syndrome were reported by Lowy in 1983 [Lowy, 1983] and have been reiterated many times since. [Graham and Mills, 1984][Gow, 1987][Lockwood and Lindsay, 1989] The presence of affective responses has also been frequently commented on. Stone stated that “The constant pain and inability to work, both at home and in employment, leads to frustration and depression.” [Stone, 1983a] Fry stated of Non-specific Occupational Overuse Syndrome (his term Overuse Injury Syndrome), “There are two major symptoms: pain and mild fluctuating depression.” [Fry, 1986b] McDermott commented that “Depression is common in patients with persistent symptoms.” [McDermott, 1986].

One of the features of physiological pain as described by Woolf, that of a “flexion withdrawal reflex”, is not evident in the phenomenology of Non-specific Occupational Overuse Syndrome. In the normal and healthy operation of the Physiological Activity Limitation Process, however, the generation of such a response would not be expected. The circumstance in which this process occurs is that of a safe and routine situation of normal activity. In such a circumstance an instant unconscious movement would not be likely to be beneficial or “protective”. On the

other hand, the characteristics of the unpleasant sensations, which it is proposed be called “Physiological Activity-related Pain” are not inappropriate for this circumstance. They are not sudden or sharp sensations but usually come on gradually at low severity, building up in intensity over time. There are other situations in which physiological pain occurs which also lack both a potential role and any detectable presence of this unconscious withdrawal response. These include the example of fatigue pain described by Parker and Denzin [Parker and Denzin, 1990a] and the example of pain induced by lying in one position for too long, described above. Further implications of the flexion withdrawal reflex are discussed in a later section (section 6.5.1.5.5.) when the possibility of dysfunction of the Physiological Activity Limitation Process is considered.

Thus the “alternative theory”, that Non-specific Occupational Overuse Syndrome could in large part be seen to be due to a Physiological Activity Limitation Process leading to Physiological Activity-related Pain, is consistent with the features of physiological pain as proposed by Woolf.

Another relevant characteristic implicit in the physiological pain concept described by Woolf, is the inherent implication of “threat of tissue damage” in the context of “non-tissue damaging noxious stimuli” and its “protective role”. Both the concepts of protection and threat of damage imply the potential for damage if the cause of the stimulus which generated the physiological pain is increased or continued to a significant degree. These concepts imply the existence of a second threshold which is higher than that described by Woolf for the experience of physiological pain. This second threshold is at the level of stimulus sufficient to cause significant tissue damage and consequently to transform the experience of physiological pain to that of pathological pain. Thus the concept of Physiological Activity-related Pain implies that there is a threshold level of ongoing normal activity, at which damage to the structures involved can occur. This threshold must be significantly greater than the level of activity at which Physiological Activity-related Pain is first experienced (the tolerance limit for that activity). This can be compared with the two equivalent pain thresholds for the application of heat. The lower threshold is sufficient to generate the experience of “physiological pain” but insufficient to cause tissue damage (for example placing a hand into very hot but not boiling water for a few seconds) and the higher threshold is sufficient to cause a burn and consequently generate the experience of “pathological pain” (for example placing a hand into boiling water).

Is there any evidence that excessive "normal" activity can cause demonstrable tissue damage?

Some authors have claimed that normal occupational activity can not cause pathological changes. “The fact is that it is physiologically impossible to cause permanent injury to striated muscle, either isometrically or dynamically” [Awerbuch, 1984]. “The issue we are considering is whether any particular musculoskeletal usage which in and of itself is reasonable, comfortable, and customary and which can be repeated without undue distress can lead to

specific musculoskeletal damage over time" [Hadler, 1990]. Other authors have put forward the opposite view. "There is ample evidence that repetitive activities can be combined with abnormal strain to produce physical abnormalities" [Grundy, 1987]. "We find considerable support from clinical reports and laboratory and epidemiologic studies that highly repetitive and high hand force activities are causally related to both pain and specific clinical syndromes of the forearm, wrist and hands" [Silverstein & Fine, 1991]. A number of authors claim to have demonstrated an association between excessive regular activity and certain specific diagnostic entities. These include objectively proven tenosynovitis [Welch, 1972] and carpal tunnel syndrome [Masear et al, 1986][Barnhart et al, 1991][Armstrong et al, 1984b].

The condition associated with excessive activity over an extended period which is most analogous to the muscle damage caused by excessive activity in acute overuse i.e. marathon running, is however, peritendinitis crepitans. Although some authors have equated peritendinitis crepitans with RSI and by implication Non-specific Occupational Overuse Syndrome [Fry, 1986b], it would seem that to do so is to fail to take into account recognised features of each allowing clear differentiation.

Peritendinitis crepitans was first labelled as such by Troell in 1918 [Troell, 1918] although descriptions of cases with similar features had been made previously [Hunter, 1975]. The particular objective feature of this condition, which demonstrates the presence of local pathology, is the presence of palpable crepitation over the muscles affected and absence of clinical signs directly relating to the tendons and tendon sheaths, thus, differentiating it from tendonitis and tenosynovitis.

Howard [Howard, 1937], in 1937 reconfirmed this work and described the condition as "A Muscle-Effort Syndrome". He described a series of 32 cases and felt that there was a causal relationship of excessive and/or unusual activity with all cases. Symptoms occurred "after a long lay-off from work or shortly after a change from habitual work to an unfamiliar task requiring new and rapid movements. Continuation of the same work gave rise to severe pain". This process occurred over a period of days or weeks. He performed biopsy studies on three cases and found obvious pathological changes in the muscle and adjacent tissue. He concluded that "Peritendinitis crepitans is the result of exhaustion of particular muscle groups by unaccustomed and unremitting toil".

Thompson et al presented equivalent results in a series of 419 cases of peritendinitis crepitans. These were part of a series of 544 cases of occupationally related upper-limb pain and these cases were noted by them to be clinically distinct from tenosynovitis (125 cases) [Thompson, 1951]. He also presented 5 cases in which tissue biopsies had been taken providing histologic confirmation of pathological changes. He identified five main aetiological factors. 1. Some occupational change necessitating unaccustomed work (144 cases). 2. return to work after absence (114 cases). 3. local strain either repetitive or single (79 cases).

4. direct trauma (76 cases) . 5. simple repetitive stereotyped movements associated with intensity of effort and speed (53).

The demonstration of pathological changes in muscle resulting from excessive occupational activity in this condition is clear evidence that it is possible to exceed the tolerance of the structures involved to the point of structural damage. This also makes peritendinitis crepitans fall within the classification of Specific Occupational Overuse Syndrome rather than Non-specific Occupational Overuse Syndrome as defined in this study.

Although this “alternative theory” has been developed on the basis of an analogy with acute overuse, it appears likely that the processes occurring with acute overuse and the hypothesised Physiological Activity Limitation Process are, most probably, fundamentally different.

There are certain fundamental features of this hypothesis which point to the possible sites of action for this process. The hypothesis proposes a physiological process which modulates the sensitivity of the structures involved in activity, in other words, which causes changes in the level of stimulation required to induce an experience of discomfort or pain. The experience of physiological pain involves the stimulation of an adequate number of appropriate peripheral sensory nerves which in turn stimulate neural centres in the central nervous system and these, through a complex system of neural processing, eventually impinge on the sensory cerebral cortex. This leads to a conscious experience of pain or discomfort, depending on the intensity of the stimulus and the interpretation by the individual. Potentially all levels involved in the control, sensation and activation of activity, are implicated. Changes in the structures involved in activity, such as changes in the oxygen tension within muscles, could change the stimulation of sensory endings within those muscles, changes within the sensory endings themselves could mean that their threshold for stimulation is altered and changes within the central nervous system could alter the peripheral level of input required to lead to propagation of that stimulus to the sensory cortex. Thus, it can be seen that the hypothesised Physiological Activity Limitation Process could be operating at a wide range of possible anatomical sites. Although this study has proposed that this physiological process is an explanation for the majority of cases of Non-specific Occupational Overuse Syndrome it can not provide specific details of its nature. This must be left for subsequent research to investigate. If the hypothesis presented in this thesis is to gain widespread support, then there will be need for such research to identify and characterise the nature of this physiological process in a normal, clearly non-pathological context.

6.4. Implications of this alternative theory.

Corollaries to the findings of this study and the resultant theory developed are as follows.

1. A significant proportion of individuals who present to a medical practitioner with symptoms consistent with Occupational Overuse Syndrome, if appropriately assessed, will be found to have specific identifiable physical pathology. The present study found 48% of the individuals presenting with Occupational Overuse Syndrome to have specific diagnoses. The findings of Sikorski [Sikorski, 1988] parallel these. He found that 58% of the patients presenting to his "RSI" clinic were eventually found to have a specific diagnosis. This fact can not be over emphasised. The medical community's failure to understand the nature of Non-specific Occupational Overuse Syndrome has led not only to failure to develop an effective consistent plan of management but also to the failure to differentiate and discriminate between Non-specific Occupational Overuse Syndrome and Specific Occupational Overuse Syndrome. It is perhaps the latter which is the most unfortunate for this must on occasion lead to delay in provision of appropriate treatment.

2. In the majority of cases with no identifiable physical pathology (those fulfilling the criteria for Non-specific Occupational Overuse Syndrome) the symptoms can be explained as due to a physical process which is directly related to the activity with which it is associated. This physical process is best described as a Physiological Activity Limitation Process. It is proposed that the symptoms generated by this process can best be labelled Physiological Activity-related Pain and as such can not be taken to imply the presence of an injury. The reason that no pathology can be detected in this situation is that no pathology is present.

3. It is likely that a wide range of factors influence the Physiological Activity Limitation Process and so act to modify Physiological Activity-related Pain. This study has identified a number of these but further research is warranted. Evidence that aspects of physical fitness are important modifiers has been shown. Evidence has also been found to show that psychological factors act to modify both the experience of pain and an individual's response to it, both directly and indirectly.

Given that the Physiological Activity Limitation Process functions in all people with varying degrees of expression then it is to be expected that symptoms attributable to it, Physiological Activity-related Pain, and symptoms caused by some completely independent process, pathological or otherwise, may coexist. In fact it would seem likely that they would interact. A localised pathology causing pain might be expected to cause increased tension, a compensatory change in posture and/or technique. Such changes would be expected to directly influence the Physiological Activity Limitation Process and so lead to increased levels of pain in associated areas. Assessment of any form of activity-related pain should take this likely combination of symptoms into account. Identification of a site of localised pathology does not rule out

Physiological Activity-related Pain as a contributory source of symptoms and conversely the presence of features consistent with Physiological Activity-related Pain does not rule out the additional presence of some form of Specific Occupational Overuse Syndrome. It may well be that this is one of the factors which makes assessment of upper-limb pain difficult and confusing.

4. Previously, the medico-legal debate centred around whether or not occupational activity was the cause of the disease “Non-specific Occupational Overuse Syndrome”. This alternative theory argues that symptoms of Non-specific Occupational Overuse Syndrome can be seen, in the majority of cases, to be due to a physical process directly affected by the occupational activities in question but rarely imply the presence of a disease or injury. What this means as far as compensation is concerned must be left to the courts to decide.

6.5. Consequences of the acceptance of this “alternative theory”

If this theory is confirmed by further research and so becomes generally accepted, then there would be a number of benefits to be gained for the community as well as certain consequences which may not be seen as favourable by some groups of people.

6.5.1. Explanation of significant observed phenomena

There are certain observed phenomena occurring within the framework of Non-specific Occupational Overuse Syndrome which have been difficult to understand on the basis of previous theories. This theory enables a number of these phenomena to be explained.

6.5.1.1. The success of certain empirical measures

Initial attempts to develop management programs were based largely on empirical observations. McPhee presented a systematic approach to both prevention and management at the 1983 Australian Physiotherapy Association Conference in Adelaide, South Australia [McPhee, 1983]. She based her approach on an analysis of the previous literature and her own observations. She expounded the theory that there were 6 factors which “would appear to be the direct causes of repetition injuries.”

- The number of movements
- The load or force required
- The amount of static muscle work used
- Stressful work postures of the body generally and / or particular joints
- The individual’s physical capacity
- Time

She suggested “that increased risk of strain in each individual occurs when:”

- new demands are placed on the individual
- he or she habitually works beyond his or her capacity
- personal, social or environmental factors reduce the individual’s tolerance to physical stress.

In addition she suggested that “much more attention needed to be paid to the ergonomic design of work. The ergonomic approach to work analysis consists of three elements:”

- Task design
- Workplace design
- Work organisation factors.

Ferguson summarizes the conventional approach to management which evolved out of these concepts. “Appropriate activity, the relief of anxiety and an attention to ergonomics are the preferred management, although ultimately the best management lies in prevention.” [Ferguson, 1987]

On the basis of such principles and in the absence of any convincing evidence to the contrary most large employers undertook to introduce ergonomic improvements in their work places with the aim of prevention. Management programs for individual patients sought to address these issues. On the basis that the pathological changes inferred by the presence of pain would benefit from physical treatment, such modalities as physiotherapy, manipulation, splintage, analgesics/anti-inflammatory medication, acupuncture and surgery were often utilized. The effectiveness of such programs has been reported in the literature and seems to have had varying success.

Wilson reported a series of 4 patients who “presented with pain at rest in the wrists and forearms as a result of keyboard work.” He made a particularly salient point in describing the intellectual response of his patients. “Their initial reaction was bewilderment that such a thing could happen, then severe anxiety as a result of the widespread publicity given to RSI.” He provided a management program on the standard model and was able to report that “All cases settled within a few weeks on a programme of rest, support, and investigation of work (and sometimes home) conditions.” [Wilson, 1985]

Fry described the results of treatment of 379 musicians [Fry, 1986b]. A number of these had received previous treatment. He reported lack of benefit from such treatment modalities as “anti-inflammatory drugs and muscle relaxant agents”. Twenty patients had previously received surgery without improvement. After classification according to his arbitrary system of grading (listed in chapter 2, section 2.4.3.), he allocated the patients to two different treatment regimes, also in a somewhat arbitrary fashion. The proportion in each grade was: Grade 1, 29.6%; Grade 2, 28.8%; Grade 3, 21.9%; Grade 4, 13.5%; Grade 5, 6.3%. “Grade 1, Grade 2 and some Grade 3 injuries were usually treated by modifications of technique, practice habits,

posture, or teaching of body awareness and control.” “Patients with Grade 5 , Grade 4, most Grade 3 and occasionally Grade 2 were treated with a radical rest programme”. This programme proscribed “Music making, writing, driving, housework, and any other use of the hand which could cause pain”. Although he did not state the outcome from the conventional treatment programme, the implication was that this was successful or else the “radical rest programme” was applied. It is consequently unclear from his article exactly what proportion received the conventional treatment and what proportion undertook the “radical rest programme”. It would seem to be the case that at least 50% recovered with use of the conventional approach.

Wright [Wright, 1986] developed a management programme in which the factors of “ergonomics, work organisation and social aspects of work” were addressed. Although he took an unorthodox stance with respect to the issue of causality, avoiding both the “notions of injury and hysteria”, the actual approach to management can be seen to conform to the conventional model as described by McPhee. The cause of symptoms utilised for descriptive purposes in his management programme was that of a “muscle-based pain cycle”. It is unclear what this description was meant to imply in terms of pathology but was clearly designed to avoid controversy. The denial of both injury and hysteria would suggest that the middle ground of fatigue-generated pain might have been the implied pathology. Whatever the assumed pathology, this management programme appeared to be effective. This programme was applied to a complete population of keyboard operators in one university, 160 individuals, of which 13 cases of Non-specific Occupational Overuse Syndrome were dealt with. “All 13 returned to work”.

In 1987 a Lancet Editorial commented “Despite their essentially empirical nature, the present approaches to treatment are largely effective” [Lancet Editorial, 1987a].

Sikorski sought volunteer patients with symptoms consistent with Occupational Overuse Syndrome in whom no single specific diagnosis had previously been made [Sikorski, 1989]. A series of 204 individuals was collected. By thorough assessment a specific diagnosis was able to be established in 58% of these. Sikorski described previous treatments. “A wide variety of specific treatment modalities had been previously tried on these people, with none showing a high rate of success.” These included the full range of possibilities i.e. physiotherapy, manipulation, splintage, analgesics/anti-inflammatory medication, acupuncture and surgery. The sampling method used in this study, however, was such that those who had failed to find benefit from previous medical attention would have been likely to volunteer. Recognition that a small proportion of cases fail to respond to conventional treatment has been widely acknowledged.

Bouvier [Bouvier, 1992a] described a management programme devised in the late 1960s. Despite the fact that no specific psychological or psychiatric treatment modalities were involved,

he claimed that he was treating “the workers as one treats conversion hysteria”. In explaining the symptoms he told patients that the pain “was just sore muscles from unaccustomed exercise, reassured them that it was not serious” (not an injury). He “never told them about hysteria”. The management programme consisted of helping “them sort out their occupational, personal, social, family, and financial problems.” Despite a different premise with regard to pathology this programme appears to be almost identical to that of Wright mentioned above. He described the effectiveness of this programme as follows. “From having 20 out of 220 employees off work long term with ‘tenosynovitis’ in 1969, we got to over 300,000 hours without any lost-time injuries by 1972.”

It is interesting to note that management programmes, developed on the basis of differing pathological premises, have marked similarities. The 1986 Australian National Occupational Health and Safety Commission’s report on RSI (section 2.12) comments “It is fortunate that whatever model of causation is adopted, the recommended method of prevention and management is the same.” The reason for this was not clarified. A possible explanation, however, is that the management programmes were all based on empirical evidence and the pathological models with which they had been associated, none of which had been verified, were superimposed.

This “alternative theory” offers a rational explanation as to why the empirically based conventional approach, which deals with the issues of excessive activity and associated psychological stresses, should have been effective in the management of Non-specific Occupational Overuse Syndrome in the workplace.

Both the conventional physical pathology theory and the “alternative theory” proposed in this thesis contend that excessive activity is the cause of symptoms and that excessive activity may lead to physical harm. Empirical observations have shown that by controlling and modifying activity, symptoms can be reduced. Indeed, this is one of the observations upon which both of these theories are based. Whereas the conventional theory takes the presence of symptoms to indicate that pathological effects have already occurred, the “alternative theory” proposes that long before physical harm occurs a healthy physiological process will intervene by inducing the symptom of discomfort. This will limit activity and so prevent the excessive activity being continued to the point where harm does occur.

As the cause of symptoms remains the same, so the fundamentals of prevention and treatment remain the same. It is obvious that there is a major difference between the theories in relation to the implications associated with the development of symptoms. The conventional theory proposes that when symptoms are experienced physical damage has already occurred whereas the “alternative theory” proposes that actual pathological changes would only occur if it were possible to ignore the symptoms and continue with the excessive activity for a sufficient period.

It is this difference which indicates the need for alterations in the management programme, as suggested by the “alternative theory”.

There would be no difference in the preventative strategies. A treatment programme based on the “alternative theory” would, however, call for prompt rather than immediate intervention. Carefully graded changes in intensity of activity, introduced over time, would be considered appropriate rather than immediate extreme changes such as complete rest. Changes in other factors, such as work practices, technical skills, improvements in the ergonomics of the tasks involved and measures to deal with psychological factors, would be undertaken as appropriate in due course. The conventional theory assumes that pathology is present and as such would suggest that specific modalities such as physiotherapy, splintage, anti-inflammatory medication and surgery might be beneficial. The “alternative theory” proposes that no pathology is present and this would explain the previous observation that the application of such physical modalities is usually not helpful.

The previous conventional theory is based on the assumption that there should be no discomfort associated with work activities and that when discomfort or pain develops, an injury or disease has occurred. If the “alternative theory” were widely accepted, then workers engaged in extensive continuous activity would expect the occasional development of minor levels of discomfort and would understand that this discomfort was not an injury but an indicator that the body was acting to prevent an injury occurring. If appropriate intervention were untaken then symptoms would resolve and no harm would be expected. Thus, the acceptance of the “alternative theory” would lead to a major change in the expectations associated with, and understanding of, the occurrence of symptoms.

6.5.1.2. Epidemics

Non-specific Occupational Overuse Syndrome has been seen to occur in the form of epidemics in different countries at different times [Welch, 1972][Hocking, 1987a]. It was the occurrence of this phenomenon in Australia during the early 1980's which created the intense interest and speculation regarding this problem and indeed which prompted the undertaking of this study.

If the hypothesised Physiological Activity Limitation Process is to be accepted as the source of the symptoms which were observed in epidemic proportions then its proposed features must be consistent with the observations made concerning this epidemic.

Early in the progress of the epidemic the observation was made that users of computer keyboards appeared to be suffering with an increased incidence of arm pain. This also occurred at a time when the use of such keyboards was becoming increasingly wide-spread. Bell observed that “Historically, the use of equipment which requires the use of repetitive movements and the introduction of innovations have provided the focus for the generation of

craft neuroses. The 'RSI' epidemic commenced among those who made highly repetitive movements in data processing" [Bell, 1989a].

In the absence of an alternative, accepted medical explanation for these observations and with precedents from research in other countries the quite reasonable conclusion was reached that there was something inherently dangerous about the new equipment and that serious injuries were being caused by its use. Previously, in Japan, the interpretation was made that occupational related upper-limb pain indicated the presence of a disease. In a review article in 1977 Maeda stated that this "occupational disease ... has come to be frequently noticed among workers of the offices and factories in Japan since 1955. The disease was at first seen among punch-card perforators who appeared with the progress of technical innovation" [Maeda, 1977]. These observations and the common interpretations of them were "grist for the mill" where the mass media was concerned. Headlines such as "Risk in the keyboard" [Smith, 1981, National Times] and "Technology dumped here, says expert" [McIntosh, 1984, The Age] are representative of the media response. This led to a rapid increase in public awareness of the condition which was initially labelled tenosynovitis and later RSI.

It is generally accepted and has been well documented that the introduction of new technology involving hand-use intensive activity is associated with an increased incidence of upper-limb pain. The characteristics proposed for the hypothesised Physiological Activity Limitation Process provides a coherent explanation as to how the introduction of a new form of technology, such as computer keyboards in the Australian "epidemic" of the 1980's, could trigger a real rise in the incidence of occupationally related arm pain.

The theory of the Physiological Activity Limitation Process states that sensitivity levels of the structures involved in daily activities are directly affected by an individual's activity but the perception of those sensitivity levels only occurs when a threshold value is reached. The unpleasant sensations generated when that threshold value is reached is called Physiological Activity-related Pain. The tolerance limit for an activity is defined as the level of activity which will raise the sensitivity of the tissues to the threshold at which Physiological Activity-related Pain occurs. At any one time an individual's tolerance limit is determined by his/her current level of "fitness" for that activity. Fitness is determined by a number of factors, several of which may be modified. All ranges of activity below the tolerance limit cause no symptoms. Ranges of activity above the tolerance limit induce proportionate increases in sensitivity above the threshold value with a related increase in severity of symptoms.

In a population of individuals engaged in some occupational activity such as typing there will be a certain distribution in the values of the sensitivity levels of the individuals involved. Given a normal stable occupational setting the prevalence of individuals with a sensitivity level above the threshold value would be expected to be low (that is the normal endemic rate for occupationally related discomfort for that occupational group would be low). It remains possible that the

distribution of the values for sensitivity could be such that the sensitivity level of a large proportion of the individuals could be just below the threshold and still only very few individuals would experience any occupationally related discomfort. A situation in which this is likely to occur is one in which the employees have become accustomed to a high level of productivity over time.

The introduction of new technology, such as changing from typewriter to computer keyboard, could have a large effect on the incidence of activity related arm pain as follows.

The muscle actions and forces involved in typing on a typewriter and typing on a computer keyboard are not identical [McPhee, 1985] although the final result of the activity (printed documents) may be the same. As physical fitness is task specific, the fitness of the individuals for the two tasks would not be the same. Individuals undertaking a new task will have a lower fitness for that task than their fitness for the tasks to which they have grown accustomed. Expressed in the terms of the "alternative theory" the activity tolerance limit for the new task will be lower than that which was previously achieved for the old task, even though those tasks appear similar. An individual achieving the same work output but using new equipment will develop higher levels of sensitivity. Whether or not this goes above the threshold level for the experience of Physiological Activity-related Pain will depend on the sensitivity level which was being maintained using the old equipment and the differences in the activities involved in using the new.

If the new technology is introduced to a group of people, many of whom have a sensitivity level near to the threshold level, even if only a small average increase in sensitivity levels occurs, then the sensitivity level of a large proportion of that group could change from a level below the threshold to a sensitivity level over the threshold. This would mean that even with a small effect on sensitivity the introduction of a new technology could cause the transition from a low to a high incidence of Physiological Activity-related Pain in such a group.

Thus, although the tasks involved in the use of the typewriter and the computer keyboard appear to be very similar and the differences of the muscle actions and forces involved are not obvious, these differences could, in the right circumstances, cause a marked increase in the incidence of Physiological Activity-related Pain.

The "alternative theory" developed in this thesis can provide an explanation for the source of a real increase in incidence of Non-specific Occupational Overuse Syndrome which appears to have occurred at the beginning of the Australian epidemic. Although this effect in no way explains the magnitude of the Australian epidemic, the reporting of this increase may well have been one of the major initiating factors leading to increased public awareness.

The fact that public awareness increased dramatically in parallel with the development of the epidemic is another accepted observation. Considerable research has been focused on the complex sociological processes which led to the continued promotion of "RSI" as an important occupational hazard. [Willis, 1986][Hopkins, 1990] A number of authors have proposed that it was the promotion of the public awareness of the condition which was a major factor in the continued increase in incidence of the problem and consequently to the magnitude of the Australian epidemic.

The situation in which the observation of an increase in arm pain associated with new technology was followed by a similar increase in other unrelated occupations where no new technology was involved, was previously reported in Japan by Maeda. "The disease was at first seen among punch-card perforators who appeared with the progress of technical innovation in business work. But several years later, it was revealed that the disease affected not only those perforators but also keyboard operators of other machines, typists, copying-slips writers, conveyor-line workers, and others" [Maeda, 1977]

A number of different mechanisms have been proposed as to how promotion of public awareness could lead to increases in the incidence of the condition. Lucire proposed "mass hysteria" [Lucire, 1986]. Cleland proposed "RSI as a model of social iatrogenesis" [Cleland, 1987]. Bell claimed that "The misplaced emphasis on the early recognition of the disorder promoted the spread of 'RSI'". [Bell, 1989a] Ferguson proposed that the majority of the cases reported in the epidemic were the result of unmasking of endemic occupational discomfort which, had it not been for the increased public awareness, would have gone unreported [Ferguson, 1987].

The concepts of Lucire, Cleland, Bell and Ferguson have marked similarities and point to psychological and social mechanisms which could lead to the amplification of the sensations which commonly occur during occupational activity. Although all four provide similar compatible explanations as to how psychological and social factors could amplify the experience of common place occupational discomfort they do not agree as to the nature of the original sensations that were being amplified.

Lucire contended that the epidemic of RSI could be explained by psycho-social phenomena due to epidemic hysteria on the part of patients and the result of altered medical perception of endemic symptoms within the community. The principle espoused is that psycho-social forces led to an exaggerated response to symptoms which were endemic in the community. Lucire's fundamental theory was that the origin of the endemic symptoms was that of conversion neurosis. As she proposed that the original symptoms were hysterical in nature she labelled the psychosocial amplification of these symptoms "mass hysteria". As indicated above, conversion neurosis is not a satisfactory explanation for the endemic symptoms.

Cleland suggested that the “pain may be due to a disturbance of sensory function”, a similar concept to the pathology of fibromyalgia as postulated by Smythe [Smythe, 1979]. Cleland provided an excellent explanation as to how psychosocial processes could alter the personal interpretation of occupationally related discomfort so that symptoms which would previously have been ignored are reinterpreted as serious and therefore lead affected individuals to seek medical help. Consequently, this would lead to an increase in reported incidence. The following is a quotation of his complete argument.

“Educational programs that impute the seriousness of upper limb discomfort and the potentially noxious effects of manual tasks will prey on the minds of individuals who perceive themselves to be at risk. Worry and introspection will follow. Discomfort, even when appropriate for activities that have been undertaken, will cause concern, and may thus be amplified, and cause further concern which, in turn, further heightens regional sensory awareness thereby completing a vicious cycle. Therapists who diagnose injury, who apply local therapies and prescribe radical avoidance of use, will further focus the victim’s attention on the uncomfortable part. The result is that no task can be performed without enhanced attention to discomfort and apprehensive concern as to the potentially damaging effects that are associated with it. Through this process, otherwise trivial discomfort may become transformed into a protracted, painful, disabling condition which precludes effective work and degrades the quality of life.” [Cleland, 1987]

Cleland emphasised his belief that symptoms associated with normal repetitive occupational activity are normally only of “trivial” degree. He explained how the experience of such symptoms could be amplified in the minds of affected individuals. This would lead to both an increase in the severity of the pain experience in such individuals as well as an apparent increase in the reported incidence of cases of occupationally related arm pain. The apparent increased incidence occurs because more people are induced to seek help. His explanation did not, however, suggest that psychosocial processes create new symptoms but only that they amplify pre-existing symptoms which would otherwise remain of minor degree. The source and nature of these pre-existing trivial symptoms was not explained.

Regarding the psychosocial factors involved in the promotion of the Australian epidemic Bell stated “The measures that were introduced to manage the disorder (Non-specific Occupational Overuse Syndrome) were disastrously inappropriate and instead promoted its spread. The belief that the condition was an injury reinforced the hypochondriacal concern of the complainants and facilitated unjustified claims for compensation. The misplaced emphasis on the early recognition of the disorder promoted the spread of ‘RSI’. The suggestive elements in these messages flourished in an atmosphere of mistrust and resentment which was fostered by some trade unions and pressure groups.” With reference to the pathophysiology he claimed that “The psychological basis of ‘repetition strain injury’ (RSI) is revealed by its rapid rise and fall.” Once again reasonable explanations were provided as to how psychosocial factors can promote awareness and severity of the condition. The claim that epidemiological observations of increasing and decreasing incidence over a decade proves a psychological pathology, is not convincingly argued.

In his article written in 1987 Ferguson indicated that a major component of the Australian epidemic may have been an artefact due to psychosocial processes. "This epidemic has represented a remarkable psychosocial expansion of a long-recognised and continuing occupational, medical and work-design matter. It was brought about by a complex concatenation of several streams of social development in work health and safety which include legislation, industrial relations, worker-health movements and unemployment, among others."

Ferguson stressed the fact that the epidemic occurred in the context of a significant "endemic base". "Aching and discomfort are commonplace in life, whether or not they are attributable to work, and may be elicited in any workforce by suitable enquiry." [Ferguson, 1987] He described his epidemiological study of the incidence of unreported occupational arm pain in the Australian telecommunications industry [Ferguson, 1971a] in which "about 40% of telephonists in Sydney upon enquiry complained of unreported work-related back pain, and about 20% complained of neck pain. Furthermore, 22% of telegraphists in Sydney were judged to have craft palsy and related pain, although again this had gone unreported." He suggested that the importance of the endemic problem of Non-specific Occupational Overuse Syndrome remains. "Although evident in the late 1970's, the epidemic proper arose in the early 1980's, peaked in the mid 1980s and is now declining rapidly ." "The epidemic, to which the medical and legal professions, managements ,unions, governments and the media have all contributed, is now waning, but endemic work-related musculoskeletal syndromes remain." His discussion leaves open the question as to whether or not high incidence of existing endemic morbidity, represented cases of unrecognised disease.

Further discussion of the psychosocial circumstances of the Australian epidemic is beyond the scope of this thesis, however, an exposition follows of the way in which the Physiological Activity Limitation Process provides a possible explanation for the endemic, occupational discomfort which is widely recognised.

The results of the research reported in this thesis confirm Ferguson's finding that unreported minor occupational arm pain is commonplace. This study found that 37% of first year Instrumental Music Students and 15% of the first year students from other courses experienced minor levels of pain associated with activity (subjects classified as having "Inconsequential symptoms"). The "alterative theory" of this thesis provides an explanation for this and in so doing explains the origin of the symptoms which were amplified by the psychosocial circumstances of the Australian epidemic.

The "alternative theory" developed in this thesis states that the Physiological Activity Limitation Process is universal in scope. It is operating in everyone, continually adjusting the sensitivity of the structures involved in activity in a manner appropriate for the individual's current level of fitness and activity. This process acts, in appropriate circumstances, to protect from the potentially damaging effects of excessive activity by raising the sensitivity level to the threshold

where Physiological Activity-related Pain is experienced. The theory proposes that this is a healthy physiological protective mechanism. Just as one should expect to feel pain when one touches a pin or a hot cup or goes for an unaccustomed run so should the experience of discomfort be expected if new or increased occupational activity is undertaken.

Any physically active population engaged in continuous repetitive activities would consist of individuals with varying physical characteristics, physical fitness, psychological tendencies and occupational demands. It would be expected that the Physiological Activity Limitation Process would induce degrees of Physiological Activity-related Pain in a proportion of the individuals within such a population.

A clearer understanding of this proposition can be reached if the diagnostic classification of Occupational Overuse Syndrome developed in chapter 3 , section 3.10, is reconsidered in the light of the subsequent "alternative theory". This diagnostic classification divides the cases of Non-specific Occupational Overuse Syndrome into those in which symptoms are "Inconsequential" (of no consequence) and those in which symptoms are "Consequential" (of some consequence). It has been shown that this division depends on a complex combination of the intensity of the symptoms and the individual's psychological response to those symptoms. The "alternative theory" proposes that in the majority of cases such symptoms are "Physiological Activity-related Pain" of varying intensity. The subdivision of Non-specific Occupational Overuse Syndrome into cases with "Inconsequential symptoms" and "Consequential symptoms" can be seen to be a classification of the impact of Physiological Activity-related Pain on the individual. By definition, cases with "Inconsequential symptoms" will go unreported except in epidemiological surveys, they will not form part of the statistics reporting the number of cases seeking treatment. It is only cases with "Consequential symptoms" that are included in the "occupational health statistics" which are taken to indicate the prevalence of the problem within the community, the cases which would be taken to indicate the magnitude of the "epidemic".

In the population of "Music Students" surveyed in this study the prevalence of "Inconsequential symptoms" was 37% and the prevalence of "Consequential symptoms" was 6%. Other surveys have demonstrated prevalence of "Inconsequential symptoms" of similar magnitude in a variety of occupational settings. It can be seen that if some psychosocial process were to cause the majority of those with "Inconsequential symptoms" to feel the need to seek treatment and thus cause their diagnostic classification to change from "Inconsequential" to "Consequential" then an "epidemic" would become apparent. The psychosocial pressures outlined by the authors above can thus be seen to be acting by changing the interpretation of symptoms which are already being experienced. It is not necessary that the psychosocial pressures actually generate symptoms which would not otherwise be present. This is to say that it is not necessary to invoke psychological pathology either individually or en-mass to explain the presence of symptoms.

The suggestion that the majority of individuals seeking treatment during “epidemics” have what would have been classified as “Inconsequential” symptoms at other times does not imply that all such people can be dismissed as not having a significant problem. It does imply, however, that in the context of such an epidemic, a large proportion of cases should be able to be managed by simple reassurance and minor modifications of activity. The remainder would still be those in which the intensity of Physiological Activity-related Pain has reached a level at which function has been compromised and in whom management would be expected to be less straightforward.

The “alternative theory”, which indicates the source of symptoms in the majority of cases of Non-specific Occupational Overuse Syndrome, can explain the high prevalence of the unreported cases of endemic occupational discomfort seen in epidemiological surveys and the cause of certain increases in the incidence of discomfort associated with introduction of new technologies. There must, however, be a set of appropriate social beliefs and processes in place for such phenomena to cause the prevalence of treated cases to be amplified to the magnitude of the Australian epidemic.

A requirement for this amplification to take place must be the propagation of the belief, both among the medical profession as well as the general public, that all forms of occupationally related arm pain are indicative of injury or disease. The failure of medical research to define an accepted pathophysiology for Non-specific Occupational Overuse Syndrome has allowed a state of epistemological indeterminacy to persist (previously discussed in section 2.8). Without an established medical paradigm in which occupational discomfort can be explained in the absence of pathology, this belief is likely to be “rediscovered” and propagated at times of technological innovation. The acceptance of the “alternative theory” developed in this thesis, with the concept of the Physiological Activity Limitation Process, would create a paradigm in which this belief would not be generated.

It can also provide a rationale for the proposition that the initial management of minor degrees of occupationally related discomfort should be undertaken in the workplace rather than through medical intervention. Without the addition of appropriate measures to prevent it, an increase in levels of discomfort should be expected when new equipment is introduced. The theory would suggest that appropriate preventive measures should include both a phased introduction of new equipment and systematic training dealing with all aspects of the workers’ interface with their equipment. This would include ergonomics, technique and physical fitness as well as careful, thorough assessment of anyone reporting significant symptoms and reassurance to those who have simply worked too hard too quickly. A period of decreased productivity should be expected and planned for, as employees adjust and adapt to new equipment.

This explanation of the phenomenon of epidemic increases in the incidence of Non-specific Occupational Overuse Syndrome has further implications. The fact that the association between a minor change in working conditions and a dramatic change in the incidence of symptoms has been repeatedly observed in the literature implies that the vast majority of busy people in the modern world are operating at the edge of their limit of tolerance. The “alternative theory” suggests that this is not an inappropriate situation in which to operate, provided however, that the potential for going too far is recognised. The theory developed here implies that normal human physiology has a built-in safety margin which is a protection against damage due to overuse. If people understand what to expect from their bodies when they undertake extensive activity and respond appropriately to the stimuli which it produces, then activity can safely be continued at a level near to the tolerance limit. Thus, it can be safe for people to operate at their maximum productivity level. If they do this, however, then occasional aches and pains are to be expected. These symptoms should be recognised for what they are and a response made consistent with their severity. Such symptoms should certainly not be ignored and indeed measures to minimise their incidence and severity should be maintained and extended. Ferguson pointed out the need for continued research “so that work can be made more comfortable, satisfying and efficient.” [Ferguson, 1987]

6.5.1.3. Variation in Incidence

One of the phenomena which has previously been difficult to explain is the situation where there is a marked difference in the incidence of Non-specific Occupational Overuse Syndrome between two work sites where the tasks involved appear to be identical [Bell, 1986][McDermott, 1986]. Theories of contagion [Awerbuch, 1985][Forsyth, 1989] and mass hysteria [Ireland, 1986a][Lucire, 1986] have both been proposed in the attempt to explain this. This theory, however, offers an alternative explanation which is far less controversial.

The process which leads to the development of symptoms, although it is essentially a physical process, is affected by a wide range of factors not solely physical. If there were some cause for high levels of psychological stress in one work site and not in the other then this in itself could be the reason for the increased incidence in the former. For example, the supervisor in one office might have an abrasive managerial style leading to high levels of interpersonal friction whereas the supervisor in the other office might have a more conciliatory approach, producing a more relaxed working atmosphere, although the tasks being performed in both offices are identical.

There are any number of possibilities in the work place which could cause small overall differences in psychological variables among employees, producing consistent, but probably small, changes in the levels of sensitivity in the muscles of all employees. Because symptoms occur at threshold level of sensitivity, an effect which causes a small mean difference in sensitivity levels could lead to a large difference in the incidence of symptoms.

This implies that if an employer wishes to provide the environment for optimum productivity, then the psychological as well as the physical well being of the employees must be considered.

6.5.1.4. Progression of Symptoms and the Presence of Tender Sites

The tendency for symptoms to spread from an initial site of pain to adjacent areas and even to the opposite limb is an accepted characteristic of Occupational Overuse Syndrome. Browne et al suggested that “the anatomical pattern of symptom evolution” is an important aspect of the clinical history [Browne et al, 1984]. Crockett reported the observation that “The initial burning ache spreads gradually and secondarily to the whole of the forearm and then to the other anti-gravity muscles of the arm and shoulder” [Crockett, 1985]. Fry described the characteristic progression of symptoms, “ If he or she persists ... the condition spreads from one muscle group to the adjacent group both proximally and distally.” “The condition may spread to the non-involved side” [Fry, 1986a]. This progression of symptoms is not an issue of contention. Any theory of causation should be able to explain this phenomenon satisfactorily. On the basis of the theory of local pathology which Fry advocated, he argued that this and other features of Occupational Overuse Syndrome suggest production of pharmacologically active substances by the affected muscles [Fry, 1986f]. In contrast, the theory developed in this thesis can explain this phenomenon without recourse to the presence of a spreading disease or the release of abnormal substances.

This theory proposes that the hypothesised Physiological Activity Limitation Process acts to vary the sensitivity of the structures involved in continuous or repeated musculo-skeletal activity. This includes all the structures involved throughout the body but the level of sensitivity at each site may be different and will be the result of the level of activity at that site. The theory proposes that Physiological Activity-related Pain occurs when the sensitivity of a structure reaches a certain threshold.

It is well known that no action or task uses one muscle or joint alone but rather utilises a combination of muscles and joints in a coordinated pattern. Depending on the task, different structures are more or less active, with one of those structures stressed the most. Thus, it is likely that given overuse from any particular task or tasks the sensitivity of one of the structures involved will reach the threshold for Physiological Activity-related Pain first and so symptoms will commonly occur initially in one area.

The explanation for the progression of symptoms suggested by theories of local pathology is that pain spreads from the site where it is first experienced as the pathological process spreads to involve adjacent structures. The explanation for the progression of symptoms suggested by the hypothesised Physiological Activity Limitation Process involves the modulation of sensitivity in the structures of activity. Where this physiological process has raised the level of

sensitivity of one structure to the point of Physiological Activity-related Pain, the level of sensitivity in the other structures involved in the associated task or tasks will also be approaching the threshold at which discomfort will occur. If no modification or reduction in the task(s) occurs as a response to the initial Physiological Activity-related Pain, then the level of sensitivity in those other structures will continue to increase past the threshold so that discomfort is experienced in additional structures and thus the pain appears to spread.

The action of the Physiological Activity Limitation Process, whereby modulation of the sensitivity of the tissues occurs, also explains a number of the phenomena found in this study relating to the presence of sites of tenderness. These include the presence of tender sites both in the absence of symptoms as well as in the asymptomatic areas of individuals with symptoms. It also explains the reason why individuals with more severe symptoms should have been found to have a greater number of sites of tenderness and increased prevalence of tenderness in asymptomatic areas than those with lesser symptoms. In this study the mean number of sites of tenderness increased from 2.5 in the “No Symptoms” category to 7.8 in the “Inconsequential Symptoms” category to 16.2 in the “Consequential Symptoms” category. In line with this the mean prevalence of tenderness in asymptomatic areas increased from 16.6% in the “No Symptoms” category to 37.9% in the “Inconsequential Symptoms” category to 68.9% in the “Consequential Symptoms” category (chapter 4, section 4.4.3.2).

Along with the proposed threshold of sensitivity at which Physiological Activity Related Pain is experienced, the theory proposed in this thesis also suggests that there exists a threshold level of sensitivity at which tenderness can be detected which is lower than the threshold for the experience of Physiological Activity-related Pain.

Asymptomatic individuals

The Physiological Activity Limitation Process will, therefore, cause the sensitivity levels to reach the threshold for tenderness before the threshold for the experience of Physiological Activity-related Pain will occur. It is possible for sensitivity to reach levels high enough for sites of tenderness to be present without ever causing the experience of discomfort associated with activity. This study, as well as previous research [Sola, 1955][Yunus, 1981], would suggest that this is likely to be the case in at least 50% of asymptomatic individuals.

Individuals with “Inconsequential symptoms”

Where an individual is experiencing a minor level of Physiological Activity-related Pain the theory states that at least one site must have reached the appropriate sensitivity threshold. For this to occur it would be expected that the sensitivity levels in the symptomatic structures, as well as in associated asymptomatic structures, would be higher than in an asymptomatic individual. Thus, it is likely that, in the individuals experiencing pain, the sensitivity of more

structures would have reached the threshold for tenderness. A systematic examination would, therefore, be likely to find a greater number of sites of tenderness, both overall as well as in the remaining asymptomatic areas.

Individuals with “Consequential symptoms”

The same argument applies for the difference between individuals with “Inconsequential” and “Consequential” symptoms. Although this study has shown that there are a number of complex interacting factors which lead to the distinction between these two categories, it is reasonable to suggest that one of the major factors is the intensity of the symptoms experienced. If the average severity of the Physiological Activity-related Pain experienced by those in the “Consequential Symptoms” category is greater than that of the “Inconsequential Symptoms” category then it is reasonable to suggest that the Physiological Activity Limitation Process has generated higher levels of sensitivity in the structures where symptoms are being experienced. This in turn would suggest that the associated structures which remain asymptomatic also have higher levels of sensitivity and so more of those structures are likely to have reached the threshold for tenderness.

6.5.1.5. Refractory Occupational Overuse Syndrome

Another phenomenon which has been difficult to explain is the subset of patients classifiable as suffering with the Occupational Overuse Syndrome whose symptoms persist over months to years despite provision of accepted methods of treatment. The methods of treatment, which are referred to as failing to be effective, are usually those which have previously been shown to be suitable for other conditions such as tenosynovitis with which Occupational Overuse Syndrome has been equated. Such treatment often involves rest and splintage. The failure to respond to what has been considered appropriate treatment has been used as evidence of a psychological aetiology for Non-specific Occupational Overuse Syndrome [Brooks, 1986][Ireland, 1986a][Lucire, 1988][Fildes, 1988].

This study suggests four alternative reasons why there is this subset of patients.

6.5.1.5.1. Inadequate Diagnosis

The reason that a number of patients do not respond to treatment will often be because an inadequate diagnosis has been made. The term Occupational Overuse Syndrome (“RSI”) is an umbrella diagnosis covering a whole range of more specific diagnoses [Australian National Occupational Health and Safety Commission, 1986]. The aim of the medical assessment of a patient with activity-related pain should be to reach as precise a diagnosis as possible. If the diagnostic process ceases once the diagnosis of Occupational Overuse Syndrome is reached then there would be a failure to distinguish between Non-specific Occupational Overuse

Syndrome and the various diagnoses contained within the classification Specific Occupational Overuse Syndrome. The result could be that some patients with specific pathology would be given the imprecise diagnosis Occupational Overuse Syndrome and consequently, offered inappropriate management with subsequent poor results.

In this study 27 out of the 56 subjects (48%) in the patient sample were eventually found to have a "Specific" diagnosis. Everyone of these patients originally believed themselves to be suffering from "RSI". The high incidence of specific disease entities in the group of individuals presenting with symptoms consistent with Occupational Overuse Syndrome has been confirmed in the study by Sikorski et al [Sikorski, 1989] where 58% of a group of 204 individuals with occupationally related upper limb or neck pain, were eventually found to have a "recognisable, pathologically discrete, musculo-skeletal disorder" when adequately investigated.

Given that all suitable measures have been taken to detect "specific" pathology and so making the diagnosis of Non-specific Occupational Overuse Syndrome appropriate, there does appear to be a group of such patients who continue to have persistent symptoms, despite accepted management. The theory developed in this study provides the basis for three further compatible, alternative explanations as to how this phenomenon could occur .

6.5.1.5.2. Accepted methods of treatment inappropriate

The contention of this "alternative theory" is that, in the majority of cases of Non-specific Occupational Overuse Syndrome, the symptoms experienced are Physiological Activity-related Pain which is not the result of any disease process from which the patient has to recover. It suggests that management of the condition should involve some temporary reduction in level of activity, provision of appropriate remedial training and/or work modification. This would serve to increase the patients' tolerance to the required tasks.

The usual methods of treatment suitable for pathological conditions, such as complete rest and splintage, will not achieve this, rather the absence of activity which is part of those treatments would decrease the patients fitness and thus their tolerance limit. Also, with rest and splintage, no attention is being directed towards identifying and changing habitual activity patterns, which could be a factor leading to a low tolerance limit. Whenever patients attempt to return to the activities initially causing the problems they often do so using the same techniques as before with the hope that the "disease" causing the symptoms has resolved making the resumption of activity "safe". They are then trying to perform the same activities with a lower tolerance limit and so symptoms re-occur even more quickly. Thus, the level of activity needed to cause symptoms will progressively decrease and subsequently other types of activity at progressively lower intensity will produce the same symptoms. Clearly there will be psychological factors which have an influence on whether this cycle of decreasing fitness and increasing sensitivity develops. It can be seen however that inappropriate treatment could be the primary cause.

6.5.1.5.3. Difficulty in changing habits

A second mechanism which the “alternative theory” implies evolves out of consideration of the nature of the process hypothesised. It suggests that the discomfort experienced occurs within normal healthy tissues as a result of normal day to day activities such as writing, typing, playing musical instruments, all of which have been learnt and practiced over years. In effect, it means that those who have developed pain as a consequence of their activities are in some way doing something inappropriate for their physical state at that time. It can be said that there is no external problem, such as a disease, the problem is internal, quite possibly caused by the habits of years. Any approach to treatment should achieve some change in that person’s life style or occupational habits if resolution of symptoms and comfortable return to the activity is to occur.

The reason being proposed here, for the failure of treatment in a proportion of cases, is the acknowledged difficulty that the medical profession has in achieving beneficial life-style changes in many areas of health, be it fitness, obesity, smoking or daily professional activities.

In many ways it would seem to be easier to accept that a disease has been contracted than it is to accept that the approach to day to day activities must be changed to overcome the problem in hand. Those who wish to find a “quick fix” for their problems will not welcome this explanation. The factors which lead to the development of Physiological Activity-related Pain are multi factorial and indeed have not yet been fully enunciated. The research findings here, as well as the wider literature, suggest that the factors include at least the level of aerobic fitness, muscle strength, poor technique, unsuitable ergonomics of the environment in which activity is undertaken, inefficient work practices, work stress, personal attitudes and psychological tendencies and age of the individual.

In any individual the differing factors will have varying importance. Where the solution to the problem requires changes in established attitudes and habits, there is understandably going to be conscious and/or unconscious resistance from the individual involved. To accept and undertake a change, such as learning a new hand writing style or increasing physical fitness, is both difficult and stressful in itself. This is perhaps the most common reason for failure of treatment.

The following case example taken from the patients treated as a result of this study illustrates this point.

History

Ms NS, a 14 year old right handed school girl who had been playing the violin since age 9 presented with pain in the right hand when writing and intermittent pain in the left when playing

the violin. At presentation, the pain associated with writing had been present for two years. It always occurred after a variable period of writing and settled over 30 to 40 minutes. During the previous few months this pain had started to interfere with her ability to do school work. The pain associated with violin playing had been present for 12 months and only occurred intermittently, approximately twice per week. It did not interfere with her music activities at all.

Assessment

Assessment included thorough physical examination, plain x-rays of cervical spine and hands, bone scan, complete blood examination, Rheumatoid blood screens, ESR and detailed motor skills assessment by the occupational therapist. The only abnormality found was widely distributed sites of tenderness throughout the both upper-limbs and neck and some problems with her writing style. The occupational therapists report stated "She was found to have decreased grip strength (particularly in the right hand), poorly established dominance for non-writing tasks, and a fluctuating pencil grip."

The diagnosis made was that she had Non-specific Occupational Overuse Syndrome due to an habitual poor writing style compounded by increasing demands for writing at school and high personal and parental expectations.

Management Plan

The management plan was to establish a systematic routine of writing activities during the day such that writing activities could be undertaken for short period only and so minimising discomfort. A program of retraining of writing style was undertaken by the occupational therapist including use of biofeedback techniques. She was also instructed in relaxation techniques.

Progress.

Over the next month she reported a marked reduction in discomfort.

One month later symptoms had re-occurred and she stated that she had difficulty maintaining the new writing style because of pressure to keep up with work at school.

The situation remained essentially unchanged for the next 3 months. There appeared to be a significant resistance from her, and her mother, to commit herself to the management program. She reported that she often had to do more than the occupational therapist had decided seemed appropriate and was unable to limit herself to using the improved writing style she had been taught. The possibility that there may have been psychological factors involved which were not initially apparent was considered and she was referred for psychiatric assessment.

The assessment by a consultant child psychiatrist was very enlightening as the following extracts show.

“Ms NS was unable to make use of the programme at the hand clinic at the Royal Adelaide Hospital due to lack of commitment. She did not want to stop writing completely as was suggested and often continues writing until she achieves pain, something which she has been told not to do. “

“On mental status examination, she was extremely tense throughout the session. There was no evidence of any depressive disorder or any thought disorder. She is extremely worried about her school work and has set herself the high standard of wanting to get into Medical School after year 12. She seems to have very few outlets for her tension and does not seem to know how to relax.”

“I don't think that she is suffering from a Conversion Reaction as I could not find any dynamic meaning for the loss of use of hand function and nor was there any secondary gain present. However, I do feel that her tense, perfectionistic temperamental style is aggravating her problem and has led to her sabotaging her treatment plan at the Royal Adelaide Hospital.”

The psychiatrist spent some time discussing the various options open to her. These included making a firm commitment to the management programme at the Royal Adelaide Hospital as well as the option of no further treatment.

After that interview she decided to make a commitment to the previous management programme. After 1 week of no writing and 2 weeks gradual reintroduction of remedial writing training with the parallel strengthening and relaxation exercises she had become pain free. One month later she had returned to a normal writing style and continued to be pain free.

The difficulty in achieving a persistent change in habitual activities may lead to both the patient's failure to improve and the treating practitioner's frustration in the attempt to deal with the problem. Once again psychological factors must have an influence on what is primarily a physical problem but in this situation the treating practitioner should have an understanding and tolerance of the psychological difficulties that the patient is experiencing.

6.5.1.5.4. “New” Pathological Conditions.

This “alternative theory” proposes that the symptoms of the majority of individuals with Non-specific Occupational Overuse Syndrome are the result of a healthy physiological process. Nevertheless, as the classification Non-specific Occupational Overuse Syndrome is by definition a diagnosis of exclusion, it can not be claimed that all cases are due to this proposed healthy physiological process. It remains likely that a number of cases will be due to other conditions as yet uncharacterised.

The characteristics identified in this study which are common to the group of individuals with Non-specific Occupational Overuse Syndrome, have led to the development of this hypothesised healthy Physiological Activity Limitation Process. It is these characteristics which should help to differentiate between Physiological Activity-related Pain and some other pathological entity. A history of extensive or unaccustomed activity, signs of widely distributed sites of tenderness, particularly in asymptomatic areas, and reduction in symptoms after an appropriate period of reduced or modified activity would be reassurance that the problem was Physiological Activity-related Pain. Where this is not the case, then an open mind would have to be maintained as to the possibility that either a specific diagnosis has been missed or that some as yet uncharacterised condition is present, and the possibility that this could be either physical or psychological would need to be considered.

One of the fundamental implications of the theory developed in this study is that it is possible for extensive regular activity to cause a pathological change if taken to extremes. The theory proposes that it is this risk which has led to the evolutionary development of a protective physiological process aimed at averting this occurrence. This study does not attempt to investigate the nature of such pathological changes. It is reasonable, however, to conjecture that those conditions which fall under the classification of "Specific Occupational Overuse Syndrome" such as Peritendinitis Crepitans, Carpal Tunnel Syndrome, Tenosynovitis (with histologically demonstrable synovitis) are enough to warrant the proposition that such a protective physiological process might exist.

6.5.1.5.5. Pathological Derangement of the Physiological Activity Limitation Process.

It is proposed that the physiological process hypothesised in this thesis, the Physiological Activity Limitation Process, functions by inducing unpleasant sensations, Physiological Activity-related Pain, in appropriate circumstances so that the body is protected from the potentially harmful effects of excessive activity. If this is the case then it would be expected that there would be detectable differences between individuals with and without the experience of Physiological Activity-related Pain. Providing that this process is functioning in a physiological manner, that is responding appropriately to environmental stimuli, then any physical changes which it might generate should be physiological in both magnitude and character. They should be reversible, quantitative changes rather than qualitative changes which would be suggestive of irreversibility and by implication damage or injury. No opportunity has yet been available to undertake physiological studies looking for such changes.

All physiological processes, on the other hand, have the potential to become deranged in certain circumstances and such derangements could cause problems themselves. Consequently the very existence of the Physiological Activity Limitation Process implies the potential for the existence of pathological conditions which are caused by the development of derangements in

the function of the primary physiological process. Conceptually, any or all of the anatomical locations and processes involved in this hypothetical physiological process, have the potential for such dysfunction. Dysfunction could either involve normal physiological changes occurring in inappropriate circumstances, for example, increased sensitivity leading to Physiological Activity-related Pain in the absence of excessive activity or pathological changes represented by detectable qualitative abnormalities.

If the search for physical abnormalities in medical conditions directly related to Non-specific Occupational Overuse Syndrome is interpreted in the light of the possibility that the Physiological Activity Limitation Process might become deranged, evidence for such dysfunction is present.

One study which has sought for a peripheral anatomical site of pathology in the context of Non-specific Occupational Overuse Syndrome is that by Dennett and Fry [Dennett & Fry, 1988]. They have performed muscle biopsies from the 1st dorsal interosseous muscle in a series of patients (N=29) with Non-specific Occupational Overuse Syndrome and compared them with similar biopsies from controls (N=8). Patients were defined as individuals previously diagnosed as suffering from "painful chronic overuse syndrome" and who had been treated for a minimum of 6 months with a limitation of activity regime devised by one of the authors. Changes which were described were morphological in nature including changes in fibre diameter and distribution of fibre type. Other abnormalities described included mitochondrial changes with "scalloped and moth-eaten" mitochondrial fibres and muscle fibre changes with the presence of "ring fibres". These changes were present in controls but were more common in the patient group. Notably the study did not find evidence of acute changes, necrosis or atrophy. The fact that the patients had been treated for at least 6 months with a "radical rest" program amounting to almost complete inactivity led to the paper being criticised on the grounds that the histological changes observed may have been due to treatment rather than due to primary pathology. Their study was of a preliminary nature with only a small number of cases. In addition, abnormalities were not seen in all the patients and clear distinction between controls and patients was not achieved. The significance of the abnormalities remains uncertain although there is the possibility that the variation detected in this highly selected group of patients with refractory Non-specific Occupational Overuse Syndrome represents changes within the normal range, indicative of a physiological rather than pathological alteration.

Other studies, looking for local changes at sites of tenderness in muscles, have been performed on patients with fibromyalgia. This condition has similarities to the Non-specific Occupational Overuse Syndrome, the main difference being that an association with excessive physical activity is not required. Pain and tenderness exacerbated by activity is a common feature, as is the lack of objective laboratory or radiological abnormalities. Ferguson has pointed out that this term has been used in the Australian context as the diagnostic label for patients who do fall within the classification of Non-specific Occupational Overuse Syndrome [Ferguson, 1984].

Smythe [Smythe, 1988] reiterated the theory that the Non-specific Occupational Overuse Syndrome is in fact a form of fibromyalgia. A Swedish group [Bengtsson et al, 1986b][Bengtsson et al, 1986c][Lund et al, 1986] have reported abnormalities within muscle in patients with “primary fibromyalgia”. They found “reduced high energy phosphate levels”, “decreased muscle tissue oxygen pressure” and similar histological changes to the findings of Fry and Dennett in the muscles of patients at sites of increased sensitivity or in other words “trigger points” in “fibromyalgia terminology”. Once again the changes found, demonstrated a quantitative difference rather than a qualitative difference between those affected and the control subjects and so may only have indicated a variation within physiological limits. In a “Controlled and Blinded Study” Yunus compared the electron microscopy findings from trapezius muscle biopsies of 21 fibromyalgia patients with 11 healthy controls. “Common findings in both the fibromyalgia and the control groups were mild myofibrillar separation, papillary projections and subsarcolemmal accumulation of glycogen. However, the differences between the 2 groups were not statistically significant in any of the findings.” As a result of the absence of statistical difference between patients and controls, Yunus suggested “that such changes may be physiological in the trapezius muscle.” [Yunus et al, 1989c] Jacobsen investigated the light microscopy abnormality of “rubber band morphology” comparing 48 randomly selected cases of fibromyalgia with 24 randomly selected cases of “Chronic Myofascial Pain”. This abnormality was found in 63% of the fibromyalgia cases and 29% of the myofascial pain cases.[Jacobsen, 1991]. A statistically significant difference was found in this study but once again this was quantitative rather than qualitative.

Although these studies did not definitely confirm the presence of local pathological change, the demonstration of objective evidence of quantitative changes within muscles at sites of increased sensitivity is evidence of some physical process occurring, which is involved in the expression of discomfort due to mechanical stimulation of the structures of physical activity. The failure to find any qualitative difference between patients and controls with such detailed histological analysis might be taken as evidence against the theory proposing the presence of truly pathological changes (damage or injury) in the muscles of such cases.

A study by Russell [Russell, 1989] investigating changes in neurotransmitter concentrations in the Cerebro-Spinal Fluid of patients with fibromyalgia, discovered elevated levels of “substance P” as compared with controls. This was thought to indicate increased primary nociceptor sensitivity.

There has been considerable research looking for neural rather than muscular abnormalities, specifically in the context of Non-specific Occupational Overuse Syndrome. Observations of aberrant neural behaviour in this context have been reported and commented on repeatedly. The inconsistency of such observations, however, has meant that, although many theories have been proposed, no accepted conclusions have been reached concerning their implications for Occupational Overuse Syndrome.

Hochberg described the experience of “Hand Difficulties Among Musicians” at the Massachusetts General Hospital with a series of 100 patients. “Disorders of motor control” made up 24% of cases. “The most frequently occurring motor control problem, manifested as involuntary palmar curling of the right fourth and fifth fingers (18%), or the second, third and fourth fingers (9%) into to palm.

In December 1983 Lowy [Lowy, 1983] proposed the hypothesis that “occupational overuse and RSI-type disorders, result from abnormal neurovascular activity caused by dysfunction of the autonomic receptors”. He based this hypothesis on his observation that “common symptoms experienced by patients with RSI include pain and burning, paraesthesiae, altered sensation, restriction of movement, swelling and tenderness” and that in “more severe cases, changes such as discoloration and mottling, hyperhydrosis, temperature alteration , oedema, and alteration in sensitivity, take place”. He raised the possibility that the “underlying mechanism is a sympathetic disturbance” akin to “reflex sympathetic dystrophy”. This hypothesis was dismissed out of hand by Stone [Stone, 1983b] who commented that such changes are only “infrequently” observed and concluded by stating “No direct link between repetitive strain injury and reflex sympathetic dystrophy is known to exist.” Graham and Mills supported Lowy however. They stated, “Our experience, covering more than 350 cases of upper limb repetitive strain injury, indicates that sympathetic overactivity in the affected limb is common, particularly in patients who have a long history of symptoms.” [Graham and Mills, 1984]

Crockett reported observations consistent with aberrations of motor function. “Some cases of “RSI” in fact exhibit writer’s cramp, and there may be evidence that, after a period of discomfort caused by a particular type of use, confusion develops in the pathways for voluntary fine repetitive movements in the brain” [Crockett, 1985]

In a review article McDermott commented “Autonomic disturbances (such as Raynaud’s phenomenon or reflex sympathetic dystrophy) occasionally have been described.” [McDermott, 1986]

Gow proposed an inter-relationship between various forms of “occupational pain syndrome” in which he suggested that “Autonomic nervous system overactivity” played a role in the progression of “overuse syndrome” to “Regional pain syndrome” and thence to “Reflex sympathetic dystrophy”. He suggested that these three conditions could be equated with the three stages of RSI as defined by Browne, Nolan and Faithfull [Browne, Nolan and Faithfull, 1984]. The basis for his proposal was the observation of features suggestive of “autonomic nervous system” dysfunction “such as dysaesthesia and subjective swelling” [Gow, 1987]. He also likened “Stage 2” RSI to “generalised fibrositis syndrome” referring to the equivalence between the two suggested by Littlejohn [Littlejohn, 1986c]. It is interesting to note that one of

the many theories proposed to explain fibrositis is that of “an abnormality of pain modulation” [Smythe, 1979].

A paper on Occupational Cramp by Fry and Hallett [Fry and Hallett, 1988] compared and contrasted this condition, which they equated with Focal Dystonia, and Non-specific Occupational Overuse Syndrome or as they labelled it “painful musculo-ligamentous overuse”. They commented that “The relationship between the very common painful musculo-ligamentous overuse and the less common painless occupational cramp is unclear, although both occur in patients involved in hand-use-intense activity”. “Occupational cramp seems to be a true focal dystonia, and it may be precipitated by overuse”. In this paper Fry reported that he “has seen patients with a spectrum of symptoms bridging these two presentations ... Patients with advanced musculoligamentous overuse may demonstrate loss of voluntary control similar to patients with ‘cramp’.” Despite what appeared to be an attempt to link these two conditions with the sensory abnormality of Non-specific Occupational Overuse Syndrome at one end of a spectrum and the motor abnormality of Occupational Cramp at the other, the paper concluded its discussion with the suggestion that current evidence favours the hypothesis that the primary pathology of Non-specific Occupational Overuse Syndrome is “peripheral” whereas the primary pathology of Occupational Cramp is “central”. Objective evidence for the presence of organic neurological abnormalities in cases of Occupational Cramp were reported from previous work by Hallett. Fine wire EMG studies demonstrated absence of reciprocal inhibition, evidence of the prolonged spasms characteristic of dystonia and evidence for disordered motor control. They suggested that their most objective abnormal finding was an abnormality of H-reflex inhibition in the affected arm of patients with unilateral Occupational Cramp. They concluded that this implied the presence of “disordered spinal circuitry”.

Lockwood and Lindsay reported 4 cases of “overuse syndrome” in musicians which progressed to Reflex Sympathetic Dystrophy. One of these cases subsequently went on to develop a focal dystonia. They put forward the hypothesis “that RSD is a sensory analog of focal dystonia”. The features leading them to make the diagnosis of RSD were “burning and dysesthetic pain with intensity far in excess of that expected”, “abnormal vasomotor responses, edema, alteration in skin colour, accelerated hair growth, loss of joint mobility and spread of the syndrome”. [Lockwood and Lindsay, 1989]

Cohen et al [Cohen et al, 1992a] have proposed their own hypothesis for the pathophysiology of “RSI” for which they also proposed a new name, that of Refractory Cervicobrachial Pain Syndrome or RCPS. Their hypothesis was “that RCPS is a reflex neuropathic state due to a central disturbance of nociception induced by continual afferent barrage from nociceptors and mechano-receptors in anatomically relevant sites” which may include “spinal zygapophyseal joints, muscles, tendons, joint capsules” or peripheral neural structures. This hypothesis proposed that the site of central disturbance be the “wide-dynamic-range (WDR) neurones” in the dorsal horns of the spinal cord. They based this hypothesis on the observations from a

“careful clinical study” of a large group of patients, “in excess of 1000”, with Non-specific Occupational Overuse Syndrome, or what they call RCBP, who had failed to respond to accepted management and who were seen in a tertiary referral setting. In this group of patients they found “a number of clinical observations which constitute a remarkably homogeneous profile”. These included absence of abnormality on laboratory investigation, “consistent phenomena suggesting perturbation of neural functioning ” including both sensory and motor phenomena, “commonly signs suggestive of local sympathetic dysfunction”, presence of “exaggerated wheal/flare response” and absence of influence from “social class, cultural or ethnic origin and anxiety or depressive features”.

It is interesting to note the vehement response to this article in the letter’s to the editor in the Medical Journal of Australia. Some of the criticisms are worth considering in the light of the Physiological Activity Limitation Process proposed in this thesis. A criticism of Cohen’s theory, common to a number of the subsequent letters to the editor, pointed to the failure to “identify the source(s) of ongoing nociceptive barrage” which was required by their hypothesis to induce the pathological “central disturbance”. Awerbuch [Awerbuch, 1992a] doubted that “stimuli induced by occupational activity” could be seen to “constitute a bombardment of noxious stimuli”. Cleland [Cleland, 1992] complained that this “afferent barrage” was “uncharacterised”. Quintner [Quintner, 1992a] pointed out that while Cohen stated “that the origin of sensitisation of WDR neurones remains conjectural” this statement in itself “implies the existence of an as yet undiscovered mechanism”.

The theory developed by this thesis provides this “undiscovered mechanism” required to justify the hypothesis of Cohen et al. The issue which was being raised in these letters was that Cohen’s theory proposed a pathological change in the context of normal activity and proposed that some aspect of that normal activity induced that pathological change. Cohen stated that the “afferent barrage” of impulses originated from “anatomically relevant sites” and implied that it consisted of normal impulses resulting from “mechanical stimulation” but that, given the appropriate circumstances, they caused a change in the central nervous system (the dorsal horn neurones) leading to a lowering of the central pain threshold such that these impulses were interpreted as painful at lower intensity than normal. Previous research [Campbell et al, 1988][Yaksh, 1989][Roberts, 1986] had shown how noxious stimuli can lead to such changes. Cohen implied that the impulses resulting from normal mechanical stimulation due to activity could behave in the same way as other types of impulse resulting from clearly noxious stimuli.

The common feature of the “alternative theory” developed in this thesis and Cohen’s hypothesis is the proposed alteration in a threshold level at which a normal stimulus causes a painful experience. The exposition of the “alternative theory” proposes a physiological alteration in the mechanisms controlling the sensitivity of structures involved in activity, such that a normal stimulus causes a temporary experience of discomfort/pain with the beneficial result of limiting excessive activity. Cohen’s hypothesis proposed a pathological alteration such that a normal

stimulus causes a persistent painful experience, refractory to appropriate reduction or modification of activity. The “alternative theory” justifies itself and, by implication, justifies Cohen’s hypothesis, if it can be accepted that his hypothesis simply describes a pathological aberration of the Physiological Activity Limitation Process. The work by Cohen et al can thus be seen as pointing to the “Wide Dynamic Range anterior horn cells” of the spinal cord as one of the anatomical structures involved in the Physiological Activity Limitation Process proposed in this thesis. His hypothesis also appears to be delineating one form of aberration which might occur in that physiological process.

The likelihood that the pathological process proposed by Cohen might only describe a small proportion of the cases of Non-specific Occupational Overuse Syndrome was raised by Hocking [Hocking, 1992]. He criticised Cohen’s hypothesis on the basis that it “implies that refractory cervicobrachial pain was the endemic complaint”, in other words, Cohen’s hypothesis implied that the majority of cases of Non-specific Occupational Overuse Syndrome which occurred as part of the Australian epidemic of the early 1980’s could be explained by this hypothesised spinal pathology. Hocking suggested that “Simple pain in the arm is more likely to be the endemic complaint” and went on to propose that “dorsal horn dysfunction is responsible for but a small fraction of RSI cases.” He was, thus, reiterating the comments made by Stone in 1983 [Stone, 1983b] that, from the perspective of the primary health provider, the incidence of features consistent with a clear disturbance of neural functioning is low.

If it is accepted that the cases described by Cohen may have involved only a small subset of the cases of Non-specific Occupational Overuse Syndrome, then it is reasonable to conclude that the circumstances which led to the aberration in the Physiological Activity Limitation Process making them refractory to treatment suggested by his theory need not bear a direct relationship to the level of activity.

Helme et al [Helme et al, 1992] used a case-control study to seek abnormalities of primary afferent nociceptive mechanisms in patients with RSI. “The function of primary afferent nociceptive mechanisms was assessed by the size of capsaicin-induced flare responses. A reduction in flare size was observed in the pain affected limb of RSI subjects, but an increased response occurred in sites reported as unaffected by clinical pain.” They concluded that “these findings provide objective evidence of altered nociceptor mechanisms in RSI subjects, and are consistent with the view that this chronic pain syndrome involves somatic pathophysiology.”

The research described in Helme’s paper sought to answer the question of whether the process causing pain in Non-specific Occupational Overuse Syndrome (RSI) is somatic or psychological. The assumption was made that if a somatic abnormality were found it would imply somatic pathology. The theory developed in the present study, however, raises the possibility that a somatic abnormality might be the result of a physiological process, that of the

hypothesised Physiological Activity Limitation Process. Certainly the change in flare response size indicates a change in the primary afferent nociceptive response to a set stimulus but whether this change is due to a pathological alteration or to a physiological alteration is an unanswered question. The observations of an altered flare response are also consistent with the view that there is a physiological alteration in nociceptor mechanisms in patients with Non-specific Occupational Overuse Syndrome (RSI) and so may well represent direct objective evidence of the validity of the “alternative theory” which is proposed in this thesis.

The findings of an altered flare response in the unaffected limb of “RSI” patients described in Helme’s work offers an interesting parallel with one of the findings found in this study. Helme reported “an increase in flare size in the unaffected limb of RSI subjects when compared to controls” and suggested that this may be due to “a reduced pain threshold to mechanical stimuli” in the unaffected limb. He pointed out that “similar findings have been reported for patients with fibromyalgia” [Littlejohn, Weinstein and Helme, 1987]. The results of this thesis found that the prevalence of tenderness in the unaffected limb in the area opposite to a symptomatic area was higher than the prevalence of tenderness in asymptomatic areas in general (Paired Student's t-test, $p < 0.0001$) This also suggests a reduced pain threshold to mechanical stimuli, in this case specifically in the area of the unaffected limb opposite to the site of symptoms.

Woolf [Woolf, 1983] developed an experimental animal model of pathological pain caused by thermal burn, with the use of chronically decerebrate rats. With this model he was able to confirm previous evidence that local changes caused by noxious stimuli led to locally increased sensitivity in the areas adjacent to the area of injury which were themselves not damaged. In addition to this he was also able to show that the stimuli generated by the thermal burn led to increased sensitivity not only of the structures in the ipsilateral area adjacent to the burn but also of the structures in the contra-lateral limb. He was able to demonstrate by the careful manipulation of experimental variables that the changes observed in the contra-lateral limb were due to increased “excitability of the spinal cord”. He concluded from this that the mechanisms that modulate the sensitivity of peripheral structures as a response to one form of pathological pain, that of a thermal burn, was in part “due to changes in the central nervous system”. “The long-term consequences of noxious stimuli result from central as well as from peripheral changes”.

The findings of changes in the unaffected limb mentioned above both from Helme’s work and this study, seem to parallel those of Woolf. A reasonable conclusion is that, just as with “pathological pain”, the effect of “physiological pain” associated with the Physiological Activity Limitation Process hypothesised in this thesis, is in part modulated by changes in the central nervous system. This is further confirmation of the conclusion drawn above from consideration of the work of Cohen et al [Cohen et al, 1992a] that the Physiological Activity Limitation Process may involve processes within the spinal cord.

In summary, findings of research drawn from various sources have demonstrated changes in the local tissues and neural mechanisms involved in the sensitivity of the structures of activity. The consistency and spectrum of these findings does indeed provide support for the presence of the primary physiological process proposed in this thesis, that of a Physiological Activity Limitation Process. It also lends support to the proposition that aberrations of this mechanism may occur with significant sequelae.

One of the implications of this is that at least two sub-groups of Non-specific Occupational Overuse Syndrome can be defined. The first sub-group would consist of cases which are due to the normal function of the Physiological Activity Limitation Process in which symptoms do respond appropriately to environmental stimuli. The second sub-group would consist of cases which are due to dysfunction of the Physiological Activity Limitation Process in which evidence of neural dysfunction may be present and in which the response to both environmental stimuli and various forms of treatment may be variable. Cases from the second sub-group would explain a proportion of the cases which are refractory to treatment.

The relative prevalence of these two sub-groups is unclear. The consensus would seem to be that cases with evidence of neural dysfunction are rare. The survey reported by Gow suggested that the prevalence of minor musculo-skeletal symptoms was 50% compared with the prevalence of symptoms consistent with "regional pain syndromes or reflex sympathetic dystrophy" of 0.3% [Gow, 1987]. Most of the papers referred to above have started from the perspective of tertiary referral where common or simple cases are filtered out and only rare or difficult cases remain. On the basis of this select sample they have looked for a consistent pattern from which to derive an explanation of the cases they have seen and thence to extrapolate to Non-specific Occupational Overuse Syndrome as a whole. In contrast, the sampling method for the cross-sectional and prospective cohort arms of this thesis was that of a survey of a group of individuals from a "normal" population, without selection, firstly looking for the prevalence of abnormalities and secondarily, a followup study of a subgroup of that survey, selected on the basis of "normality". Consequently any conditions or abnormalities detected in this study would be likely to be seen commonly. Certainly none of the subjects seen in the study with "inconsequential symptoms" of discomfort associated with activity, reported additional symptoms suggestive of sensory or motor abnormalities. In the "Consequential Symptoms" diagnostic category such symptoms were also uncommon. It is reasonable to suggest that analysis of the abnormalities seen in this study would be more likely to have a direct bearing on the nature of Non-specific Occupational Overuse Syndrome as it occurs most frequently. Nevertheless, the findings of the studies from each perspective are not necessarily incompatible and indeed, if interpreted from an overall viewpoint can be seen to be complimentary. It remains reasonable to suggest that the majority of cases of Non-specific Occupational Overuse Syndrome are due to normal functioning of the hypothesised Physiological Activity Limitation Process.

The findings of this thesis have demonstrated the validity of Wright's [Wright, 1987] criticisms of the early work on Non-specific Occupational Overuse Syndrome which treated all cases as a single condition at various stages or grades [Browne et al, 1984][Fry, 1986b]. The implication inherent in these grading systems was that individuals at the various levels within the grading system were being affected by the same process and that progression from a minor level to a higher level was likely. Wright's major criticism was that there was no evidence of the prognostic value of the grading systems proposed. This study has shown that the presence of "inconsequential symptoms" does not indicate a risk of more severe or refractory symptoms. Just as forgetting a friend's telephone number on a few occasions does not mean that Alzheimer's disease is developing, so the experience of discomfort/pain in a working situation does not mean that the activity control mechanisms are failing. The presence of Physiological Activity-related Pain does not imply an increased risk of an individual developing an aberration in the process which caused that experience.

The theory developed in this thesis provides not only an explanation for the most commonly seen form of Non-specific Occupational Overuse Syndrome but also provides the basis for an explanation for a number of previously mysterious conditions which also fall within its classification. These include fibromyalgia (fibrositis), myofascial pain and reflex sympathetic dystrophy, which can be seen to be due to sensory dysfunction, as well as focal dystonia, which can be seen to be due to a motor dysfunction. It can be reasoned that these pathological conditions occur as a result of a breakdown in some aspect of function of the Physiological Activity Limitation Process hypothesised. Different aberrations lead to the different conditions and thus the various clinical syndromes which seem to be associated with hand-use intensive activity become comprehensible.

Fibromyalgia may occur in the absence of excessive activity and is characterised by wide-spread pain and tenderness. The apparent heightened sensitivity of the musculo-skeletal tissues may be caused by an inappropriate general lowering of the body's threshold for Physiological Activity-related Pain as a result of dysfunction of the Physiological Activity Limitation Process. Research discussed above would seem to implicate both peripheral and central mechanisms as involved in this dysfunction.

Myofascial pain syndrome and fibromyalgia have been equated [Simons, 1986], the former being a localised form of the latter. Myofascial pain syndrome is characterised by the presence of one or more hypersensitive areas in muscle which are exquisitely tender and which refer pain to associated areas. The advocates of the myofascial pain syndrome concept label these "trigger points".

From the interpretation of the "alternative theory" developed in this thesis, the "trigger points" of myofascial pain can be seen to occur in two contexts.

Where such hypersensitive sites occur in the context of extensive activity they could simply represent one aspect of the expression of the Physiological Activity Limitation Process, part of the mechanism whereby it generates Physiological Activity-related Pain. In this context such “trigger points” should not be considered pathological. Management of Physiological Activity-related Pain in this context should be directed at measures to decrease musculo-skeletal sensitivity and towards increasing the threshold of activity at which Physiological Activity-related Pain is experienced rather than at “treating” the associated “trigger points”.

Where “trigger points” occur in the absence of extensive activity then it is reasonable to assume that they represent a pathological response. From the interpretation of a pathological derangement of the Physiological Activity Limitation Process, myofascial pain can certainly be seen as a localised form of fibromyalgia. The apparent heightened localised muscle sensitivity may be caused by an inappropriate localised lowering of the threshold for Physiological Activity-related Pain. The anatomical location for the aberration leading to this phenomenon may or may not reside in the muscles themselves.

Woolf has suggested that autonomic changes should be expected as a response to physiological pain. An aberration in the mechanisms generating such autonomic responses could result in the development of excessive and prolonged autonomic changes, such as have been commonly reported in cases of Non-specific Occupational Overuse Syndrome. The precise stage at which such responses change from being physiological to being pathological remains to be elucidated. Nevertheless cases of Non-specific Occupational Overuse Syndrome in which severe autonomic dysfunction has developed, consistent with the diagnosis of Reflex Sympathetic Dystrophy, can be explained on the basis of a derangement of the normal physiological mechanisms leading to such autonomic responses.

An explanation for the pathophysiology of focal dystonia can be developed directly from consideration of the primary features of physiological pain as explained by Woolf [Woolf, 1989]. He indicated that one of the primary features of physiological pain is its association with an unconscious “flexion withdrawal reflex”. The normal healthy operation of this reflex must operate at centres of the central nervous system below the level of consciousness, as it is seen to be activated before the experience of the associated physiological pain reaches conscious perception.

In the normal and healthy operation of the Physiological Activity Limitation Process, however, the generation of a flexion withdrawal reflex would not be expected. The circumstance in which this process occurs is that of a safe and routine situation of normal activity. In such a circumstance an instant unconscious movement would not be likely to be beneficial or “protective”. On the other hand the characteristics of the unpleasant sensations, which it is proposed be called Physiological Activity-related Pain, are not inappropriate for the circumstances in which the Physiological Activity Limitation Process is operant. They are not

sudden or sharp sensations but usually come on gradually at low severity, building up in intensity over time. There are other situations in which physiological pain occurs which also lack both a potential role and any detectable presence of this unconscious withdrawal response. These include the example of fatigue pain described by Parker and Denzin [Parker and Denzin, 1990a] and the example of pain induced by lying in one position for too long described above.

It seems likely, however, that the neural mechanisms which process different forms of physiological pain share some common pathways. Thus, an aberration in the processing of the stimuli leading to Physiological Activity-related Pain may impinge on the centres which lead to the withdrawal reflex in other circumstances. Activities which normally only have an impact on the Physiological Activity Limitation Process could through an aberration in central processing, lead to unconscious, uncontrolled movements. This is precisely the observation made in focal dystonia.

Further research would be required to discover whether or not there are factors other than random events which may lead to the aberrations in the Physiological Activity Limitation Process which cause these conditions and indeed to better define the abnormalities themselves. Equally important would be the development of the means to discriminate between the experience of pain/discomfort due to healthy physiological function (Physiological Activity-related Pain) and the pain due to a derangement of this process. Nevertheless, it is clear that these conditions could make up a proportion of the cases of "refractory" Non-specific Occupational Overuse Syndrome.

Although the theory developed in this thesis suggests that the primary physiological process which is the substrate of these uncommon conditions, the Physiological Activity Limitation Process, is directly influenced by changing levels of activity, the possibility that aberrations of this physiological process are caused by excesses of activity is not within its frame of reference. Thus, the question as to whether or not these less common conditions can be said to be due to excessive activity remains open and no doubt will remain an issue of contention for some time. If, however, the hypothesis proposed in this thesis is proved to be correct, then future research into these conditions will be better able to focus on appropriate areas for investigation.

6.5.2. Reassurance to patients diagnosed.

The acceptance of the theory developed by this research would allow medical professionals to give reassurance to the majority of patients diagnosed as suffering from the Non-specific Occupational Overuse Syndrome and would also suggest a rational approach to management. It would be possible to reassure patients that it is likely that their symptoms are caused by the activities with which they associate them, but the presence of the symptoms does not necessarily imply an injury. Thus, contrary to the consequences of previous hypotheses, they do not need to fear that either they are "going mad", which is the common man's interpretation

of the psychological theories, that they have suffered an injury which may lead to permanent incapacity, the theory of physical pathology, or that they will be perceived as “cheats” according to the malingering theory. The expectation will be that only in a minority of cases will a serious pathological condition be the basis for the problem which in turn might cause symptoms to persist. In the vast majority of cases management of the condition would be directed towards measures to control symptoms, to optimise the methods used to undertake the activity which is causing problems and towards measures to maximise fitness, both physical and psychological, in a way appropriate for the occupation of the patient.

6.5.3. Shared Responsibility for Occupational Fitness

One of the consequences of the management approach suggested by this theory is that both employer as well as employees will have to take part of the responsibility for the prevention of Non-specific Occupational Overuse Syndrome.

Employers will need to provide ergonomically designed equipment and utilise appropriate work practices to minimise the physical and psychological stresses of the tasks required. These are, fortunately, simply the requirements of good occupational health and safety guidelines. It may be that, in addition to this, employers could provide facilities for employees to help increase their fitness in a way appropriate to the tasks involved.

Equally, employees must take responsibility for utilising the facilities provided to make the demands of the job reasonable. They will also have to ensure that they achieve and maintain the level of fitness necessary for the activities required.

What is not identified here is the level of fitness necessary for a specific activity and how it is to be achieved. The concept of “occupation specific fitness” is a new idea which arises as a direct consequence of the theory set out in this study. This would necessarily become an important area of research. Extrapolation from the results of research in sports medicine would suggest that there is no single requirement for increased fitness overall. Fitness is indeed task-specific and thus specific training regimes would need to be developed for each task or occupation where there was a risk of a significant impact from Physiological Activity-related Pain.

6.5.4. A new role for the “Occupational Health Professional” the “Occupational Trainer”

The Australian National Occupational Health and Safety Commission’s report on RSI 1986 points out that many large companies have “occupational health professionals (occupational health nurse, occupational therapist, physiotherapist or occupational physician) available to the workplace”. The conventional role for such professionals is to deal with health problems that occur at or are caused by work. Their role is seen to include both a preventative and a

rehabilitation component. If the concepts developed above concerning the management of Physiological Activity-related Pain are applied to health professionals working within the work force the need for a new role becomes apparent.

If society comes to accept that there is an inherent limitation in each individual's tolerance to activity and that measures taken to maximise the occupational activity tolerance limit (fitness for the occupational tasks) would permit greater productivity, while minimising the risk of developing pain at work, then it might seem reasonable to employ appropriate health professionals to take on the responsibility of ensuring that these measures are undertaken. This could either be done by asking those health professionals already operating in the occupational setting to take on this role or else by employing additional personnel with appropriate skills.

As Physiological Activity-related Pain would not be seen to be a medical condition but rather a normal expected part of occupational activity, the work of the professional undertaking this role would be seen as an integral part of the employment framework and not as a separate medical responsibility. The presence of intermittent minor aches and pains would be seen as a normal part of occupational activity. The role of this new profession would be to monitor, advise and train employers and employees so that the maximum productivity could be achieved for the employer, with the maximum satisfaction and minimum discomfort for the employee. A name of this new professional role might be "Occupational Trainer" as an analogy with a similar role for Sports Trainers in Sport. The professional undertaking this role would draw on knowledge from a number of current professions including sports training, sports psychology, physiotherapy, occupational therapy and ergonomics. The important difference between the Occupational Trainer and other roles performed by occupational health professionals would be the direction in which the knowledge would be used. The goal of the Occupational Trainer would be both the maintenance of health and well being and the maximisation of productivity within the work force, not the prevention or treatment of disease. Thus the management of this condition would be largely "de-medicalised".

The role of the medical professionals external to this process would be to assess individuals who develop significant persistent discomfort and thus to discriminate between cases due to a pathological condition and those whose symptoms can be attributed to the hypothesised normal physiological process. The later could then be reassured that their problem was not medical in nature and they could then be referred back to an appropriate Occupational Trainer in the occupational setting. It would then be the responsibility of the Occupational Trainer to refer back to the medical diagnostician for reassessment of those whose recovery did not progress as expected. It would be at this stage that obscure causes of occupationally related pain, including psychological causes, not detected at the primary assessment, would have to be thoroughly investigated.

6.5.5. A Final Medico-Legal Dilemma

Controversy may arise from this scenario when a worker, in whom no specific diagnosis can be found, is still unable to tolerate the tasks required although given an ideal working environment and when everything possible has been done to maximise fitness for the job. Given the normal distribution of physical characteristics within any population this situation is bound to occur. Will it be the responsibility of that individual, the employer, or society to find an alternative occupation? This decision will have to be made by the legislators of each society as the problem arises.

6.6. Conclusion

The “alternative theory” developed in this thesis suggests that activity-related discomfort can be seen as indicating that excessive activity is occurring. The goal of physical training programs is to achieve improvements in performance and stamina for the tasks to which they are directed. The concept of performance and stamina is encapsulated in the concept of fitness, this is to say that the goal of physical training programs is to increase fitness. In the context of training for a particular activity, the experience of discomfort associated with the activity can therefore be seen as an imbalance between the effectiveness of the training to increase fitness and the intensity of the activity for which the training is being undertaken. If the intensity of this activity exceeds the current tolerance level for a significant period before the level of fitness required for that intensity of activity has been achieved, then discomfort would be expected to follow. Thus one aspect of maximising the effectiveness of training must be to select an intensity close to but not over an individual's tolerance limit.

By extrapolating this principle from the context of a training program to everyday life it can be said that a minor level of discomfort should not be seen as an experience to be feared but rather it should be seen as a helpful mechanism, useful in guiding the approach to daily activities, indicating the pace at which new activities should be introduced and warning of the development of bad habits or decreasing fitness. Clearly activities should be adjusted so that the level of discomfort remains at a tolerable level and indeed in a stable occupational setting should ideally be absent. If however such discomfort is completely ignored and measures to control it are not introduced, then increased levels of discomfort to the point of severe pain and in extreme cases the development of pathological changes, are to be expected. Thus, the syllogism of “No pain, no gain” can be seen to have some validity but only to a limited extent and only with some understanding of the implications of the potential problems involved. Perhaps an addition should be made to this syllogism as follows, “No pain, no gain: Too much pain, again no gain”. Further research will be required to define the middle path to optimum performance, avoiding inactivity and consequent failure to achieve on the one hand and on the other hand, damage due to truly excessive physical overuse.

For the musicians at any stage of their career, student or professional, the goal is always to be striving for better performance. Thus, the common aim of musicians can be seen to be the same as that of athletes, which is to continually work towards maximising the ability to perform the specific task which has been chosen. The experience of minor levels of discomfort when performing should neither be feared nor ignored. Rather such experiences should be expected and assessed with close scrutiny by the musicians themselves and if appropriate by their teachers when they occur. At such times measures should be undertaken to modify the practice (training) program in order to maximise its effectiveness in attaining increased fitness. Possible measures to achieve this would include reconsideration of technique, general aerobic fitness as well as local muscle fitness and strength and psychological attitude. A temporary decrease in the intensity of the practice program might be all that would be required to allow the level of fitness to catch up with the demands of the tasks involved. If all possible steps to balance intensity of activity with fitness have been undertaken and discomfort persists then medical assessment should be sought.

In summary, the findings of this study have suggested a new theory to help explain the origin of activity-related pain. It proposes an, as yet, unrecognised physiological process (the Physiological Activity Limitation Process). When the intensity of an activity is greater than that appropriate for an individual's level of fitness for that activity, this process generates unpleasant sensations, Physiological Activity-related Pain, the function of which is to limit that activity. This "alternative theory" provides not only an explanation for the commonly seen cases of Non-specific Occupational Overuse Syndrome where symptoms do respond to appropriate management, but can also provide reasons why some cases prove refractory to treatment. In addition, it suggests a possible aetiology for a number of other activity-related conditions associated with Non-specific Occupational Overuse Syndrome which have not as yet received generally accepted explanations. These include Fibromyalgia (Fibrositis), Myofascial Pain Syndrome, Reflex Sympathetic Dystrophy and Focal Dystonia.

Appendix A
Routine Health Check Proforma
 Page 2.

FAMILY HISTORY

Member	Occupation	Present Age or, Age at Death	Location	Present State of Health (Place X if Dead) State Cause & Years since death	Major Illness
Father			Home/Div./Sep.		
Mother			Home/Div./Sep.		
B			At Home/Left Home		
R			At Home/Left Home		
O			At Home/Left Home		
T			At Home/Left Home		
H.			At Home/Left Home		
/			At Home/Left Home		
S			At Home/Left Home		
I			At Home/Left Home		
S			At Home/Left Home		
T.			At Home/Left Home		

PERSONAL HISTORY

1. Indicate thus [X] if you have ever suffered from:

- | | |
|-----------------------------|-------------------------------|
| Eye defect/wear glasses [] | Bone/Joint injury [] |
| Ear defect/deafness [] | Hernia (rupture) [] |
| Nose or throat trouble [] | Rheumatism/Back trouble [] |
| Chronic cough/asthma [] | Fainting Epilepsy [] |
| Tuberculosis [] | Migraine [] |
| Other lung complaint [] | Stomach pain or ulcer [] |
| High blood pressure [] | Other Abdominal complaint [] |
| Heart trouble [] | Kidney or bladder trouble [] |
| Rheumatic fever [] | Sexually trans. disease [] |
| Chest pain [] | Genetic disorder [] |
| Diabetes/Endocrine dis. [] | Skin complaint [] |
| Cancer or tumour [] | Operations [] |
| Mental/nervous disorder [] | Malaria/Tropical diseases [] |
| Other [] | |

Details.....

2. Do you use: (Indicate "YES" with an [X])

	Amount	Years of use	Years Since Use
Alcohol []
Tobacco []
Other []

3. Are you currently using any medication? YES/NO
 Specify.....
 Contraception.....

4. Are you allergic to anything? YES/NO Specify.....

5. Have you a Medic Alert Bracelet? YES/NO Number.....

6. Name of usual General Practitioner.....

Appendix A
Routine Health Check Proforma
Page 4.

GENERAL MEDICAL EXAMINATION

1. GENERAL

Weight:Kg Height:cm Chest:.....cm Exp:cm Abd:.....cm

Urinalysis:

Vision: R6/..... L6..... Colour Vision.....

(Cor.) R6/..... L6/..... Squint.....

Air flow:

Pulse: B/P (Indicate) Stand/Sit/Lie.....

Menses:

2. SYSTEMS

Indicate Abnormalities (X) OR Normalities (/) in the following. If not tested leave blank ():

- | | | | | | |
|---------------|-----|-----------------|-----|-----------|-----|
| Eyes | () | Heart | () | Breasts | () |
| Ears/Hearing | () | Peripheral circ | () | Endocrine | () |
| Nose & Throat | () | Lymphatics | () | Reflexes | () |
| Teeth | () | Abdomen | () | C.N.S. | () |
| Lungs | () | Genito/Urinary | () | Skeletal | () |
| | | | | Skin | () |

Detail and comment on above abnormalities or any condition likely to affect academic performance or that requires special consideration during term or for examinations:

P.N.

3. INVESTIGATIONS

- Audiogram required for hearing defect or those exposed to noise at work
- Chest/spinal/other X-ray if indicated
- E.C.G. if indicated or if greater than 40 years

Signature of Doctor

Date

Appendix B
 Music Student Health Check Proforma
History
 Page 1.

CONFIDENTIAL

HEALTH SERVICE

STUDENT ROLL NO.

(Music Student Health Check)

Surname... (BLOCK LETTERS) Other Names.....

Home Address..... Postcode..... 'Phone.....

Term Address..... Postcode..... 'Phone.....

Change of Address..... Postcode..... 'Phone.....

Date of Birth.../.../19.... Sex: Male/Female Married/Single/Other

Country of Birth-Of Parents:(fa).....(mo).....Of Self:.....

Nationality.....Years in Australia.....
 (if other than country of birth)

Category: (Circle) Under/Post-graduate Year of Course: 1/2/3/4/5

Full/Part-Time Faculty/Department(s).....

THIS SECTION TO BE COMPLETED BY DOCTOR

SUMMARY

ALLERGY
ALERT

PROBLEM LIST

Date	No.	Problem	Code	Management	Date Resolved

CAO:JC:0312Z.mus

Appendix B
 Music Student Health Check Proforma
History
 Page 2.

FAMILY HISTORY

Member	Occupation	Present Age or, Age at Death	Location	Present State of Health (Place X if Dead) State Cause & Years since death	Major Illness
Father			Home/Div./Sep.		
Mother			Home/Div./Sep.		
BROTHER/SIST.			At Home/Left Home		
			At Home/Left Home		
			At Home/Left Home		
			At Home/Left Home		
			At Home/Left Home		

PERSONAL HISTORY

1. Indicate thus [X] if you do or ever have suffered from:

- | | |
|---------------------------------|--|
| Eye defect/wear glasses [] | Bone/Joint injury [] |
| Ear defect/deafness [] | Hernia (rupture) [] |
| Ringing in the ears [] | Rheumatism [] |
| Nose or throat trouble [] | Fainting [] |
| Chronic cough/asthma [] | Epilepsy [] |
| Tuberculosis [] | Migraine [] |
| Other lung complaint [] | Stomach pain or ulcer [] |
| High blood pressure [] | Other Abdominal complaint [] |
| Heart trouble [] | Kidney or bladder trouble [] |
| Rheumatic fever [] | Sexually trans. disease [] |
| Chest pain [] | Genetic disorder [] |
| Diabetes/Endocrine dis. [] | Skin complaint [] |
| Cancer or tumour [] | Operations [] |
| Mental/nervous disorder [] | Malaria/Tropical diseases [] |
| Trouble with your: Back [] | Neck [] Shoulders [] Arms [] |
| Wrists [] | Hands [] Legs [] Feet [] Embouchure [] |
| Any other medical complaint [] | |

Details.....

IMMUNIZATION

Have you been immunized against the following?

	DATE		DATE	OTHER	DATE
Diphtheria		Rubella			
Whoopingcough		Oral Polio			
Tetanus		BCG (T.B.)			

**Appendix B
Music Student Health Check Proforma**

**History
Page 3.**

PERSONAL HISTORY (cont.)

2. Are you allergic to anything? YES/NO Specify.....
3. Have you a Medic Alert Bracelet? YES/NO Number.....
4. Have you ever used? (Indicate "YES" with an [X])
- | | Frequency | Amount | Years of use | Years since use |
|---------------|-----------|--------|--------------|-----------------|
| Alcohol | [] | | | |
| Tobacco | [] | | | |
| Tea/Coffee | [] | | | |
| Beta Blockers | [] | | | |
| Tranquilizers | [] | | | |
| Other | [] | | | |
5. Are you currently using any prescribed medication? YES/NO
Specify.....
Contraception.....
6. Have you suffered any major upsets within the past 6 months? YES/NO
If so please state number of major upsets.
7. Name of usual General Practitioner.....

EXTRACURRICULAR ACTIVITIES

Specify type of hobby, sport, fitness activity, hand-craft, electronic/computer game, reading, relaxation technique, other

Type of Activity	hours/week during 1st term	hours/week during 2nd term	hours/week during 3rd term	hours/week during exams	hours/week during holidays

JOB

- Are you employed or doing volunteer work during the term? YES/NO
If so, number of hours/week
nature of work
- What is your final goal in music. (Indicate with an [X])
Musicologist [] Classroom Teacher [] Instrumental Teacher []
Performer [] Composer [] Other (specify)

FINANCE

- Do you have financial worries? YES/NO Do you receive TEAS? YES/NO
Are you supporting anyone? YES/NO Specify.....

ACCOMMODATION

- Do you live: in your parents home/own home/flat/college/lodgings/elsewhere?
Does this provide you with a satisfactory place to study? YES/NO
Does this provide you with a satisfactory place to practice? YES/NO

Appendix B
Music Student Health Check Proforma

History
Page 4.

STUDY

Secondary education: High School/ College/ Community College

Matriculation: First attempt 19.... Total Marks []

Second attempt 19.... Total Marks []

Are you satisfied with your university results to date? YES/FAIR/NO

Are you enrolled in the course of your first choice? YES/NO

IF NO, are you now content? YES/NO/UNSURE

IF NO, proposed action.....

Do you hold any tertiary qualifications? YES/NO

If YES specify.....

How many hours per day do you study(excluding music practise)?

Do you often study after midnight? YES/NO

How many hours per week, on average, do you listen to or play very loud music?

PRACTICAL STUDIES

INSTRUMENT(s) (name)	Principal	Second
Age when instrument was first studied		
Previous exams passed i.e. AMEB, Matric		
REGULAR PRACTICAL ACTIVITIES		
No. of days practise per week		
Total no. of hours practise per day		
Max. time of practise without a break		
No. of breaks during practise		
Length of breaks during practise		
No. of hours Lessons per week		
No. of Rehearsals per week		
Total duration of Rehearsals per week		
Total no. of performances per year		
PRACTICAL ASSESSMENT		
No. of Masterclasses per year		
No. of Recitals per year		
No. of Practical Exams per year		

Signature of Student

Date

Appendix C
Music Student Health Check Proforma
Examination
Page 1.

CONFIDENTIAL

HEALTH SERVICE

NAME

(Music Student Health Check)

STUDENT ROLL NO......

THIS SECTION TO BE COMPLETED BY SISTER

	DATE	B. C. G.	DATE	OTHER	DATE
MANTOUX					
TETANUS		Booster		ORAL POLIO	
1		Booster		1	
2		Booster		2	
3		Booster		3	

SYSTEMS EXAMINATION (This section to be completed by Doctor)

Indicate Abnormalities (X) OR Normalities (/) in the following. If not tested leave blank ():

- | | | | | | | | |
|---------------|-----|-----------------|-----|----------------|-----|----------|-----|
| Eyes | () | Lungs | () | Abdomen | () | Reflexes | () |
| Ears/Hearing | () | Heart | () | Genito/Urinary | () | C. N. S. | () |
| Nose & Throat | () | Peripheral circ | () | Breasts | () | Skin | () |
| Teeth | () | Lymphatics | () | Endocrine | () | Posture | () |

Musculo-skeletal	ROM		Power		Abnormality i.e.	
	right	left	right	left	Swelling/Tenderness	Deformity
Face						
Neck						
Back						
Shoulders						
Upper-arm						
Elbow						
Fore-arm						
Wrist						
Hand						
Hips						
Thigh						
Knee						
Calf						
Ankle						
Foot						

Appendix C
Music Student Health Check Proforma
Examination
- Page 2.

<p>LEFT HAND</p>	<p>RIGHT HAND</p> <p>Hand Span Measurement</p>
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Appendix C
Music Student Health Check Proforma
Examination
Page 3.

Grip Strength Assessment

NAME

DATE

S.R.N

DOMINANCE

VIGORIMETER

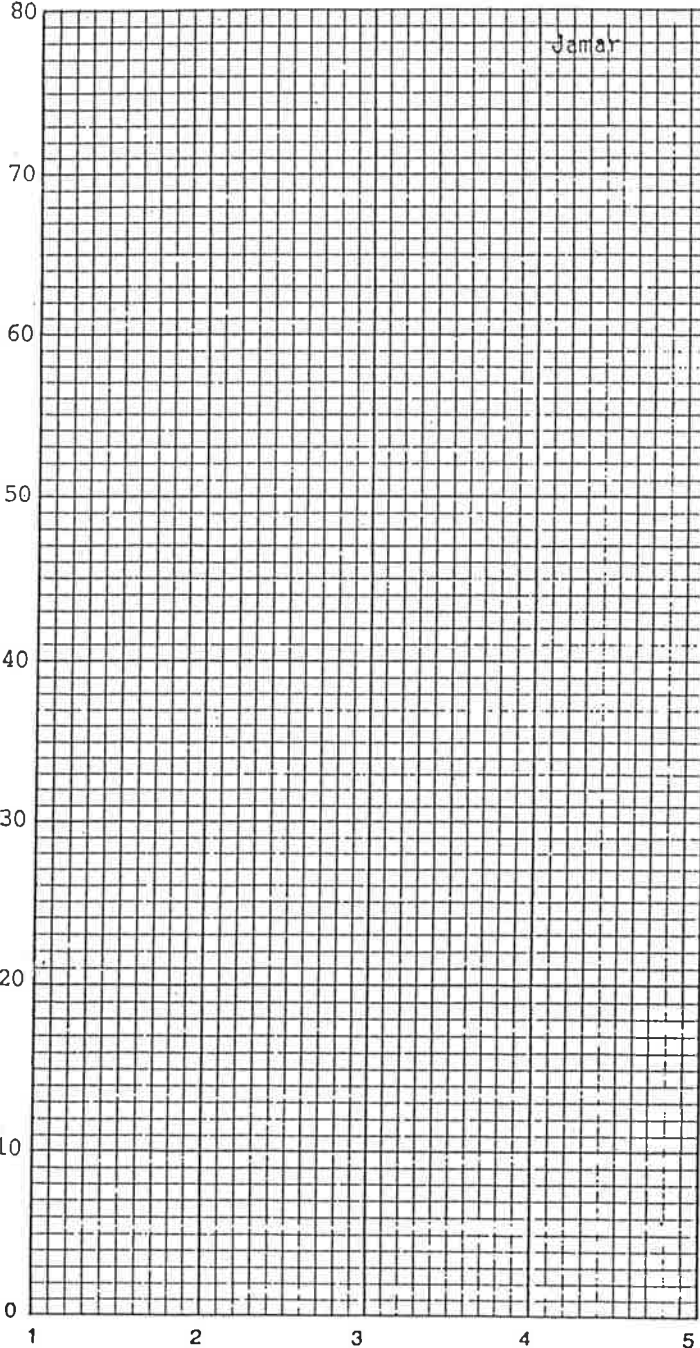
R-Ped (o)
L-Blue (x)

R

L

Pinch

Kg



KPa

Cooperation 1 2 3

Observation during test

Interpretation:

Appendix C
Music Student Health Check Proforma
Examination
Page 4.

PHYSIOLOGICAL MEASUREMENTS

Pulse: B/P: (Stand/Sit/Lie) Weight:Kg Height:cm
 Chest: inspir ...cm expir ...cm Abdo:cm Hand Span: R ...mm L ...mm
 Peak Exp Flow Rate Vital Capacity

Urinalysis:

Vision: R6/..... L6..... Colour Vision.....
 (Cor.) R6/..... L6/..... Squint.....

Grip Strength: (details see separate sheet)
 Total Dynamometer Reading right left
 Max. Vigorometer Pinch Reading right left

McArdle Step Test: Duration of Effortsec (if less than 3 mins)
 No. of heart beats from 5-20 secs post effort
 Estimate V O₂ Max.(ml/kg/min) percentile

Dexterity Test: Maximal Tap Rate Index-finger right left
 (beats/minute) Ring- finger right left
 Wrist right left

Menses:

Detail and comment on above abnormalities or any condition likely to affect academic performance or that requires special consideration during term or for examinations:

P.N. |

|

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|

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3. INVESTIGATIONS

Audiogram required for hearing defect or those exposed to noise at work
 Chest/spinal/other X-ray if indicated

E.C.G. if indicated or if greater than 40 years

Other

Signature of Doctor

Date

Appendix D
 Psychological Self Assessment Questionnaire
The Illness Behaviour Questionnaire
 Page 1.

Health Attitude Questionnaire

Instructions.

On the following pages you will find a number of questions about your health and how it affects you. It is important that you complete every question even though some of them may not be directly applicable to you. Circle either YES or NO to indicate your answer to each question.

- | | | |
|---|-----|----|
| 1. Do you worry a lot about your health? | YES | NO |
| 2. Do you think there is something seriously wrong with your body? | YES | NO |
| 3. Do you have an illness which interferes with your life a great deal? | YES | NO |
| 4. Are you easy to get on with when you are ill? | YES | NO |
| 5. Does your family have a history of illness? | YES | NO |
| 6. Do you think you are more liable to illness than other people? | YES | NO |
| 7. If a doctor told you that he could find nothing wrong with you would you believe him or her? | YES | NO |
| 8. Is it easy for you to forget about yourself and think about all sorts of things? | YES | NO |
| 9. If you feel ill and someone tells you that you are looking better, do you become annoyed? | YES | NO |
| 10. Do you find that you are often aware of various things happening in your body? | YES | NO |
| 11. Do you ever think that you have an illness which is a punishment for something you have done wrong in the past? | YES | NO |
| 12. Do you have trouble with your nerves? | YES | NO |
| 13. If you feel ill or worried can you be easily cheered up by the doctor? | YES | NO |
| 14. Do you think that other people realise what it's like to be sick? | YES | NO |
| 15. Does it upset you to talk to a doctor about an illness? | YES | NO |
| 16. Are you bothered by many aches or pains? | YES | NO |
| 17. Do you have an illness which affects the way you get on with your family or friends a great deal? | YES | NO |
| 18. Do you find that you get anxious easily? | YES | NO |
| 19. Do you have an illness which is the same as anybody you know has had? | YES | NO |
| 20. Are you more sensitive to pain than other people? | YES | NO |
| 21. Are you afraid of illness? | YES | NO |
| 22. Can you express your personal feelings easily to other people? | YES | NO |
| 23. Do people feel sorry for you when you are ill? | YES | NO |
| 24. Do you think that you worry about your health more than most people? | YES | NO |
| 25. Do you have an illness which affects your sexual relations? | YES | NO |
| 26. Do you have an illness with a lot of pain? | YES | NO |
| 27. Except for illness, do you have any problems in your life? | YES | NO |
| 28. Do you care whether or not people realise when you are ill? | YES | NO |
| 29. Do you find that you get jealous of other people's good health? | YES | NO |

Appendix D
 Psychological Self Assessment Questionnaire
The Illness Behaviour Questionnaire
 Page 2.

30.	Do you ever have silly thoughts about your health which you can't get out of your mind, no matter how hard you try?	YES	NO
31.	Do you have any financial problems?	YES	NO
32.	Are you upset by the way people take your illness when you are sick?	YES	NO
33.	Is it hard for you to believe a doctor when he or she tells you there is nothing for you to worry about?	YES	NO
34.	Do you often worry about the possibility that you have got a serious disease?	YES	NO
35.	Are you sleeping well?	YES	NO
36.	When you are angry do you tend to bottle up your feelings?	YES	NO
37.	Do you often think that you might suddenly fall ill?	YES	NO
38.	If a disease is brought to your attention (through the radio, television, newspapers or someone you know) do you worry about getting it yourself?	YES	NO
39.	Do you get the feeling that people are not taking your illness seriously enough when you are sick?	YES	NO
40.	Are you upset by the appearance of your face or body?	YES	NO
41.	Do you find that you are bothered by many different symptoms?	YES	NO
42.	Do you frequently try to explain to others how you are feeling?	YES	NO
43.	Do you have any family problems?	YES	NO
44.	Do you think there is something the matter with your mind?	YES	NO
45.	Are you eating well?	YES	NO
46.	Is bad health the biggest difficulty in your life?	YES	NO
47.	Do you find that you get sad easily?	YES	NO
48.	Do you worry or fuss over small details that seem unimportant to others?	YES	NO
49.	Are you always a co-operative patient?	YES	NO
50.	Do you often have the symptoms of a serious disease?	YES	NO
51.	Do you find that you get angry easily?	YES	NO
52.	Do you have any work problems?	YES	NO
53.	Do you prefer to keep your feelings to yourself?	YES	NO
54.	Do you often find that you get depressed?	YES	NO
55.	Would all your worries be over if you were physically healthy?	YES	NO
56.	Are you more irritable towards other people?	YES	NO
57.	Do you have symptoms which may be caused by worry?	YES	NO
58.	Is it easy for you to let people know when you are cross with them?	YES	NO
59.	Is it hard for you to relax?	YES	NO
60.	Do you have personal worries which are not caused by physical illness?	YES	NO
61.	Do you often find that you lose patience with other people?	YES	NO
62.	Is it hard for you to show people your personal feelings?	YES	NO

Appendix E
 Psychological Self Assessment Questionnaire
The State-Trait Anxiety Inventory
 Page 1.

Music Student Health Check Questionnaires

Student Roll No.

Anxiety Questionnaire

Date.

Instructions

Read each statement and then tick to indicate how you GENERALLY FEEL. There are no right or wrong answers. Do not spend too much time on any one statement But give the answer which seems to best describe how you GENERALLY FEEL.

	ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
1. I feel pleasant.				
2. I tire quickly.				
3. I feel like crying.				
4. I wish I could be as happy as others seem to be.				
5. I am losing out on things because I can't make up my mind soon enough				
6. I feel rested.				
7. I am calm, cool and collected.				
8. I feel that difficulties are piling up so that I can not overcome them.				
9. I worry too much over something that doesn't matter.				
10. I am happy.				
11. I am inclined to take things hard.				
12. I lack self confidence.				
13. I feel secure.				
14. I try to avoid facing a crisis or difficulty.				
15. I feel blue.				
16. I am content.				
17. Some unimportant thought runs through my mind and bothers me.				
18. I take disappointments so keenly that I can't put them out of my mind.				
19. I am a steady person.				
20. I get into a state of tension or turmoil as I think over my recent concerns and interests.				

Appendix F
 Psychological Self Assessment Questionnaire
The General Health Questionnaire
 Page 1.

UNIVERSITY OF ADELAIDE.
 STUDENT HEALTH SERVICE.
GENERAL HEALTH QUESTIONNAIRE

Name.

Date.

Research No.

University No.

Please read this carefully:

We should like to know if you have had any medical complaints, and how your health has been in general, over the past few weeks. Please answer ALL the questions on the following pages simply by underlining the answer which you think most nearly applies to you. Remember that we want to know about present and recent complaints, not those that you had in the past.

It is important that you try to answer ALL the questions.

Thank you very much for your co-operation.

HAVE YOU RECENTLY:-

- | | | | | |
|---|-------------------|--------------------|------------------------|-----------------------|
| 1. - been feeling perfectly well and in good health? | Better than usual | Same as usual | Worse than usual | Much worse than usual |
| 2. - been feeling in need of a good tonic? | Not at all | No more than usual | Rather more than usual | Much more than usual |
| 3. - been feeling run down and out of sorts? | Not at all | No more than usual | Rather more than usual | Much more than usual |
| 4. - felt that you are ill? | Not at all | No more than usual | Rather more than usual | Much more than usual |
| 5. - been getting any pains in your head? | Not at all | No more than usual | Rather more than usual | Much more than usual |
| 6. - been getting a feeling of tightness or pressure in your head? | Not at all | No more than usual | Rather more than usual | Much more than usual |
| 7. - been able to concentrate on whatever you're doing? | Better than usual | Same as usual | Less than usual | Much less than usual |
| 8. - been afraid that you were going to collapse in a public place? | Not at all | No more than usual | Rather more than usual | Much more than usual |
| 9. - been having hot or cold spells? | Not at all | No more than usual | Rather more than usual | Much more than usual |
| 10. - been perspiring (sweating) a lot? | Not at all | No more than usual | Rather more than usual | Much more than usual |

PLEASE TURN OVER

Appendix F
 Psychological Self Assessment Questionnaire
The General Health Questionnaire
 Page 2.

HAVE YOU RECENTLY:-

11. - found yourself waking early and unable to get back to sleep?	Not at all	No more than usual	Rather more than usual	Much more than usual
12. - been getting up feeling your sleep hasn't refreshed you?	Not at all	No more than usual	Rather more than usual	Much more than usual
13. - been feeling too tired and exhausted even to eat?	Not at all	No more than usual	Rather more than usual	Much more than usual
14. - lost much sleep over worry?	Not at all	No more than usual	Rather more than usual	Much more than usual
15. - been feeling mentally alert and wide awake?	Better than usual	Same as usual	Less alert than usual	Much less alert
16. - been feeling full of energy?	Better than usual	Same as usual	Less energy than usual	Much less energetic
17. - had difficulty in getting off to sleep?	Not at all	No more than usual	Rather more than usual	Much more than usual
18. - had difficulty in staying asleep once you are off?	Not at all	No more than usual	Rather more than usual	Much more than usual
19. - been having frightening or unpleasant dreams?	Not at all	No more than usual	Rather more than usual	Much more than usual
20. - been having restless, disturbed nights?	Not at all	No more than usual	Rather more than usual	Much more than usual
21. - been managing to keep yourself busy and occupied?	More so than usual	Same as usual	Rather less than usual	Much less than usual
22. - been taking longer over the things you do?	Quicker than usual	Same as usual	Longer than usual	Much longer than usual
23. - tended to lose interest in your ordinary activities?	Not at all	No more than usual	Rather more than usual	Much more than usual
24. - been losing interest in your personal appearance?	Not at all	No more than usual	Rather more than usual	Much more than usual
25. - been taking less trouble with your clothes?	More trouble than usual	About same as usual	Less trouble than usual	Much less trouble
26. - been getting out of the house as much as usual?	More than usual	Same as usual	Less than usual	Much less than usual
27. - been managing as well as most people would in your shoes?	Better than most	About the same	Rather less well	Much less well
28. - felt on the whole you were doing things well?	Better than usual	About the same	Less well than usual	Much less well

Appendix F
 Psychological Self Assessment Questionnaire
The General Health Questionnaire
 Page 3.

HAVE YOU RECENTLY:-

29. - been late getting to work, or getting started on your housework?	Not at all	No later than usual	Rather later than usual	Much later than usual
30. - been satisfied with the way you've carried out your task?	More satisfied	About same as usual	Less satisfied than usual	Much less satisfied
31. - been able to feel warmth and affection for those near to you?	Better than usual	About same as usual	Less well than usual	Much less well
32. - been finding it easy to get on with other people?	Better than usual	About same as usual	Less well than usual	Much less well
33. - spent much time chatting with people?	More time than usual	About same as usual	Less than usual	Much less than usual
34. - kept feeling afraid to say anything to people in case you made a fool of yourself?	Not at all	No more than usual	Rather more than usual	Much more than usual
35. - felt that you are playing a useful part in things?	More so than usual	Same as usual	Less useful than usual	Much less useful
36. - felt capable of making decisions about things?	More so than usual	Same as usual	Less so than usual	Much less capable
37. - felt you're just not able to make a start on anything?	Not at all	No more than usual	Rather more than usual	Much more than usual
38. - felt yourself dreading everything that you have to do?	Not at all	No more than usual	Rather more than usual	Much more than usual
39. - felt constantly under strain?	Not at all	No more than usual	Rather more than usual	Much more than usual
40. - felt you couldn't overcome your difficulties?	Not at all	No more than usual	Rather more than usual	Much more than usual
41. - been finding life a struggle all the time?	Not at all	No more than usual	Rather more than usual	Much more than usual
42. - been able to enjoy your normal day-to-day activities?	More so than usual	Same as usual	Less so than usual	Much less than usual
43. - been taking things hard?	Not at all	No more than usual	Rather more than usual	Much more than usual
44. - been getting edgy and bad-tempered?	Not at all	No more than usual	Rather more than usual	Much more than usual
45. - been getting scared or panicky for no good reason?	Not at all	No more than usual	Rather more than usual	Much more than usual
46. - been able to face up to your problems?	More so than usual	Same as usual	Less able than usual	Much less able

PLEASE TURN OVER

Appendix F
 Psychological Self Assessment Questionnaire
The General Health Questionnaire
 Page 4.

HAVE YOU RECENTLY:-

47. - found everything getting on top of you?	Not at all	No more than usual	Rather more than usual	Much more than usual
48. - had the feeling that people were looking at you?	Not at all	No more than usual	Rather more than usual	Much more than usual
49. - been feeling unhappy and depressed?	Not at all	No more than usual	Rather more than usual	Much more than usual
50. - been losing confidence in yourself?	Not at all	No more than usual	Rather more than usual	Much more than usual
51. - been thinking of yourself as a worthless person?	Not at all	No more than usual	Rather more than usual	Much more than usual
52. - felt that life is entirely hopeless?	Not at all	No more than usual	Rather more than usual	Much more than usual
53. - been feeling hopeful about your own future?	More so than usual	About same as usual	Less so than usual	Much less hopeful
54. - been feeling reasonably happy, all things considered?	More so than usual	About same as usual	Less so than usual	Much less than usual
55. - been feeling nervous and strung-up all the time?	Not at all	No more than usual	Rather more than usual	Much more than usual
56. - felt that life isn't worth living?	Not at all	No more than usual	Rather more than usual	Much more than usual
57. - thought of the possibility that you might make away with yourself?	Definitely not	I don't think so	Has crossed my mind	Definitely have
58. - found at times you couldn't do anything because your nerves were too bad?	Not at all	No more than usual	Rather more than usual	Much more than usual
59. - found yourself wishing you were dead and away from it all?	Not at all	No more than usual	Rather more than usual	Much more than usual
60. - found that the idea of taking your own life kept coming into your mind?	Definitely not	I don't think so	Has crossed my mind	Definitely has

Appendix G. Statistical Tables ; Supplementary details of results from Chapter 4.

Demographic characteristics and differences between Study Samples

Table 1.

The occupations of the individuals in the three study samples are listed below

**Frequency Distribution for Occupation
Split By: Study Sample**

	Total Count	Sample1 Count	Sample2 Count	Sample3 Count
Inst_Music_Student	112	93	0	19
Vocal_Music_Student	2	0	2	0
Dentistry_Student	4	0	4	0
Law_Student	7	0	5	2
Maths_Student	7	0	7	0
Arts_Student	12	0	11	1
Science_Student	11	0	11	0
Engineering_Student	7	0	7	0
Economics_Student	3	0	3	0
Geology_Student	1	0	1	0
Medical_Student	2	0	1	1
Psychology_Student	1	0	1	0
Music_Teacher	3	0	0	3
Pianist	1	0	0	1
High_School_Student	1	0	0	1
University_Lecturer	1	0	0	1
Total	175	93	53	29

Table 2.

Within the Instrumental Music Students' group, Sample 1, the following table lists the principal instrument played.

Principal Instrument	Number
Piano	47
Guitar	8
Violin	8
Flute	7
Clarinet	6
Trumpet	4
Cello	2
Oboe	2
Organ	2
Trombone	2
Viola	1
Bassoon	1
Double Bass	1
Piccolo	1
Percussion	1

Table 3.

Of the 93 Instrumental Music Students, 45 played a second instrument.

Second Instrument	Number
Piano	13
Violin	10
Flute	5
Percussion	3
Clarinet	3
Cello	2
Recorder	2
Double Bass	1
Viola	1
Oboe	1
Lute	1
Trombone	1
Piccolo	1
Saxophone	1

Table 4.

Patient group, Sample 3, The following table lists the principal instrument played.

(Unusual instruments are grouped as "Other" to preserve anonymity).

Principal Instrument	Number
Piano	15
Violin	2
Saxophone	2
Guitar	1
Oboe	1
Bassoon	1
Double Bass	1
Percussion	1
Other	1
None	4

Table 5.

Patient group, Sample 3, 10 played a second instrument.

Second Instrument	Number
Piano	3
Flute	2
Violin	1
Recorder	1
Cello	1
Double Bass	1
Guitar	1

Table 6.

Age at time of Initial Assessment.

Descriptive Statistics
Split By: Study Sample

	Mean	Std. Dev.	Std. Error	Count	Minimum	Maximum
Age Assessed, Total	20.653	5.172	.391	175	14.767	45.049
Age Assessed, Sample1	19.443	3.247	.337	93	17.014	39.707
Age Assessed, Sample2	20.612	5.364	.737	53	16.899	45.049
Age Assessed, Sample3	24.607	7.557	1.403	29	14.767	43.649

Table 7.

The difference between the mean ages of the different study samples was compared with Analysis of Variance.

ANOVA Table for Age Assessed

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Study Sample	2	589.733	294.867	12.476	<.0001
Residual	172	4065.134	23.634		

Model II estimate of between component variance: 5.18

Fisher's PLSD Post-hoc test of between groups differences.

Fisher's PLSD for Age Assessed

Effect: Study Sample

Significance Level: 5 %

	Mean Diff.	Crit. Diff	P-Value	
Sample1, Sample2	-1.169	1.652	.1641	
Sample1, Sample3	-5.164	2.041	<.0001	S
Sample2, Sample3	-3.995	2.216	.0005	S

Table 8.

Contingency table analysis comparing sex ratio of Sample 1 with Sample 2.

Observed Frequencies

	Sample1	Sample2	Totals
F	63	25	88
M	30	28	58
Totals	93	53	146

Percents of Column Totals

	Sample1	Sample2	Totals
F	67.742	47.170	60.274
M	32.258	52.830	39.726
Totals	100.000	100.000	100.000

Summary Table

Num. Missing	0
DF	1
Chi Square	5.967
Chi Square P-Value	.0146
G-Squared	5.930
G-Squared P-Value	.0149
Contingency Coef.	.198
Phi	.202
Cty. Cor. Chi Square	5.139
Cty. Cor. P-Value	.0234
Fisher's Exact P-Value	.0218

Table 9.

Contingency table analysis comparing sex ratio of Sample 1 with Sample 3.

Observed Frequencies				Summary Table	
	Sample1	Sample3	Totals		
F	63	20	83	Num. Missing	0
M	30	9	39	DF	1
Totals	93	29	122	Chi Square	.015
				Chi Square P-Value	.9018
				G-Squared	.015
				G-Squared P-Value	.9016
				Contingency Coef.	.011
				Phi	.011
				Cty. Cor. Chi Square	0.000
				Cty. Cor. P-Value	>.9999
				Fisher's Exact P-Value	>.9999

Percents of Column Totals			
	Sample1	Sample3	Totals
F	67.742	68.966	68.033
M	32.258	31.034	31.967
Totals	100.000	100.000	100.000

Table 10.

Contingency table analysis comparing sex ratio of Sample 2 with Sample 3.

Observed Frequencies				Summary Table	
	Sample2	Sample3	Totals		
F	25	20	45	Num. Missing	0
M	28	9	37	DF	1
Totals	53	29	82	Chi Square	3.596
				Chi Square P-Value	.0579
				G-Squared	3.667
				G-Squared P-Value	.0555
				Contingency Coef.	.205
				Phi	.209
				Cty. Cor. Chi Square	2.770
				Cty. Cor. P-Value	.0961
				Fisher's Exact P-Value	.0673

Percents of Column Totals			
	Sample2	Sample3	Totals
F	47.170	68.966	54.878
M	52.830	31.034	45.122
Totals	100.000	100.000	100.000

Table 11.

There was no significant difference in the age of musicians vs non-musicians.

Unpaired t-test for Age Assessed
Grouping Variable: Occupational Classification
Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
Instrumental Musician, Non-musician	-.861	173	-1.032	.3036

Group Info for Age Assessed
Grouping Variable: Occupational Classification

	Count	Mean	Variance	Std. Dev.	Std. Err
Instrumental Musician	118	20.372	20.086	4.482	.413
Non-musician	57	21.233	40.650	6.376	.844

Table 12.

There was a significant difference in the age between diagnostic categories.

ANOVA Table for Age Assessed

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Initial Diagnostic Classification	2	347.814	173.907	6.945	.0013
Residual	172	4307.053	25.041		

Model II estimate of between component variance: 2.834

Means Table for Age Assessed

Effect: Initial Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	95	19.897	4.039	.414
Inconsequential	43	19.981	4.729	.721
Consequential	37	23.375	7.139	1.174

Fisher's PLSD for Age Assessed

Effect: Initial Diagnostic Classification

Significance Level: 5 %

	Mean Diff.	Crit. Diff	P-Value	
No Symptoms, Inconsequential	-.085	1.815	.9268	
No Symptoms, Consequential	-3.478	1.914	.0004	S
Inconsequential, Consequential	-3.393	2.215	.0029	S

Table 13.

Contingency table analysis comparing sex ratio of musicians with non-musicians.

Observed Frequencies				Summary Table	
	Instrumental Musician	Non-musician	Totals		
F	81	27	108	Num. Missing	0
M	37	30	67	DF	1
Totals	118	57	175	Chi Square	7.363
				Chi Square P-Value	.0067
				G-Squared	7.272
				G-Squared P-Value	.0070
				Contingency Coef.	.201
				Phi	.205
				Cty. Cor. Chi Square	6.490
				Cty. Cor. P-Value	.0108
				Fisher's Exact P-Value	.0081

Percents of Column Totals			
	Instrumental Musician	Non-musician	Totals
F	68.644	47.368	61.714
M	31.356	52.632	38.286
Totals	100.000	100.000	100.000

Table 14.

There was no significant difference in the sex ratio between diagnostic categories overall.

Observed Frequencies					Summary Table	
	No Symptoms	Inconsequential	Consequential	Totals	Num. Missing	
F	53	28	27	108	DF	0
M	42	15	10	67	Chi Square	2
Totals	95	43	37	175	Chi Square P-Value	3.607
					G-Squared	.1647
					G-Squared P-Value	3.685
					Contingency Coef.	.1584
					Cramer's V	.142
						.144

Percents of Column Totals				
	No Symptoms	Inconsequential	Consequential	Totals
F	55.789	65.116	72.973	61.714
M	44.211	34.884	27.027	38.286
Totals	100.000	100.000	100.000	100.000

Cross-sectional Analysis

Table 15.

Contingency table analysis for an effect of sex on the prevalence of upper-limb activity symptoms (analysis limited to the control group, samples 1 and 2) shows no significant effect.

Observed Frequencies					Summary Table	
	No Symptoms	Inconsequential	Consequential	Totals	Num. Missing	
F	53	28	7	88	DF	0
M	42	15	1	58	Chi Square	2
Totals	95	43	8	146	Chi Square P-Value	3.696
					G-Squared	.1576
					G-Squared P-Value	4.122
					Contingency Coef.	.1273
					Cramer's V	.157
						.159

Percents of Column Totals				
	No Symptoms	Inconsequential	Consequential	Totals
F	55.789	65.116	87.500	60.274
M	44.211	34.884	12.500	39.726
Totals	100.000	100.000	100.000	100.000

Table 16.

Prevalence of symptoms comparing Instrumental Music Students with Other Students

Observed Frequencies

	Music Student	Other Student	Totals
No Symptoms	52	43	95
Inconsequential	35	8	43
Consequential	6	2	8
Totals	93	53	146

Prevalence Estimate Is Column Percentage

	Music Student	Other Student	Totals
No Symptoms	55.914	81.132	65.068
Inconsequential	37.634	15.094	29.452
Consequential	6.452	3.774	5.479
Totals	100.000	100.000	100.000

Contingency table analysis of the prevalence of symptoms comparing Instrumental Music Students with Other Students; the "Consequential Symptoms" subgroups are excluded because of small expected cell frequencies.

Observed Frequencies

	Music Student	Other Student	Totals
No Symptoms	52	43	95
Inconsequential	35	8	43
Totals	87	51	138

Expected Values

	Music Student	Other Student	Totals
No Symptoms	59.891	35.109	95.000
Inconsequential	27.109	15.891	43.000
Totals	87.000	51.000	138.000

Summary Table

Num. Missing	0
DF	1
Chi Square	9.029
Chi Square P-Value	.0027
G-Squared	9.646
G-Squared P-Value	.0019
Contingency Coef.	.248
Phi	.256
Cty. Cor. Chi Square	7.921
Cty. Cor. P-Value	.0049
Fisher's Exact P-Value	.0040

Table 17

Contingency table analysis of the prevalence of “Inconsequential Symptoms” associated with writing comparing Instrumental Music Students with Other Students; the “Consequential Symptoms” subgroups are excluded because of small expected cell frequencies.

Observed Frequencies				Summary Table	
	Music Student	Other Student	Totals		
No Symptoms	80	45	125	Num. Missing	0
Inconsequential	12	6	18	DF	1
Totals	92	51	143	Chi Square	.049
				Chi Square P-Value	.8252
				G-Squared	.049
				G-Squared P-Value	.8245
				Contingency Coef.	.018
				Phi	.018
				Cty. Cor. Chi Square	0
				Cty. Cor. P-Value	>.9999
				Fisher's Exact P-Value	>.9999

Case-control Analysis

Table 18.

Contingency table analysis of association between recent change in activity and onset of symptoms . (only individuals with symptoms included).

Observed Frequencies				Summary Table	
	Inconsequential	Consequential	Totals		
No Change	28	17	45	Num. Missing	0
Change	15	20	35	DF	1
Totals	43	37	80	Chi Square	2.970
				Chi Square P-Value	.0848
				G-Squared	2.983
				G-Squared P-Value	.0842
				Contingency Coef.	.189
				Phi	.193
				Cty. Cor. Chi Square	2.242
				Cty. Cor. P-Value	.1343
				Fisher's Exact P-Value	.1143

Percents of Column Totals			
	Inconsequential	Consequential	Totals
No Change	65.116	45.946	56.250
Change	34.884	54.054	43.750
Totals	100.000	100.000	100.000

Table 19.

Contingency table analysis of association between recent change in activity prior to on set of symptoms and Occupational Classification. (only individuals with symptoms included).

Observed Frequencies				Summary Table	
	Musician	Non-Musician	Totals	Num. Missing	95
No Change	34	11	45	DF	1
Change	32	3	35	Chi Square	3.436
Totals	66	14	80	Chi Square P-Value	.0638
				G-Squared	3.667
				G-Squared P-Value	.0555
				Contingency Coef.	.203
				Phi	.207
				Cty. Cor. Chi Square	2.424
				Cty. Cor. P-Value	.1195
				Fisher's Exact P-Value	.0799

Percents of Column Totals			
	Musician	Non-Musician	Totals
No Change	51.515	78.571	56.250
Change	48.485	21.429	43.750
Totals	100.000	100.000	100.000

Table 20.

Two way ANOVA comparing mean "extent of symptoms" for the factors "Occupational Classification" and "Diagnostic Classification".

ANOVA Table for Number of Symptomatic Areas

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Initial Diagnostic Classification	1	1.773	1.773	.421	.5186
Occupational Classification	1	7.526	7.526	1.785	.1856
Interaction	1	.593	.593	.141	.7086
Residual	76	320.463	4.217		

Means Table for INumber of Symptomatic Areas

Effect: Initial Diagnostic Classification * Occupational Classification

	Count	Mean	Std. Dev.	Std. Err.
Inconsequential, Musician	35	2.086	1.634	.276
Inconsequential, Non-Musician	8	1.500	1.069	.378
Consequential, Musician	31	2.710	2.673	.480
Consequential, Non-Musician	6	1.667	1.211	.494

Table 21.

Simultaneous comparisons of the number of sites of tenderness found on examination were made between Occupational Categories (musicians cf non-musicians) and the different Diagnostic Categories, with a two way analysis of variance.

ANOVA Table for No. of Tender Sites

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Occupational Classification	1	.261	.261	.012	.9129
Initial Diagnostic Classification	2	3384.815	1692.408	77.731	<.0001
Interaction	2	11.666	5.833	.268	.7653
Residual	169	3679.552	21.772		

Means Table for No. of Tender Sites

Effect: Occupational Classification * Initial Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
Musician, No Symptoms	52	2.923	3.661	.508
Musician, Inconsequential	35	7.686	5.940	1.004
Musician, Consequential	31	16.129	6.702	1.204
Non-Musician, No Symptoms	43	2.000	2.225	.339
Non-Musician, Inconsequential	8	7.750	2.659	.940
Non-Musician, Consequential	6	16.667	6.186	2.525

Fisher's PLSD for No. of Tender Sites

Effect: Initial Diagnostic Classification

Significance Level: 5 %

	Mean Diff.	Crit. Diff	P-Value	
No Symptoms, Inconsequential	-5.192	1.693	<.0001	S
No Symptoms, Consequential	-13.711	1.785	<.0001	S
Inconsequential, Consequential	-8.519	2.066	<.0001	S

Table 22.

Simultaneous comparisons of the “prevalence of tenderness in symptomatic areas” were made between Occupational Categories (musicians cf non-musicians) and the different Diagnostic Categories, with a two way analysis of variance (individuals with no symptomatic areas were excluded from this analysis).

ANOVA Table for Prevalence of Tenderness in Symptomatic Areas

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Initial Diagnostic Classification	1	.420	.420	3.014	.0866
Occupational Classification	1	.236	.236	1.695	.1969
Interaction	1	.258	.258	1.850	.1778
Residual	76	10.590	.139		

Means Table for Prevalence of Tenderness in Symptomatic Areas**Effect: Initial Diagnostic Classification * Occupational Classification**

	Count	Mean	Std. Dev.	Std. Err.
Inconsequential, Musician	35	.580	.489	.083
Inconsequential, Non-Musician	8	.875	.354	.125
Consequential, Musician	31	.923	.215	.039
Consequential, Non-Musician	6	.917	.204	.083

Table 23.

Simultaneous comparisons of the “prevalence of tenderness in asymptomatic areas” were made between Occupational Categories (musicians cf non-musicians) and the different Diagnostic Categories, with a two way analysis of variance (individuals with no asymptomatic areas were excluded from this analysis).

ANOVA Table for Prevalence of Tenderness in Asymptomatic Areas

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Initial Diagnostic Classification	2	5.332	2.666	50.864	<.0001
Occupational Classification	1	.010	.010	.191	.6624
Interaction	2	.088	.044	.841	.4329
Residual	165	8.648	.052		

Means Table for Prevalence of Tenderness in Asymptomatic Areas**Effect: Initial Diagnostic Classification * Occupational Classification**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms, Musician	52	.188	.224	.031
No Symptoms, Non-Musician	43	.140	.150	.023
Inconsequential, Musician	34	.374	.291	.050
Inconsequential, Non-Musician	8	.401	.170	.060
Consequential, Musician	28	.674	.262	.049
Consequential, Non-Musician	6	.759	.248	.101

Fisher's PLSD for Prevalence of Tenderness in Asymptomatic Areas**Effect: Initial Diagnostic Classification****Significance Level: 5 %**

	Mean Diff.	Crit. Diff	P-Value	
No Symptoms, Inconsequential	-.213	.084	<.0001	S
No Symptoms, Consequential	-.523	.090	<.0001	S
Inconsequential, Consequential	-.310	.104	<.0001	S

Table 24.

The “Prevalence of tenderness in unilateral asymptomatic areas” and the “Prevalence of tenderness in asymptomatic areas in general” were compared, in each individual with at least one unilateral asymptomatic area, with the paired Student’s t-test.

Paired t-test
Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
Unilateral Asyptomatic, Asymptomatic	.248	46	4.759	<.0001

Table 25.

The “Prevalence of tenderness in unilateral asymptomatic areas” and the “Prevalence of tenderness in symptomatic areas” were compared, in each individual with at least one unilateral asymptomatic area, with the paired Student’s t-test.

Paired t-test
Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
Unilateral Asyptomatic, Symptomatic	-.074	46	-1.310	.1968

Table 26.

The “Prevalence of tenderness in unilateral asymptomatic areas” and the “Prevalence of tenderness in symptomatic areas” were compared, in each individual with at least one unilateral asymptomatic area, with the paired Student’s t-test, stratified by both Occupational and Diagnostic Classification.

Paired t-test
Split By: Occupational Classification
Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
Unilateral Asyptomatic, Symptomatic: Total	-.074	46	-1.310	.1968
Unilateral Asyptomatic, Symptomatic: Musician	-.097	35	-1.313	.1978
Unilateral Asyptomatic, Symptomatic: Non-Musician	0.000	10	.	.

Paired t-test
Split By: Initial Diagnostic Classification
Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
Unilateral Asyptomatic, Symptomatic: Total	-.074	46	-1.310	.1968
Unilateral Asyptomatic, Symptomatic: Inconsequential	-.083	23	-1.000	.3277
Unilateral Asyptomatic, Symptomatic: Consequential	-.065	22	-.826	.4175

Cohort Prospective-Analysis

Table 27

Contingency table analysis of association between “Presence of Tenderness at Initial Assessment” and “Followup Diagnostic Classification”.

Observed Frequencies					Summary Table	
	No Symptoms	Inconsequential	Consequential	Totals		
No	16	10	4	30	Num. Missing	0
Yes	22	18	12	52	DF	2
Totals	38	28	16	82	Chi Square	1.434
					Chi Square P-Value	.4883
					G-Squared	1.480
					G-Squared P-Value	.4772
					Contingency Coef.	.131
					Cramer's V	.132

Table 28

The difference in the means of the “Number of sites of tenderness at initial assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

ANOVA Table for Initial No. of Tender Sites

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	87.610	43.805	1.740	.1822
Residual	79	1988.780	25.174		

Model II estimate of between component variance: .721

Means Table for Initial No. of Tender Sites

Effect: Followup Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	38	3.711	4.392	.713
Inconsequential	28	5.964	5.840	1.104
Consequential	16	4.000	4.858	1.214

Table 29

The difference in the mean values of the "Prevalence of tenderness in asymptomatic areas at initial assessment" between the categories of "Followup Diagnostic Classification" were compared with a one-way ANOVA.

ANOVA Table for Prevalence of tenderness in asymptomatic areas

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	.092	.046	.636	.5323
Residual	78	5.656	.073		

Model II estimate of between component variance: •

Means Table for Prevalence of tenderness in asymptomatic areas**Effect: Followup Diagnostic Classification**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	38	.228	.266	.043
Inconsequential	27	.300	.290	.056
Consequential	16	.228	.238	.059

One case was omitted due to lack of asymptomatic areas.

Table 30.

Student's t-test was used to compare the differences in physical fitness parameters Aerobic Fitness as given by an estimate of V O₂ max., Grip Strength, Pinch Strength and Peak Expiratory Flow Rate (PEFR) between males and females.

Unpaired t-test for VO₂ Max.

Grouping Variable: Sex

Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
F, M	-16.954	75	-12.671	<.0001

Group Info for VO₂ Max.

Grouping Variable: Sex

	Count	Mean	Variance	Std. Dev.	Std. Err
F	49	37.736	10.234	3.199	.457
M	28	54.690	70.426	8.392	1.586

Unpaired t-test for Maximum Grip Strength

Grouping Variable: Sex

Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
F, M	-21.520	68	-12.394	<.0001

Group Info for Maximum Grip Strength

Grouping Variable: Sex

	Count	Mean	Variance	Std. Dev.	Std. Err
F	44	32.211	22.111	4.702	.709
M	26	53.731	95.985	9.797	1.921

Unpaired t-test for Maximum Pinch Strength

Grouping Variable: Sex

Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
F, M	-8.024	67	-5.447	<.0001

Group Info for Maximum Pinch Strength

Grouping Variable: Sex

	Count	Mean	Variance	Std. Dev.	Std. Err
F	43	43.860	39.075	6.251	.953
M	26	51.885	28.586	5.347	1.049

Unpaired t-test for PEFR

Grouping Variable: Sex

Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
F, M	-115.378	77	-6.973	<.0001

Group Info for PEFR

Grouping Variable: Sex

	Count	Mean	Variance	Std. Dev.	Std. Err
F	51	446.765	4369.824	66.105	9.257
M	28	562.143	6019.312	77.584	14.662

Table 31.

The difference in the mean values among males of the “V O₂ max. estimate at initial assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

ANOVA Table for VO₂ Max.**For : Sex = Male**

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	4.509	2.255	.030	.9708
Residual	25	1896.982	75.879		

Model II estimate of between component variance: •

Means Table for VO₂ Max.**Effect: Followup Diagnostic Classification****For : Sex = Male**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	12	54.910	10.392	3.000
Inconsequential	11	54.210	7.245	2.185
Consequential	5	55.218	6.783	3.033

Table 32.

The difference in the mean values among females of the “V O₂ max. estimate at initial assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

ANOVA Table for VO₂ Max.**For : Sex = Female**

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	8.024	4.012	.382	.6847
Residual	46	483.219	10.505		

Model II estimate of between component variance: •

5 cases were omitted due to missing values.

Means Table for VO₂ Max.**Effect: Followup Diagnostic Classification****For : Sex = Female**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	24	37.551	3.595	.734
Inconsequential	15	38.327	3.342	.863
Consequential	10	37.292	1.816	.574

5 cases were omitted due to missing values.

Table 33.

The difference in the mean values of the “Number of Hours of Physical Activity per Week at initial assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

ANOVA Table for Hours Physical Activity per Week

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	16.815	8.407	.326	.7229
Residual	79	2038.371	25.802		

Model II estimate of between component variance: •

Means Table for Hours Physical Activity per Week
Effect: Followup Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	38	4.118	5.180	.840
Inconsequential	28	3.196	4.530	.856
Consequential	16	4.219	5.724	1.431

Table 34.

The difference in the mean values of the “Dexterity Test at initial assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

ANOVA Table for Dexterity

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	39.751	19.876	.159	.8536
Residual	77	9647.436	125.291		

Model II estimate of between component variance: •
 2 cases were omitted due to missing values.

Means Table for Dexterity
Effect: Followup Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	37	131.622	8.254	1.357
Inconsequential	27	130.370	14.539	2.798
Consequential	16	132.188	10.641	2.660

2 cases were omitted due to missing values.

Table 35.

The difference in the mean values among males of the “Grip Strength at initial assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

ANOVA Table for Maximum Grip Strength
For : Sex = Male

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	24.321	12.161	.118	.8894
Residual	23	2375.307	103.274		

Model II estimate of between component variance: •
2 cases were omitted due to missing values.

Means Table for Maximum Grip Strength
Effect: Followup Diagnostic Classification
For : Sex = Male

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	11	54.512	8.400	2.533
Inconsequential	11	52.603	10.376	3.128
Consequential	4	54.682	14.062	7.031

2 cases were omitted due to missing values.

Table 36.

The difference in the mean values among females of the “Grip Strength at initial assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA. Fisher’s PLSD was used to determine the significance of between group differences.

ANOVA Table for Maximum Grip Strength
For : Sex = Female

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	152.627	76.314	3.920	.0277
Residual	41	798.162	19.467		

Model II estimate of between component variance: 4.002
10 cases were omitted due to missing values.

Means Table for Maximum Grip Strength
Effect: Followup Diagnostic Classification
For : Sex = Female

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	19	32.536	4.541	1.042
Inconsequential	15	33.958	4.476	1.156
Consequential	10	28.973	4.035	1.276

10 cases were omitted due to missing values.

Fisher's PLSD for Maximum Grip Strength
Effect: Followup Diagnostic Classification
Significance Level: 5 %
For : Sex = Female

	Mean Diff.	Crit. Diff	P-Value	
No Symptoms, Inconsequential	-1.422	3.078	.3563	
No Symptoms, Consequential	3.563	3.481	.0451	S
Inconsequential, Consequential	4.985	3.638	.0084	S

10 cases were omitted due to missing values.

Table 37.

The difference in the mean values among males of the "Pinch Strength at initial assessment" between the categories of "Followup Diagnostic Classification" were compared with a one-way ANOVA.

ANOVA Table for Maximum Pinch Strength
For : Sex = Male

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	2.927	1.463	.047	.9539
Residual	23	711.727	30.945		

Model II estimate of between component variance: •
2 cases were omitted due to missing values.

Means Table for Maximum Pinch Strength
Effect: Followup Diagnostic Classification
For : Sex = Male

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	11	51.545	4.132	1.246
Inconsequential	11	52.000	7.225	2.178
Consequential	4	52.500	2.517	1.258

2 cases were omitted due to missing values.

Table 38.

The difference in the mean values among females of the "Pinch Strength at initial assessment" between the categories of "Followup Diagnostic Classification" were compared with a one-way ANOVA.

ANOVA Table for Maximum Pinch Strength
For : Sex = Female

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	105.563	52.781	1.375	.2646
Residual	40	1535.600	38.390		

Model II estimate of between component variance: 1.031
11 cases were omitted due to missing values.

Means Table for Maximum Pinch Strength
Effect: Followup Diagnostic Classification
For : Sex = Female

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	18	42.667	6.362	1.499
Inconsequential	15	46.000	3.381	.873
Consequential	10	42.800	8.741	2.764

11 cases were omitted due to missing values.

Table 39.

The difference in the mean values among males of the "V O₂ max. estimate at initial assessment" between the categories of "Followup Diagnostic Classification" were compared with a one-way ANOVA.

ANOVA Table for PEFR

For : Sex = Male

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	22295.785	11147.892	1.987	.1581
Residual	25	140225.644	5609.026		

Model II estimate of between component variance: 627.888

Means Table for PEFR

Effect: Followup Diagnostic Classification

For : Sex = Male

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	12	547.917	78.724	22.726
Inconsequential	11	595.455	60.723	18.309
Consequential	5	523.000	93.782	41.940

Table 40.

The difference in the mean values among females of the "V O₂ max. estimate at initial assessment" between the categories of "Followup Diagnostic Classification" were compared with a one-way ANOVA.

ANOVA Table for PEFR

For : Sex = Female

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	7647.176	3823.588	.870	.4253
Residual	48	210844.000	4392.583		

Model II estimate of between component variance: •
3 cases were omitted due to missing values.

Means Table for PEFR

Effect: Followup Diagnostic Classification

For : Sex = Female

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	25	457.800	61.273	12.255
Inconsequential	16	442.500	70.285	17.571
Consequential	10	426.000	71.988	22.764

3 cases were omitted due to missing values.

Table 41.

Contingency table analysis of association between “Poor Postural at Initial Assessment” and “Followup Diagnostic Classification”.

Contingency table analysis: Poor Posture of Followup Diagnostic Classification

Observed Frequencies					Summary Table	
	No Symptoms	Inconsequential	Consequential	Totals		
No	21	10	8	39	Num. Missing	1
Yes	17	18	7	42	DF	2
Totals	38	28	15	81	Chi Square	2.666
					Chi Square P-Value	.2637
					G-Squared	2.695
					G-Squared P-Value	.2599
					Contingency Coef.	.179
					Cramer's V	.181

Table 42.

Student’s t-test was used to compare the differences in the physical attributes “Hand Span” and “Body Mass Index” between males and females.

Unpaired t-test for Hand Span

Grouping Variable: Sex

Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
F, M	-25.886	79	-9.672	<.0001

Group Info for Hand Span

Grouping Variable: Sex

	Count	Mean	Variance	Std. Dev.	Std. Err
F	53	202.792	125.706	11.212	1.540
M	28	228.679	141.856	11.910	2.251

Unpaired t-test for Body Mass Index

Grouping Variable: Sex

Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
F, M	.436	79	.655	.5141

Group Info for Body Mass Index

Grouping Variable: Sex

	Count	Mean	Variance	Std. Dev.	Std. Err
F	53	22.174	8.028	2.833	.389
M	28	21.738	8.261	2.874	.543

Table 43.

The difference in the mean values among males of the “Maximum Hand Span Measurement” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

**ANOVA Table for Hand Span
For Sex = Male**

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	36.875	18.438	.122	.8861
Residual	25	3793.232	151.729		

Model II estimate of between component variance: •

**Means Table for Hand Span
Effect: Followup Diagnostic Classification
For Sex = Male**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	12	229.750	14.900	4.301
Inconsequential	11	227.273	9.634	2.905
Consequential	5	229.200	10.281	4.598

Table 44.

The difference in the mean values among females of the “Maximum Hand Span Measurement” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

**ANOVA Table for Hand Span
For Sex = Female**

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	64.502	32.251	.249	.7804
Residual	50	6472.215	129.444		

Model II estimate of between component variance: •
One case was omitted due to missing values.

**Means Table for Hand Span
Effect: Followup Diagnostic Classification
For Sex = Female**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	26	202.769	11.742	2.303
Inconsequential	17	204.000	12.047	2.922
Consequential	10	200.800	8.842	2.796

One case was omitted due to missing values.

Table 45.

The difference in the mean values of the “Body Mass Index at initial assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

ANOVA Table for Body Mass Index

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	.441	.220	.027	.9737
Residual	78	643.546	8.251		

Model II estimate of between component variance: •
 One case was omitted due to missing values.

Means Table for Body Mass Index
Effect: Followup Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	37	22.062	2.760	.454
Inconsequential	28	22.056	2.318	.438
Consequential	16	21.875	3.867	.967

One case was omitted due to missing values.

Table 46.

Student’s t-test was used to compare the differences in “mean number of hours music instrument performance per week” between males and females.

Unpaired t-test for Hrs Music Instrument Performance per Week

Grouping Variable: Sex

Hypothesized Difference = 0

	Mean Diff.	DF	t-Value	P-Value
F, M	1.305	78	.621	.5362

Group Info for Hrs Music Instrument Performance per Week

Grouping Variable: Sex

	Count	Mean	Variance	Std. Dev.	Std. Err
F	52	19.234	88.987	9.433	1.308
M	28	17.929	63.958	7.997	1.511

Table 47.

The differences in “mean number of hours music instrument performance per week at time of Initial Assessment” between the categories of “Followup Diagnostic Classification” were compared with a one-way ANOVA.

ANOVA Table for Hrs Music Instrument Performance per Week

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	629.752	314.876	4.279	.0173
Residual	77	5666.467	73.590		

Model II estimate of between component variance: 9.599
 2 cases were omitted due to missing values.

Means Table for Hrs Music Instrument Performance per Week
Effect: Followup Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	37	16.554	9.592	1.577
Inconsequential	28	22.589	8.061	1.523
Consequential	15	17.144	6.544	1.690

2 cases were omitted due to missing values.

Fisher's PLSD for Hrs Music Instrument Performance per Week
Effect: Followup Diagnostic Classification
Significance Level: 5 %

	Mean Diff.	Crit. Diff	P-Value
No Symptoms, Inconsequential	-6.035	4.279	.0063
No Symptoms, Consequential	-.590	5.229	.8227
Inconsequential, Consequential	5.445	5.466	.0509

2 cases were omitted due to missing values.

Table 48.

Unpaired t-test was used to assess whether there were significant sex differences in the mean values in the scores from the psychological questionnaires (Standard Error of the Mean in brackets adjacent to mean values).

Psychological Questionnaire	Mean Value Females	Mean Value Males	Unpaired t-test t Value	t-test p Value
GHQ	4.37 (0.83)	4.46 (1.02)	-0.069	0.95
SAQ (Trait)	37.18 (1.27)	36.25 (1.35)	0.471	0.64
IBQ_GH	1.41 (0.27)	1.14 (0.24)	0.659	0.51
IBQ_DC	1.02 (0.15)	1.04 (0.15)	-0.070	0.94
IBQ_PvSC	1.98 (0.10)	2.21 (0.16)	-1.291	0.20
IBQ_AI	2.39 (0.24)	2.07 (0.31)	0.807	0.42
IBQ_AD	1.71 (0.23)	1.07 (0.26)	1.724	0.09
IBQ_D	3.18 (0.17)	3.00 (0.19)	0.667	0.51
IBQ_I	1.16 (0.18)	0.82 (0.17)	1.211	0.23

Table 49.

The Correlation Coefficient of each of the factors with its associated "Normal Scores" was calculated. This correlation coefficient is compared with the critical value for the 5% statistical significance level which for N = 80 is 0.9843 [MiniTab Reference Manual]. Factors whose correlation coefficient lies below this value have less than 5% probability of being normally distributed and so are analysed with the use of non-parametric statistics.

Psychological Questionnaire	Correlation Coefficient of Factor with its Normal Scores
GHQ	0.908
SAQ (Trait)	0.992
IBQ_GH	0.938
IBQ_DC	0.953
IBQ_PvSC	0.985
IBQ_AI	0.994
IBQ_AD	0.994
IBQ_D	0.992
IBQ_I	0.989

Table 50.

The means of the General Health Questionnaire (GHQ) scores for the different Diagnostic Classification categories were compared with use of the Kruskal-Wallis non-parametric one-way ANOVA. No significant difference was found.

Kruskal-Wallis Test for GHQ**Grouping Variable: Followup Diagnostic Classification**

DF	2
# Groups	3
# Ties	11
H	2.450
P-Value	.2937
H corrected for ties	2.520
Tied P-Value	.2837

Kruskal-Wallis Rank Info for GHQ**Grouping Variable: Followup Diagnostic Classification**

	Count	Sum Ranks	Mean Rank
No Symptoms	38	1410.500	37.118
Inconsequential	28	1285.500	45.911
Consequential	16	707.000	44.188

Table 51.

The means of the Spielberger Anxiety Questionnaire; Trait Factor (SAQ) scores for the different Diagnostic Classification categories were compared with use of parametric one-way ANOVA. No significant difference was found.

ANOVA Table for SAQ (Trait)

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	220.725	110.363	1.618	.2051
Residual	75	5115.429	68.206		

Model II estimate of between component variance: 1.727
4 cases were omitted due to missing values.

Means Table for SAQ (Trait)**Effect: Followup Diagnostic Classification**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	36	35.083	7.736	1.289
Inconsequential	28	38.750	8.885	1.679
Consequential	14	37.571	8.271	2.211

4 cases were omitted due to missing values.

Table 52.

The means of the Illness Behaviour Questionnaire: factor “General Hypochondriasis” scores (IBQ_GH) for the different Diagnostic Classification categories were compared with use of the Kruskal-Wallis non-parametric one-way ANOVA. A significant difference between categories was found. Mann-Whitney U-test was used post-hoc to assess between group differences.

Kruskal-Wallis Test for IBQ_GH

Grouping Variable: Followup Diagnostic Classification

DF	2
# Groups	3
# Ties	5
H	8.487
P-Value	.0144
H corrected for ties	9.347
Tied P-Value	.0093

Kruskal-Wallis Rank Info

	Count	Sum Ranks	Mean Rank
No Symptoms	37	1194.500	32.284
Inconsequential	28	1365.500	48.768
Consequential	14	600.000	42.857

3 cases were omitted due to missing values.

Mann-Whitney U applied post-hoc to assess between group differences for IBQ_GH

Grouping Variable: Followup Diagnostic Classification

Rank Information Comparison 1

	Count	Sum Ranks	Mean Rank
No Symptoms	37	1007.000	27.216
Inconsequential	28	1138.000	40.643

One case was omitted due to missing values.

Summary Table 1

U	304.000
U Prime	732.000
Z-Value	-2.835
P-Value	.0046
Tied Z-Value	-2.986
Tied P-Value	.0028
# Ties	5

Rank Information Comparison 2

	Count	Sum Ranks	Mean Rank
No Symptoms	37	890.500	24.068
Consequential	14	435.500	31.107

3 cases were omitted due to missing values.

Summary Table 2

U	187.500
U Prime	330.500
Z-Value	-1.509
P-Value	.1313
Tied Z-Value	-1.632
Tied P-Value	.1027
# Ties	4

Rank Information Comparison 3

	Count	Sum Ranks	Mean Rank
Inconsequential	28	633.500	22.625
Consequential	14	269.500	19.250

2 cases were omitted due to missing values.

Summary Table 3

U	164.500
U Prime	227.500
Z-Value	-.840
P-Value	.4006
Tied Z-Value	-.863
Tied P-Value	.3879
# Ties	5

Table 53.

The means of the Illness Behaviour Questionnaire: factor “Disease Conviction” (IBQ_DC) scores for the different Diagnostic Classification categories were compared with use of the Kruskal-Wallis non-parametric one-way ANOVA. No significant difference was found.

Kruskal-Wallis Test for IBQ_DC

Grouping Variable: Followup Diagnostic Classification

DF	2
# Groups	3
# Ties	4
H	2.739
P-Value	.2542
H corrected for ties	3.190
Tied P-Value	.2029

Kruskal-Wallis Rank Info

	Count	Sum Ranks	Mean Rank
No Symptoms	37	1312.000	35.459
Inconsequential	28	1223.500	43.696
Consequential	14	624.500	44.607

3 cases were omitted due to missing values.

Table 54.

The means of the Illness Behaviour Questionnaire: factor “Psychological vs somatic concern” (IBQ_PvSC) scores for the different Diagnostic Classification categories were compared with use of parametric one-way ANOVA. A significant difference between categories was found. Fisher’s PLSD post-hoc test was used to assess between group differences.

ANOVA Table for IBQ_PvSC

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	5.041	2.520	4.600	.0130
Residual	76	41.643	.548		

Model II estimate of between component variance: .08

3 cases were omitted due to missing values.

Means Table for IBQ_PvSC

Effect: Followup Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	37	2.000	.577	.095
Inconsequential	28	2.357	.951	.180
Consequential	14	1.643	.633	.169

3 cases were omitted due to missing values.

Fisher's PLSD for IBQ_PvSC

Effect: Followup Diagnostic Classification

Significance Level: 5 %

	Mean Diff.	Crit. Diff.	P-Value
No Symptoms, Inconsequential	-.357	.369	.0578
No Symptoms, Consequential	.357	.463	.1283
Inconsequential, Consequential	.714	.483	.0042

3 cases were omitted due to missing values.

Table 55.

The means of the Illness Behaviour Questionnaire: factor "Affective inhibition" (IBQ_AI) scores for the different Diagnostic Classification categories were compared with use of parametric one-way ANOVA. No significant difference was found.

ANOVA Table for IBQ_AI

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	6.452	3.226	1.138	.3258
Residual	76	215.422	2.834		

Model II estimate of between component variance: .016

3 cases were omitted due to missing values.

Means Table for IBQ_AI

Effect: Followup Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	37	2.486	1.592	.262
Inconsequential	28	1.893	1.729	.327
Consequential	14	2.500	1.829	.489

3 cases were omitted due to missing values.

Table 56.

The means of the Illness Behaviour Questionnaire: factor "Affective disturbance" (IBQ_AD) scores for the different Diagnostic Classification categories were compared with use of parametric one-way ANOVA. A significant difference between categories was found. Fisher's PLSD post-hoc test was used to assess between group differences.

ANOVA Table for IBQ_AD

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	16.150	8.075	3.418	.0379
Residual	76	179.571	2.363		

Model II estimate of between component variance: .232

3 cases were omitted due to missing values.

Means Table for IBQ_AD

Effect: Followup Diagnostic Classification

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	37	1.000	1.291	.212
Inconsequential	28	1.929	1.720	.325
Consequential	14	1.857	1.748	.467

3 cases were omitted due to missing values.

Fisher's PLSD for IBQ_AD

Effect: Followup Diagnostic Classification

Significance Level: 5 %

	Mean Diff.	Crit. Diff	P-Value
No Symptoms, Inconsequential	-.929	.767	.0183
No Symptoms, Consequential	-.857	.961	.0795
Inconsequential, Consequential	.071	1.002	.8875

3 cases were omitted due to missing values.

Table 57.

The means of the Illness Behaviour Questionnaire: factor "Denial" (IBQ_D) scores for the different Diagnostic Classification categories were compared with use of parametric one-way ANOVA. No significant difference was found.

ANOVA Table for IBQ_D

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	.537	.269	.210	.8114
Residual	76	97.437	1.282		

Model II estimate of between component variance: •
3 cases were omitted due to missing values.

Means Table for IBQ_D**Effect: Followup Diagnostic Classification**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	37	3.027	1.067	.175
Inconsequential	28	3.179	1.219	.230
Consequential	14	3.214	1.122	.300

3 cases were omitted due to missing values.

Table 58.

The means of the Illness Behaviour Questionnaire: factor "Irritability" (IBQ_I) scores for the different Diagnostic Classification categories were compared with use of parametric one-way ANOVA. No significant difference was found.

ANOVA Table for IBQ_I

	DF	Sum of Squares	Mean Square	F-Value	P-Value
Followup Diagnostic Classification	2	3.819	1.909	1.381	.2575
Residual	76	105.068	1.382		

Model II estimate of between component variance: .021
3 cases were omitted due to missing values.

Means Table for IBQ_I**Effect: Followup Diagnostic Classification**

	Count	Mean	Std. Dev.	Std. Err.
No Symptoms	37	.892	1.265	.208
Inconsequential	28	1.000	1.122	.212
Consequential	14	1.500	1.019	.272

3 cases were omitted due to missing values.

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[Vancouver style used for formatting references; Abbreviations used from Index Medicus "List of Journals Indexed".]

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