PSYCHOPHYSIOLOGICAL CORRELATES OF LOW BACK PAIN

by

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Abstract

Low Back Pain (LBP) is extremely common and is perhaps the single most socially-costly medical disorder. Yet, very little is known about the etiology of LBP, and current treatments for the disorder are thus correspondingly ineffective.

The research reported here was designed to test a general psychophysiological model of the etiology of psychosomatic disorders, applied to LBP and lumbar intervertebral disc degeneration by way of established biomechanical principles. The general model was proposed by Sternbach (1966) who hypothesized that, in the event of repeated, excessive environmental stress, that body part which is the most psychophysiologically responsive will break down. This process is promoted by the lack of normal homeostatic restraints, restraints which are often found lacking in neurotic individuals (Alexander, 1972; Goldstein, 1972).

In specific application of the Sternbach model to the LBP condition, it was hypothesized that electromyographic stress responses of abnormal magnitude and duration are evident in the posterior lumbar and abdominal oblique muscles of LBP subjects. On the basis of well researched biomechanical and pathophysiological mechanisms, reviewed in this paper, such muscle response abnormalities would be expected to give rise to LBP and to hasten degeneration of the lumbar intervertebral discs.

Asymptomatic subjects with a minimal history of LBP, when compared to normal Control subjects without such a history, were in fact not found to exhibit the critical characteristics of the Sternbach model. The LBP subjects were not more neurotic than members of the general population, and in response to various stressors, neither their posterior lumbar muscles nor

their abdominal oblique muscles showed activity that was of excessive magnitude or duration.

Two unexpected findings, however, provided new information which can be incorporated into established biomechanical processes which, in additive or synergistic fashion, would be expected to contribute to the occurrence of LBP and lumbar intervertebral disc degeneration. First, it was found that the LBP subjects showed less activity in the posterior lumbar muscles than did the Control subjects. This finding is discussed in the context of established biomechanical principles of spinal stabilization and in terms of pathophysiological processes of intervertebral disc degeneration resulting from shear forces acting on the poorly stabilized spine. Second, it was found that during the occurrence of psychological and physical stressors, LBP subjects did not restrict their respiration rate as much as did Control subjects. This finding is discussed in terms of the hydraulic abdominal "balloon effect" which, if decreased, could be expected to expose the lumbar spine to destructive forces and trauma, producing LBP and lumbar intervertebral disc degeneration.

Possible causes for the apparent psychophysiological anomalies found in LBP subjects and possible corrective procedures to overcome them are discussed, and suggestions for further research are given.

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Psychophysiological Correlates of Low Back Pain

INTRODUCTION

Low Back Pain (LBP) is one of the most frequent and costly health problems. It has been described by Finneson, a senior authority on the condition, as "...the worst plague of the twentieth century" (Neal, 1978). Hult (1954), on the basis of early Swedish research, suggested that about two-thirds of all people experience LBP at some time in their lives and over one-third are at some time incapacitated by it, but these figures are probably too conservative (e.g., Nachemson, 1976).

Rowe (1969) has shown that LBP is the second most common cause of time loss from work (second only to upper respiratory infections). The incidence of compensable time loss from work would appear to be about two percent of workers per year (Kelsey, White, Pastides & Bisbee, 1979; Nachemson, 1976). There are some eight million Americans with permanent impairments of the spine, and of the chronic health conditions these are the most common and costly during the prime working years (Kelsey et al., 1979; Nachemson, 1976).

In industrial settings, 13% - 38% of all injury claims involve the low back (Drouin, 1973; Kosiak, Aurelius & Hartfiel, 1966; Schein, 1968; Sternbach, Wolf, Murphy & Akeson, 1973; Troup, 1966), and at the British Columbia Workers' Compensation

Board (BCWCB) over 25,000 new LBP claims are now received each year (Satterberg, 1978). Over 380,000 compensated working days were lost because of LBP in British Columbia in 1977[1].

Estimates of the annual cost of LBP problems have often been attempted and have given rise to overwhelming, perhaps subjectively incomprehensible figures. Neal (1978) has estimated the loss in productivity due to LBP to be 14 to 15 billion dollars per year in the U.S.A. Fordyce (1979) has reported that the direct costs of LBP problems at the Washington State WCB amounted to 63 million dollars in 1977, and Satterberg (1978) estimated that in British Columbia the longest 19% of LBP claims (over 8 weeks of disability) cost in excess of 15 million dollars in time loss and pension awards in 1976. Many other annual cost figures, stated in millions if not billions of dollars, can be found (Drouin, 1973; Hayes, 1970; Sternbach et al., 1973; Troup, Roantree & Archibald, 1970).

The magnitude of the LBP problem is perhaps best appreciated in settings such as a WCB, which are inundated by LBP cases which are typically the most chronic and difficult-to-manage cases[2]. Over one third of all admissions to the BCWCB Rehabilitation Clinic involve the low back (Gunn & Milbrandt,

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1. Farish, J. R. Surgical Consultant, BCWCB, Personal Communication, 1979.

2. The present author has been employed at the BCWCB for over five years.

1976), and many BCWCB front-line personnel estimate that they spend up to 80% of their working time on LBP claims.

Even more disturbing than the current incidence and cost figures concerning the LBP problem are recent analyses indicating that the incidence of LBP disability is growing more rapidly than the work force or other disabilities generally (Brown, 1977; Drouin, 1973; Kelsey et al., 1979; Kosiak et al., 1966; Tunturi & Patiala, 1980; Wickstrom, 1978).

Despite the magnitude of the problem of LBP, the status of knowledge pertaining to causative pathological organic conditions is very poor (Fahrni, 1975; MacNab, 1978; Nachemson, 1976). This current situation is probably explicable by the facts that, firstly, the low back is a highly complex structure having some 140 bony segments, ligaments and muscles all intertwined with neural tissues and operating in multiple planes (Casa Colina, 1976) and, secondly, the methods on which most current orthopaedic practice is based are unscientific, mainly empirical, and often ancient (Fahrni, 1975). MacNab (1978) has referred to "...our remarkable and disturbing ignorance...", and Troup et al. (1970) have remarked on "...the lack of scientific data" concerning LBP. There are dozens of pathological conditions suggested in the medical literature to be causes of LBP, and the acceptance of these explanations often appears to be influenced by the status of the author, his status not being necessarily determined by the scientific adequacy of his

work or by the successes of the corrective procedure directed at the pathological condition by the author, his students, and his followers. As MacNab (1978) has noted, "...we have stumbled from hunch to hunch".

The orthopaedic clinical-impression/empirical approach appears to be highly error-prone for several reasons: Firstly, there are many types of congenital and degenerative anomalies of the human spine and after the second decade of life, one or more such anomalies can be found in the spines of up to 70% of individuals (McGill, 1968). Methodologically adequate studies have in fact shown little or no difference between symptomatic and asymptomatic groups in the incidence of various forms of spinal pathology (Fullenlove & Williams, 1957; LaRocca & MacNab, 1969; Splitoff, 1953). Secondly, there appears to be a high spontaneous recovery rate of LBP left untreated (Nachemson, 1976), and this spontaneous recovery rate is rarely taken into account in evaluating the efficacy of active treatment modalities. In fact, the symptomatic recovery rate from various forms of conservative treatment or spinal surgery rarely appears to be better than the spontaneous recovery rate (Kark, 1972; Nachemson, 1976). The present situation confronting the clinically-empirically-oriented physician then, is that there are many types of pathology to choose from, many of the patients presenting with LBP will exhibit a given pathology, and many of the patients will show improvement with time, almost

irrespective of the treatment applied to that pathology. In many ways the present status of knowledge concerning LBP is similar to the status of knowledge concerning psychotherapy in 1952, when Eysenck showed that the then-popular psychotherapies appeared to be contributing to symptomatic improvement in two-thirds of patients treated, when in fact this apparent efficacy was illusory because the same percentage of untreated patients was recovering from symptoms spontaneously.

Medical Aspects of Low Back Pain

Introduction

This paper will deal with a psychosomatic model of LBP which by its nature is interdisciplinary, and some level of knowledge of the anatomy and pathology of the human back is thus required by psychologists considering it. Some understanding of current medical approaches to the treatment of LBP is also necessary for an appreciation of why other approaches are necessary. This section is thus written to provide necessary general medical information to non-orthopaedists and, as such, the consideration of various topics covered will not be exhaustive of the literature available, but rather will cover only the major current trends. The reader wishing a more comprehensive review of medical information is referred to some of the excellent overviews available (Adams, 1962; Brown, 1977; Nachemson, 1976; Rothman & Simeone, 1975).

Anatomy of the Lumbar Spine

The lumbar spine is composed of five bony vertebrae extending caudally from the 12th thoracic vertebra, which is the lowest vertebra having an attached rib, to the fused vertebrae which form the sacrum. The lumbar vertebrae are numbered in the caudal direction, and the spinous process of the 5th lumbar vertebra (designated as L5) can be felt approximately three inches superior to the upper extent of the natal cleft (the buttocks' vertical fold). The lumbar vertebrae and sacrum form a curve, concave posteriorly, referred to as the lumbar lordosis. These aspects of the lumbar spine are demonstrated in Figure 1.

A vertebra consists of a solid, approximately cylindrical, vertebral body with a number of bony posterior projections (see Figure 2). These rearward projections provide transverse and spinous attachment processes for ligaments binding the stack of vertebrae together and superior and inferior articular processes forming joints between adjacent vertebrae.

Figure 3 demonstrates the articulation (interpositioning) of two vertebrae from the lateral and posterior views. The inferior articular process of one vertebra and the superior articular process of the next lower vertebra form the facet or apophyseal joint, the plane of this joint lying at approximately 45 degrees to the sagittal plane of the body. The articular processes are wedge shaped, with the inferior processes of the cephalad vertebra being medial to the superior processes

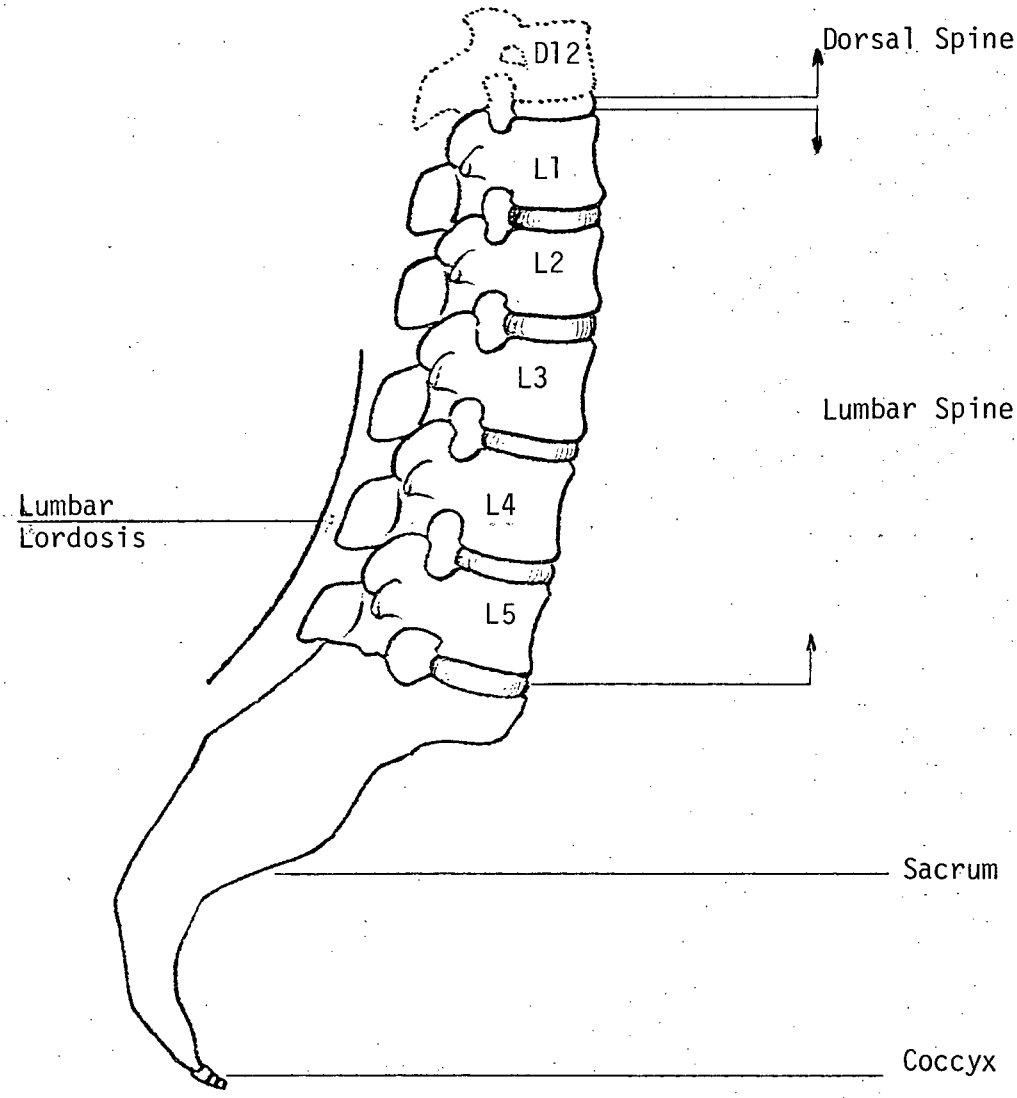


FIGURE 1. Lateral View of Lumbar Spine

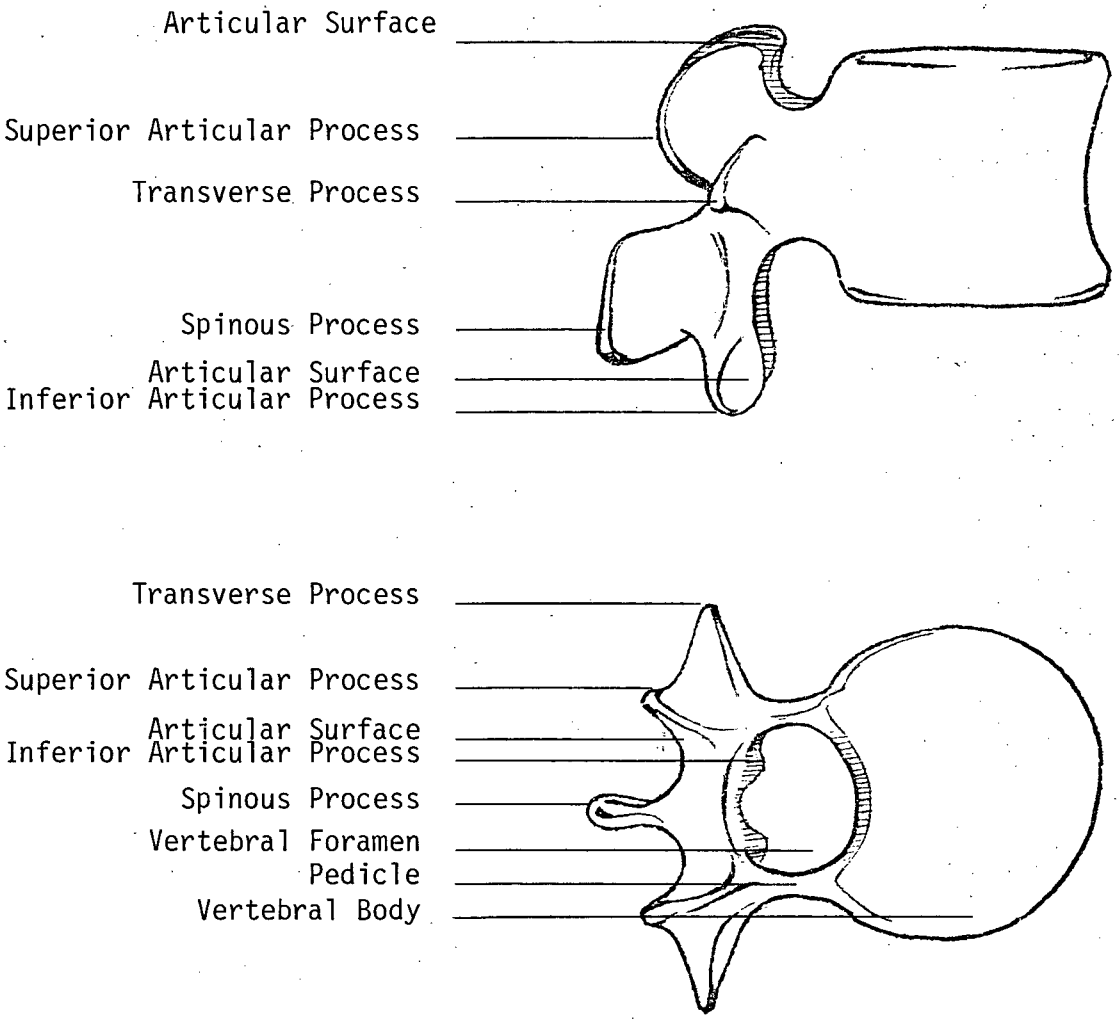


FIGURE 2. Lateral and Superior Views of a Lumbar Vertebra

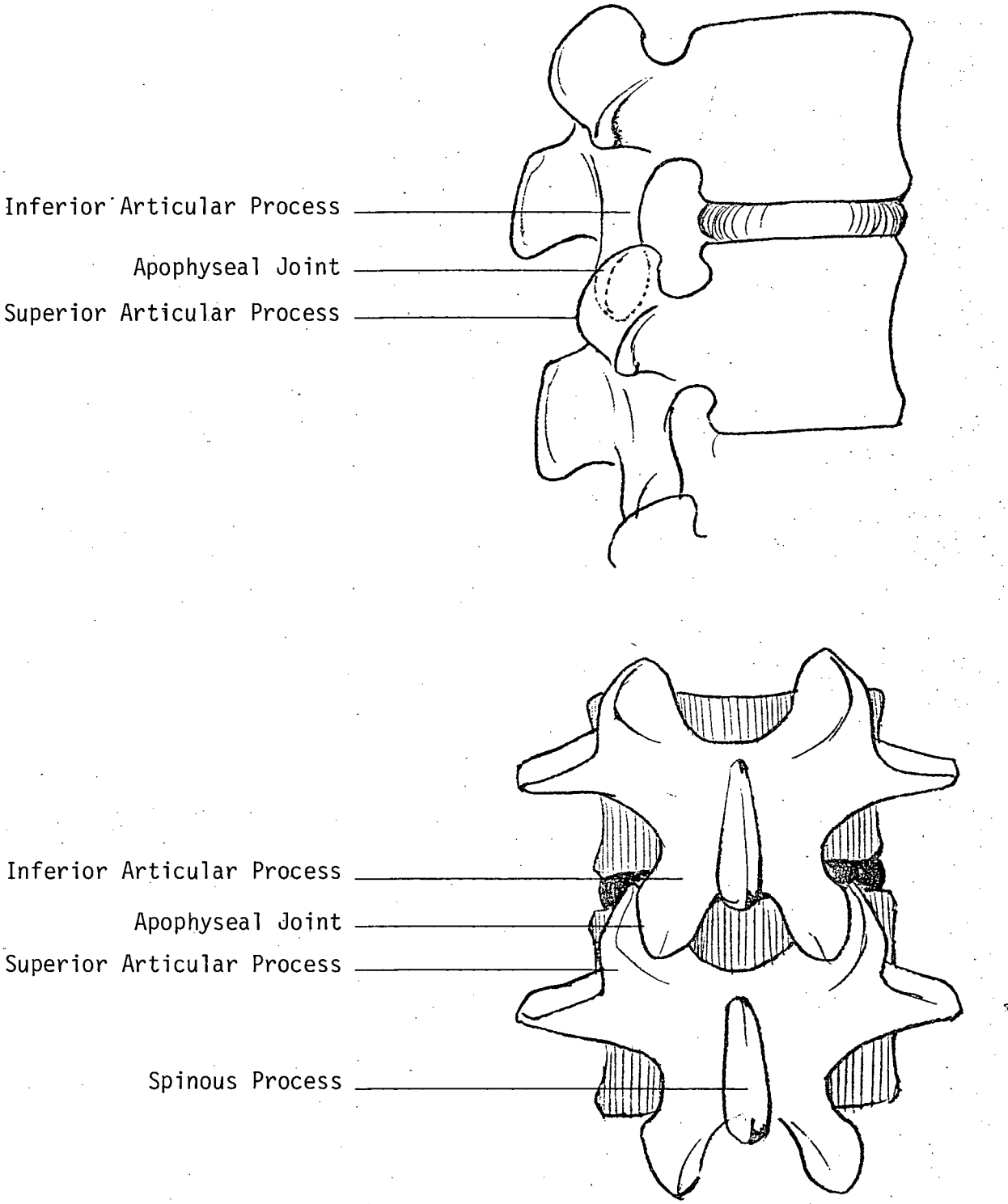


FIGURE 3. Lateral and Posterior Views of the Articulation of Two Lumbar Vertebrae

of the caudal vertebra. The dura-clad spinal nerves, or cauda equina, lies within the spinal canal formed by the vertebral foramina. The pedicles of each vertebra are arched between the vertebral body and apophyseal joint, creating inferior and superior notches. The nerve roots, which subserve sensory and motor functions in the lower body, exit the spinal canal through the holes or foramina formed by these notches between each two stacked vertebrae.

As illustrated in Figures 1 and 3, the vertebrae are separated by intervertebral discs. The disc acts as a cushion and is structurally similar to a flattened golf ball, having cartilaginous end plates at the disc's interface with the vertebral bodies, a gelatinous centre called the nucleus pulposus, and a spirally-arranged fibrous periphery called the annulus fibrosus, which is composed of very long-chain organic molecules including collagen and mucopolysaccharides (Brown, 1971; Naylor, 1971). Hydraulic action allows the healthy disc to distribute weight evenly on the vertebral endplates while allowing movement in all directions (Nachemson, 1975; Parke & Schiff, 1971).

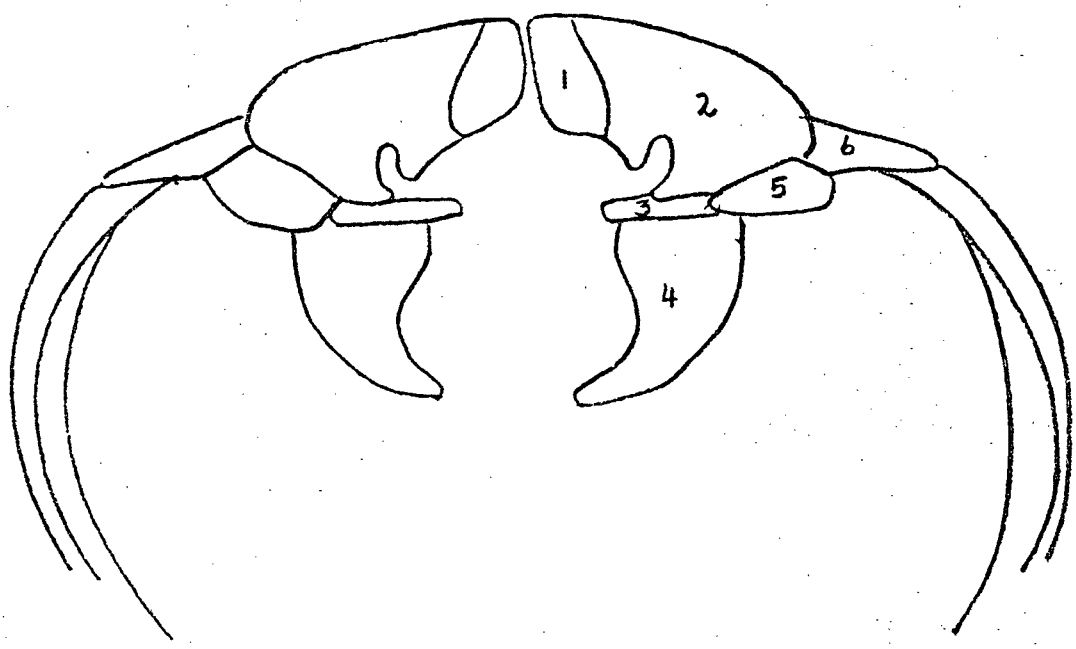
The vertebrae are bound together with numerous short ligaments between the vertebral bodies and between the transverse and spinous processes and by posterior and anterior longitudinal ligaments running the length of the spinal column.

The bony segments of the spinal column are also surrounded

by numerous muscle groups, which stabilize the column and provide motor power for movement in all planes. The arrangement of muscles seen on a transverse plane through the L3 level is demonstrated in Figure 4. Surprisingly, the exact functions of various muscles of the back are not well understood, and one of the most prominent investigators of muscle function is pessimistic that, because of their complexity, the exact functions of the various muscle groups will ever be differentiated (Basmajian, 1974). It is quite evident from a consideration of basic mechanical principles, however, that those muscles lying parallel to the spine must have major involvements in flexion/extension of the trunk, whereas those muscles with oblique orientations must have major involvements in rotation of the trunk and spinal stabilization (Farfan, 1973).

Biomechanics of the Lumbar Spine

The oblique muscles in various combinations provide the motor forces for rotational movements of the trunk, the degree of rotation being limited by the obliquely-oriented, wedge-shaped apophyseal joints. Flexion and extension of the trunk are brought about by two mechanisms: firstly, by contraction of muscles running parallel to and posterior to the spinal column, with possibly some help from the oblique muscles and; secondly, by a hydraulic "balloon effect" involving the abdomen (Bartelink, 1957). The balloon effect is created by the tightening of the oblique abdominal muscles, which causes the soft abdominal



1. Multifidus
2. Sacrospinalis
3. Iliocostalis
4. Psoas
5. Quandratus Lumborum
6. Latissimus Dorsi

FIGURE 4. Back Muscles seen in a Transverse Section Through L3 Vertebra (After Farfan, 1973)

contents to push on the pelvic floor and diaphragm, thereby promoting extension of the trunk, this being similar in principle to the industrial application of low-pressure air bags to the lifting of heavy objects. The abdominal balloon effect is in all probability very important to movements of the trunk, because extension brought about only by the muscle groups posterior to the spinal column is limited by the very ineffective mechanics of a first-class lever having a very long lever arm to the load and a very short lever arm to the mode of force. These ineffective mechanics are illustrated in Figure 5. The unloading effect on the discs of the balloon effect can be readily appreciated.

Bending of the vertebral column is made possible by the intervertebral discs which, through their contained-liquid centres, act as distensible hydraulic cushions and shock absorbers, allowing an even distribution of vertical loading forces over the vertebral end plates. The fulcrum of movement of the first class lever described above is, as shown in Figure 5, in the posterior portion of the disc (DePalma & Rothman, 1970; White & Panjabi, 1978). Because of the mechanical inefficiency of the first class lever described above, force loadings of great magnitude act on the discs. The muscles posterior to the spine, which provide the motor force on the short arm of the lever demonstrated in Figure 5, are of massive size and have been calculated by Farfan (1973) to be capable of a direct

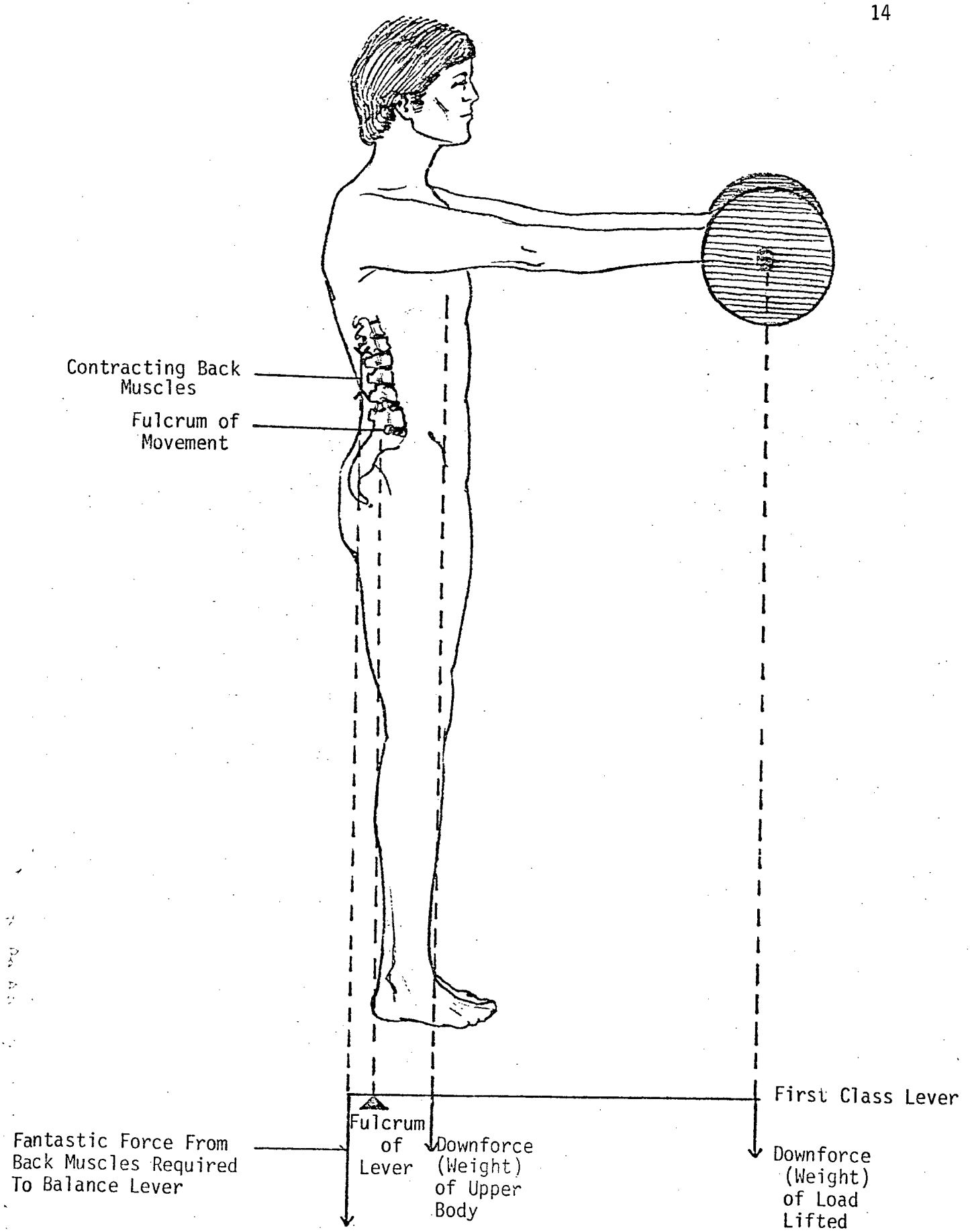


FIGURE 5. Biomechanics Involving the Back Muscles

pulling force of 650 pounds. The forces operative on the lumbar discs are maximal at the L4 and L5 levels (Nachemson & Morris, 1964), this being the instant centre of rotation of the body (DePalma & Rothman, 1970) in flexion/extension. For example, it has been suggested that a 170-pound man lifting 200 pounds can place a loading of 2000 pounds on his L5-S1 disc, but this figure may be somewhat excessive (Farfan, 1973). However, Nachemson and Morris (1964), using a pressure transducer to measure directly intradiscal forces, have reported a loading of 220 kilograms in the third lumbar disc of a man lifting a 50 kilogram weight.

Pathology of the Lumbar Spine

There are many conditions of the lumbar spine which can produce LBP, but local inflammatory reactions, neoplasia, disorders of bone metabolism, etc., are infrequently implicated (Adams, 1962; Brown, 1977). Pain may also be "referred", the pain seemingly being localized in the back when, in fact, it originates with pathology in the pelvic or abdominal viscera.

The pathology most frequently held responsible for LBP (Brown, 1977; Hirsch, 1966; Nachemson, 1975; Rothman & Simeone, 1975) involves a decrease in the height of the disc, possibly with protrusion of the nucleus pulposus into the vertebral foramen through which the nerve roots exit, and a subsequent degeneration of other parts of the joint. The etiological process responsible for degeneration of the intervertebral discs is not totally

understood and is a topic that will be discussed further below. However, once the disc degeneration has occurred, a well documented chain of other degenerative changes is initiated. Firstly, with the decrease in disc height the fulcrum of the flexion/extension movements shifts posteriorly (White & Panjabi, 1978), and the wedge-shaped apophyseal joints are driven together so that their normal, light sliding action is destroyed. A heavily laden grinding action results which soon destroys the smooth cartilaginous surfaces of the joints and results in development of inflammation and rough, arthritic surfaces. Secondly, the vertebral bodies themselves may come in close contact, creating lips or spurs on their anterior or posterior margins (MacNab, 1971).

It would appear that pain can be produced in the degenerated joint in a number of ways. Firstly, the adult disc itself does not appear to be supplied with pain fibres (Hirsch, 1966; Parke & Schiff, 1971), but the ligaments containing the disc between the vertebrae and the capsules of the apophyseal joints are richly innervated and can be sources of pain (Frymoyer & Pope, 1978; Hirsch, 1966; Shealy, 1974). Secondly, muscle spasm, thought to reflect a splinting reflex protecting a sore joint, is often seen in the posterior lumbar muscles of patients with LBP and may be a source of pain (the topic of muscle spasm may be of great importance and will be discussed separately below). Thirdly, a protrusion of nuclear disc material and/or

the lips and spurs formed on the vertebrae can impinge on the cauda equina or nerve roots, and pain and/or motor and sensory losses then result in the peripheral area innervated by the impinged nerve. This is the pathological mechanism which has been identified as being responsible for the symptom complex known as sciatica (Mixter & Barr, 1934).

Lumbar Disc Degeneration

The temporal sequence of changes occurring during the degeneration of a lumbar disc is well known, and occurs to some degree in most people, but the etiological agent initiating the degenerative process is unknown, though "...several stimulating although uncertain explanations..." exist (DePalma & Rothman, 1970, p.175). It has been suggested that an autoimmune reaction may lead to breakdown of the intradiscal material (Bobechko & Hirsch, 1965; Naylor, 1971), but this would still require an antecedent breach of the membrane which normally isolates the disc. However, it is well known that there is a diurnal variation in disc height associated with a decrease in water content after a day in the erect position (Brown, 1971; Parke & Schiff, 1971), which strongly suggests that weight bearing on the disc causes this change. With age the water content of the disc and the disc height decreases (Brown, 1971; Brown, 1977; Hendry, 1958; Nachemson, 1975; Wickstrom, 1978; White & Panjabi, 1978), this change being associated with increased viscosity of the nucleus pulposus and derangement of

the annulus fibrosus (Ritchie & Fahrni, 1970). With these changes, the disc loses its capacity as a distensible cushion and shock absorber and the gel of the nucleus pulposus may become extruded through rents in the weakened, deranged annulus (Ritchie & Fahrni, 1970). This is the most probable mechanism of disc degeneration leading to protrusion. Trauma, that is sudden, unusually high weight loading on the spine, would appear to be an insufficient explanation of disc herniation because only about 20% or less of disc herniations are preceded by trauma (Dillane et al., 1966; Hirsch, 1966; Hult, 1954; Rowe, 1969), and even those cases of trauma are usually lifts of under 50 pounds (McGill, 1968). Such lifts may well be the "last straws" precipitating rending and extrusion of already-degenerated weakened discs.

There are numerous additional facts which lead to the inference that prolonged weight loading leads to degeneration of the disc. Firstly, a primary factor leading to disc degeneration is probably the force placed on the discs by the mechanics of man's erect posture, as it has been demonstrated that quadrupeds forced to assume this posture develop disc lesions that they would not otherwise develop (Yamada, 1962). Secondly, as noted previously, the maximal forces in the human spine are operative at the L4-L5, and L5-S1 levels, and it is at these two levels that 96% of all disc protrusions occur (DePalma &

Rothman, 1970). Thirdly, Fahrni[3] has pointed out that biomechanical considerations indicate that the major forces act on the posterior aspects of the lumbar discs, and it is there that the vast majority of breaches of the annulus occur.

The evidence concerning the association between heaviness of work and the occurrence of LBP and lumbar disc degeneration is ambiguous. Swedish researchers appear not to have found an association between heavier work and increased back problems (Hult, 1954; Nachemson, 1975, 1976), whereas other researchers and reviewers have reported such an association (Brown, 1977; Lawrence, 1969; Troup, 1966; White & Panjabi, 1978). In comparing back problems in heavy manual workers with their incidence in office workers, however, the issue of disc loading on disc degeneration is clouded because the posture of sitting places very high, unvarying forces on the lumbar discs (Andersson, Murphy, Ortengren & Nachemson, 1979; Andersson, Ortengren, Nachemson & Elfstrom, 1974; Nachemson & Morris, 1964).

Muscle Spasm

Spasm (hyperactivity) of the back muscles is a very frequent observation in patients complaining of low back pain. Burke (1964) has stated that muscle spasm is always present in acute LBP patients, but unfortunately an error seems to have been made in citing the early electromyographic (EMG) research used

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3. Fahrni, W. H. Medical Rounds presentation, B.C.W.C.B., June 9, 1976.

to support this contention. DePalma and Rothman (1970), citing clinical studies, have also referred to spasm as "...a consistent finding". Nashold and Hrubec (1971) systematically documented back muscle spasm by clinical means in 72% of a series of over 1000 LBP patients at first hospitalization.

Muscle spasm as referred to in papers such as those cited above, is usually clinically assessed by palpation, a gross and highly subjective procedure which no doubt suffers a high error rate in differentiating abnormal activity of muscle lying under variable thickness fat pads from the "normal" muscle tightness resulting from posture and possibly also the patients' tenseness during examination. It may be the methodological shortcomings of this clinical assessment procedure that account for the varying percentages of LBP patients that have been reported as exhibiting spasm. Yet, the back muscles of many LBP patients are "...rigid and board-like" (DePalma & Rothman, 1970) even in a rest posture (Nashold & Hrubec, 1971). This observation hardly leaves open to doubt that profound posterior back muscle spasm is present in many acute LBP patients.

Biomechanically- and kinesiologically-oriented investigators have recently noted that little attention has been paid to abnormal muscle activity in LBP patients (Farfan, 1973; Fidler, Jowett & Troup, 1975). This almost inexplicable lack of investigation of such an obviously abnormal condition is possibly accounted for by the fact that the medical profession tends

to view muscle spasm as a "secondary" or protective phenomenon. That is, it is thought that any joint pain provokes a splinting response of the surrounding muscles, thus immobilizing the joint and preventing the aggravation of any lesion by further movement (Adams, 1962). Investigative effort has thus been expended in a search for "primary" causes of LBP.

The present author has been able to find only a very small number of studies in which quantitative electromyographic (EMG) measures from the back muscles of LBP patients have been used to study spasm or abnormal activity, especially in asymptomatic patients. Several studies concerned with this topic were recorded in a series of almost incomprehensible English abstracts of Japanese research (Itami & Hasegawa, 1968; Miyazaki & Sakou, 1968; Yamaji & Misu, 1968). Those investigators, however, appeared to conclude that, as compared to normal subjects, LBP subjects showed higher back muscle tension with various movements and in various static postures. In recent research, Jayasinghe, Harding, Anderson and Sweetman (1978) found that with prolonged standing LBP subjects showed increases in posterior back muscle EMG, whereas normal subjects showed EMG decreases. It should be noted that many EMG studies of back patients can be found in the literature, but these studies involve the qualitative diagnostic use of EMG measures for the detection of denervation of muscle groups by impingements on the nerve roots at the spinal level.

Additional indirect evidence of increased tonus in the back muscles of LBP patients is available in the English literature. It has been a frequent observation that LBP patients show a decreased lumbar lordosis (Farfan, 1973; Nashold & Hrubec, 1971; Wing, 1972), and a biomechanical analysis has shown that tightening of the posterior back muscles flattens the lordosis (Farfan, 1973). Fidler et al. (1975) have shown that the ratio of tonic (slow) muscle volume to phasic (fast) muscle volume is higher in the back muscles of patients with a history of LBP than it is in normal subjects. One explanation of this may be related to the process of hypertrophy resulting from excessive use.

It is evident, in that everyone has had the experience, that prolonged, greatly increased activity of a muscle group leads to feelings of stiffness and pain. This pain appears to arise from the pull of the muscles on their periosteal attachments (Adams, 1962) and from a decrease of blood circulation, leading to an ischemic state with accumulation of metabolic waste in the tensed muscles (Farfan, 1973). Robard (1975) has suggested that pain is produced after prolonged contraction of a muscle because catabolic waste products leave the muscle cell and increase in extracellular concentration to degrees at which the stimulation threshold of adjacent nerve fibres is reached and surpassed. One would thus expect that the extreme spasm accompanying LBP would in many cases itself be a source of pain.

A reasonable conclusion to draw from the above review of literature is that at least some of the pain of LBP is of muscular origin and that reduction of back muscle spasm would, in itself, probably be beneficial symptomatically. Moreover, in view of the biomechanical considerations discussed above, spasm would also appear to have the potential of keeping very high force loadings on the intervertebral discs, tending to perpetuate the pain resulting from disc protrusions and the forced, grinding contact of degenerated areas of the posterior joints. Schlesinger and Stinchfield (1950) have suggested that it is probable that lumbar spasm can maintain a vicious cycle of pain → reflex spasm → more pain →more spasm → etc., and they have questioned whether spasm is purposeful (i.e., as the splinting hypothesis would suggest) or whether, in fact, it may not play a part other than that of secondary splinting.

That muscle spasm may be of some primary importance is strongly suggested by the work of a number of investigators who have brought about profound muscle relaxation in LBP patients, often with startling relief of symptoms. Hafner, James and Robertshaw (1966) pharmacologically brought about total muscle paralysis in their patients for 15-20 minutes three times per week and reported dramatic, enduring relief of LBP symptoms. These investigators did not provide their data for inspection, but they provided a conceptualization of the therapeutic mechanism underlying their results, suggesting that the muscle

paralysis removed the forces acting on the spine and thereby allowed the retreat of disc protrusions. Schlesinger and Stinchfield (1950) injected the back muscles of their patients with Myanesin, a potent muscle relaxant, and reported prompt pain relief in the patients, the relief being permanent in some. These investigators suggested that the permanency of relief was related to the degree of structural damage present in the patients at the time of injection.

Current medical treatment practice, in the search for more primary pathology, appears to pay heed to lumbar muscle spasm only in passing. Drastic muscle relaxing procedures such as those described above have not found application in treatment. Rather, Diazepam and similar medications are frequently prescribed to decrease the spasm, but it is highly questionable if such compounds have any effect beyond central nervous system depression,[4] possibly producing an effect in reducing the motivational-emotional aspects of the pain experience (Chapman & Feather, 1973). Other frequently used treatments such as bedrest and traction would also quite obviously reduce lumbar muscle activity, at least the postural phasic components. Traction, especially after the initial period during which the muscles respond with a "fighting reaction" (Schlesinger & Stinchfield, 1950), would tend to keep the patient immobile. Farfan (1973) has also suggested that the apparent occasional successes

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4. Medical letter on Drugs and Therapeutics, 1973, 15(14), 57-58.

of manipulation (chiropractors' treatment) may be due to the fact that forceful stretching causes the paravertebral muscles to relax. Various forms of heat, which are central to many physiotherapy procedures, would also appear to have some muscle relaxing and circulation improving characteristics (Adams, 1962).

The effectiveness, in terms of muscle relaxation, of the above medical conservative treatments is, however, a moot point given the mediocre efficacy of these procedures in ameliorating LBP.

Conservative and Surgical Treatment of Low Back Pain

Introduction

Dr. W. J. McCracken, executive medical director of the Ontario WCB, in an address to a LBP seminar at McMaster University, provided a rather curt summary of the present status of LBP treatment (Lee, 1976). The Ontario WCB is planning to limit treatment given in LBP cases, because "Treatments are many, cures are few", despite the prescription of enough chemical medication to toxify Lake Erie, despite surgery described as "...a dismal failure", despite the efforts of physiotherapists who "...have heated, cooled, vibrated, radiated, kneaded and soaked hundreds of thousands of backs for millions of hours", despite the efforts of brace and corset fitters who "...have squeezed, twisted, forced, bent and shoved untold numbers of tortured bodies into corsets, braces, and irons made of almost every known material with the possible exception of gold and

platinum", and despite the efforts of chiropractors who "...have continued to adjust thousands of spines which somehow have developed all degrees and types of misalignment problems...".

Initial Treatments of LBP Patients

As noted previously, scientific knowledge concerning LBP is very poor (MacNab, 1978; Nachemson, 1976) and LBP has "...no generally accepted pathological lesion with a scientifically applied therapy..." (Fahrni, 1975, p.93). Yet on perusal of many case histories, there would appear to be quite a common course in the illness history of most LBP patients, with a corresponding course of treatments. As noted above, most LBP is of insidious onset or associated with only minor trauma. When the patient presents to the general practitioner, he usually complains of LBP and restricted ranges of motion of the lumbar spine. Following a clinical examination to be described below, the general practitioner then almost invariably prescribes analgesics for pain relief and supposed muscle-relaxants such as Diazepam, for relief of muscle spasm. However, as noted above, there is no convincing evidence that Diazepam or other similar drugs have any significant effect in reducing muscle spasm. At this point the patient is usually also instructed to restrict his activity to varying degrees, supposedly to allow any natural regenerative processes to take place.

Clinical LBP Examination Procedures

After taking a general history of the patient's past health

and the circumstances initiating the LBP, the physician questions the patient about the location, degree, qualitative nature, temporal characteristics and ameliorating and exacerbating antecedents of his pain. The patient is then asked to disrobe and, by palpation, the physician attempts to localize the pain in the low back and assesses the presence of muscle spasm. The localization of pain is also clarified during the determination of the ranges of motion of the spine, which the physician requests the patient to demonstrate by bending forwards, backwards and sideways. The patient is also asked to delineate the areas in his legs, if any, which are perceived to be painful. Any sensory losses in the lower trunk and extremities are assessed by pricking the skin in a grid fashion while the patient reports any decrements in sensation. Any motor losses in the lower extremities are assessed by reports concerning sphincter control and by requests for the patient to perform activities and isometric efforts (the physician supplying resistance) which maximally tax specific muscle groups. A number of reflexes of the lower body are also elicited and quantitatively compared bilaterally. Finally, a number of passive ranges of motion are tested: for example, the straight leg of the supine patient is elevated by the physician. During this manoeuvre, called the straight leg raising test, the sciatic nerve begins to move in its sheath after 30 degrees of the range of motion is completed, and an abrupt onset of pain can be expected if the nerve

root is tethered or irritated by a disc protrusion.

What the physician hopes to find during this examination procedure is a series of pain patterns and localizations in the back, coupled with pain and sensory, motor, or reflex losses in the extremities which correspond to discrete dermatomes supplied by the nerve roots exiting the vertebral foramen of the painful spinal level. Anatomically-logical consistencies of positive examination findings are, however, very often lacking and may lead the physician to a diagnosis having psychological connotations. It is also possible, however, that with repeated examinations the chronic patient can be subtly shaped by the interpersonal contexts of the examination so that he will come to exhibit the reports and behaviours of the "classical LBP syndrome" (Wilfling, Klonoff & Kokan, 1973).

Apparently, general practitioners often do not perform examinations as thorough as the one described above, and many of their medical reports contain only comments generally indicating the presence of pain and perhaps some statements concerning straight leg raising and spinal ranges of motion. In the present author's clinical experience, their psychological diagnoses such as "functional overlay" are often reached by exclusion after prolonged unsuccessful treatment, and long after spontaneous recovery is probable, and can be translated to mean "I really don't know what is wrong. I haven't found an organic explanation of the patient's continuing complaints and my usual treatment methods haven't worked."

Intermediate LBP Treatment

If after several weeks the patient is still symptomatic, he is often referred to various physiotherapies. At this point a convincing organic diagnosis is most often still lacking, as is indicated by the fact that 86% of all admissions to the BCWCB Rehabilitation Clinic carry the vague diagnosis "low back sprain" (Gunn & Milbrandt, 1976), "sprain" being a term which is loosely used by most general practitioners to indicate that no gross structural damage is evident (Adams, 1962). The percentage of LBP cases diagnosed "sprain" is similar in Ontario (Brown, 1977). Physiotherapy activities appear to be broken into a number of categories as concern goals. Firstly, there are applications of heat, ultrasound, and massage, which are oriented towards reducing muscle spasm and pain. Secondly, the goals of increasing ranges of motion and mobility are furthered by the above interventions, and a graded series of exercises may also be prescribed, generally to "loosen up" the spine. Thirdly, strengthening exercises, especially for the abdominal muscles needed in the "balloon effect" described above, are given. Fourthly, the patient is often taught postures and ways of lifting which in future will place minimal forces on the low back and discs. Corsets or lumbar spine supports of various kinds may also be prescribed at this time, or at any other point during the illness history, to support the lumbar spine and restrict its movement.

Specialist LBP Treatment

If the patient continues to be symptomatic, he is usually referred to a specialist, an Orthopaedic Surgeon or Neurosurgeon and, with chronicity, a lengthy series of specialists often becomes involved. The specialist's clinical examination corresponds to the one already described and, in addition, he may elect to use a number of more elegant investigative procedures. Qualitative EMG studies may be done, documenting motor activation potentials to ascertain whether or not the motor nerves are compromised. A sedimentation rate test may also be used to ascertain whether or not there is an inflammatory process somewhere in the body, possibly the back, and oral anti-inflammatory agents may be prescribed, or steroid injections of the back may be given. Finally, if clinical signs suggest that a disc protrusion is compromising a nerve root or the cauda equina, a myelogram may be undertaken to aid in the exact localization of the impingement for surgical purposes, but it is often apparently used as a "search" technique. A myelogram consists of an x-ray taken after radioopaque dye has been injected into the subarachnoid space, disc protrusions being seen by indentations of the dye column. Myelography has approximately an 80% accuracy rate (Raaf, 1959; White, 1969).

If a disc protrusion is identified, by compatible clinical and myelographic signs, most specialists will undertake to remove the offending disc. This is most frequently accomplished by

the surgical procedure of discectomy, whereby the soft tissue overlying the posterior elements of the vertebral column is parted and separated and the vertebral canal is entered between the posterior elements of the vertebrae so that the protrusion and nucleus pulposus can be curetted. Laminectomy, that is, partial or total removal of the bony laminae, may be undertaken along with discectomy to facilitate access to the spinal canal and also to provide more space for the cauda equina and nerve roots in the degenerated joint. Possibly because of the differing definitions of success used, a wide range of success-rate figures for laminectomy/discectomy has been reported. However, rates as low as 40% (White, 1969), or even 13% (Kosiak et al., 1966), based on indices of patient function, have been reported. White, of the Ontario WCB, defined a "good" result as the patient's ability to return to his preinjury work with minimal continuing time loss because of LBP.

A relatively new procedure, chemonucleolysis, is presently enjoying much attention and has found limited acceptance in Vancouver. Chemonucleolysis is a procedure by which the nucleus pulposus of an offending disc can be dissolved by injection of the disc (using x-ray guidance of the needle) with chymopapain, an enzyme which selectively destroys the major water-binding material of the disc. The success rates of chemonucleolysis treatment appear to be approximately equal to those of discectomy/laminectomy (Norby & Lucas, 1973).

Another surgical procedure, spinal fusion, is used when x-rays indicate vertebral instability, that is, when one vertebra is seen to move in the saggital plane in relationship to the vertebrae above and below it. Spinal fusion immobilizes the vertebra by attaching (fusing) it to another vertebra with various configurations of screws and/or bone-implant bridges, often after laminectomy/discectomy procedures have been undertaken at the same session. Fusion was also apparently used as a last-ditch resort with chronic low back pain patients in past years (Adams, 1962), but in British Columbia the use of spinal fusions has declined, probably because of research undertaken locally[5] (Kokan, Wing & Wilfling, 1975). The success rate of spinal fusion has been variously reported, with figures as low as 22% having been reported for a small group of patients with ambiguous indications for surgery (White, 1969).

A recurrent finding in many studies is that the probability of a successful outcome drops precipitously with multiple surgeries in the same patient. White (1966) has rather strongly commented on this fact, noting that "...damage to their (multiply-operated patients') productive capacity is in proportion to at least the square of the number of procedures" (p.874).

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5. Morton K. S., Professor and Head, Division of Orthopaedics Faculty of Medicine, University of British Columbia, personal communication, 1974.

Psychological Aspects of Low Back Pain

The probable importance of "emotional" factors in the etiology of some cases of low back pain was suggested as early as 1911 (Chabot, 1911), and by World War II many similar clinical judgments, phrased in terms of many different personality theories, began to appear frequently in the literature. Also, many formal and informal studies documented vocational, social, marital, and personal maladjustments in LBP patients (Tunturi & Patiala, 1980; White & Panjabi, 1978; Wilfling, 1973).

The first objective documentation of the personality characteristics of LBP patients was undertaken by Hanvik (1951) in a 1949 dissertation at the University of Minnesota. Hanvik showed that LBP patients without identified spinal pathology had much higher "neurotic triad" (e.g., hypochondriasis, depression, and hysteria) elevations on the Minnesota Multiphasic Personality Inventory (MMPI) than did LBP patients with identified spinal pathology. After a slow start in the early 1970s there has been a rapid acceleration in the number of methodologically adequate psychological studies of LBP patients. Wilfling, et al. (1973), showed MMPI neurotic triad elevations to be higher in LBP patients who were more disabled, more chronic and had had more back operations. Publications of similar findings have become commonplace in the last several years.

Beals and Hickman (1972) showed that abnormally-elevated MMPI neurotic triads characterized industrially-injured LBP

patients but not industrially-injured peripheral trauma patients. These investigators showed, furthermore, that the psychologist was more accurate than the orthopaedist in predicting the outcome of LBP treatment. This superior accuracy was thought to suggest that psychological factors are heavily involved in some primary way in the LBP problem.

Wiltse and Rocchio (1975) similarly showed the superior prognostication ability of the psychologist as compared to the orthopaedist. Those authors demonstrated that there was little correlation between the organic and MMPI examination findings of LBP patients and that the hypochondriasis and hysteria scales of the MMPI could predict 36% of the outcome variance of chemonucleolysis and laminectomy treatments. Only 10% of patients with T-scores over 85 on these two MMPI scales showed lasting relief of symptoms after treatment, whereas 90% of patients with T-scores under 55 showed such relief.

In a second study, Kokan, Wing and Wilfling (1975) showed, by the use of multivariate analyses, that independent contributions to LBP disability are made by both psychological and orthopaedic factors and that the contributions of those two types of factors are roughly equal in importance in the production of LBP disability. Of the 100 subjects in this study (also reported in Wilfling, 1973), subjects who were representative of the BCWCB population having undergone spinal fusion, fully 46% showed one or more abnormal elevations on the MMPI neurotic triad.

The Kokan et al. (1975) study is important to the thesis to be presented here in that, even though there was an independent neuroticism factor identified in the study, parts of the variance of measures of neuroticism (such as MMPI neurotic triad scores) were found to load on factors reflecting organic deficits. This finding strongly suggests that there is an interrelationship between the psychological and organic pathologies in LBP, possibly related to some integrating, etiological, psychosomatic process. Unfortunately, studies to date have followed the Cartesian dualism model, and investigators have not attempted to combine the psychological and organic characteristics of LBP patients into an integrated psychosomatic model. The present study represents a step in that direction.

Psychological Treatment of LBP

The identification of psychological contributors to LBP disability has in recent years led to the development of a number of psychologically-oriented LBP treatment programs. A program developed by Fordyce and his co-workers (Fordyce, Fowler, Lehmann & DeLateur, 1968) is based on operant theory and is concerned only with behaviours indicative of pain or disability. This in-patient treatment program thus focuses on changing the patient's social and other reinforcement contingencies to promote increases in activity and social "well" behaviours and decreases in drug taking and medical attention-seeking. Fordyce does not concern himself with the intrapersonal

experience of pain or its reduction, and his patients are individuals with chronic LBP histories. The importance of learned behaviours in these chronic patients is probably greater than in acute patients with a very short disability-reinforcement history. Fordyce (1974) has, in fact, warned that his program is inappropriate for acute patients. Fordyce has published little treatment outcome data, but those which he has published (Bonica & Fordyce, 1974), as well as informally communicated data and statements concerning the demand for his services,[6] all suggest therapeutic effectiveness. The Fordyce program has been widely adopted,[7] and some very high success rates have been reported (e.g., Anderson, Cole, Gullickson, Hudgens & Roberts, 1977; Seres & Newman, 1976).

A conceptually different, psychologically-oriented LBP treatment program has been developed at Casa Colina Hospital in Pomona, California (Gottlieb, 1975; Hockersmith, 1975; Koller, 1975; Strite, 1975). The conceptual or theoretical bases of the Casa Colina Program have not been well enunciated, but the central notion appears to be that LBP patients suffer from excessive psychological and physiological tensions, which are further exacerbated by the various additional life stresses that become associated with disability. The Casa Colina model

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6. Fordyce, W. E. Personal communication, 1974

7. See abstracts of the Second World Congress on Pain, held in Montreal, Canada, August 27 to September 1, 1978.

is illustrated here in Figure 6.

In an intensive four to six week in-patient program, the Casa Colina patients are indoctrinated with the notion that they can exert self-control on their pain experience. Individual, group and family psychotherapy sessions and sexual, financial, and vocational counselling sessions are undertaken as indicated to reduce life stress. Finally, an intensive tension-reduction program, involving biofeedback and autogenic training is undertaken. With biofeedback the patients are taught, in two one-hour sessions per day, to reduce fingertip galvanic skin response (GSR) activity, and after they have become proficient in that task, they are taught to reduce forearm EMG activity. It is thought by at least one of the Casa Colina personnel[8] that tension reduction through biofeedback techniques is the most important treatment component leading to the Casa Colina program effectiveness. While it is difficult to argue with the efficacy of this program, which returns over 80% of chronic LBP patients to functionally working status regardless of initial organic or psychological diagnoses, the program unfortunately includes so many loosely conceptualized, confounded procedures that it is impossible to understand why the program works. For example, it is controversial whether or not GSR and peripheral-muscle EMG biofeedback can be used to bring about generalized

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8. Hockersmith, V. W. Personal communication, 1976.

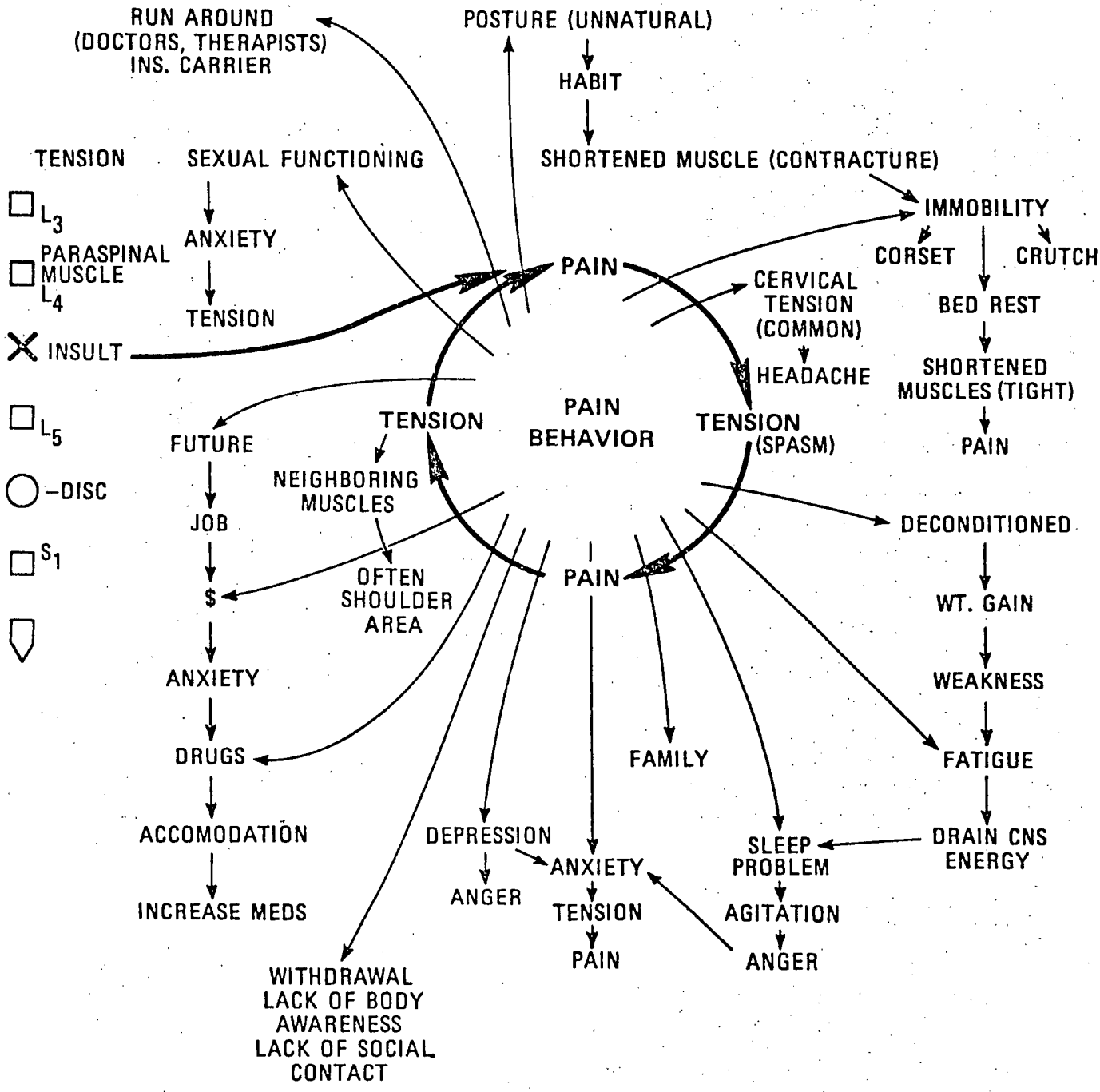


FIGURE 6. The Casa Colina "Tension-Anxiety-Pain" Model.

tension (is that anxiety?) reduction (Alexander, 1975; Stoyva, 1979).

Psychophysiological Considerations Concerning LBP Patients

There have been virtually no systematic psychophysiological studies of LBP patients, but some of their probable psychophysiological characteristics can be deduced given other known personality characteristics and the known psychophysiological correlates of those characteristics.

One early psychophysiological study of LBP, reported by Holmes and Wolff in 1952, appears to stand forgotten or unintegrated in current thought. Holmes and Wolff monitored the muscle activity in the backs and other locations in LBP patients and normal subjects, and found the former to give much greater EMG responses than the latter when confronted by social stressors. Unfortunately, Holmes and Wolff provided no data or statistical analysis in their report, and thus it is difficult to tell whether muscle groups other than the back also showed excessive responses in LBP patients. Also confusing is the fact that Holmes and Wolff called the excessive EMG activities in LBP patients a response to social stress but appear to have described the EMG in tonic terms. These investigators then conceptualized the identified excessive EMG responses in LBP patients in the "flight or fight" terminology of the day and suggested that this increased muscle activity gave rise to LBP by biomechanical mechanisms. Over the years others (Dorpat

& Holmes, 1962; Kraus, 1970; Sarno, 1978) have suggested a similar mechanism for the production of LBP, but the basis of these suggestions is unclear.

The Holmes and Wolff study cited above seems to be one of a number of investigations around 1950 in which associations between increased EMG activity and musculoskeletal symptoms were established. For example, Sainsbury and Gibson (1954), and Malmo and Shagass (1949), conducted EMG studies of patients with neck, head, and arm pains and found the EMG activity in the symptomatic areas to be higher than in other areas monitored. Similar, more recent research studies are reported by Roessler and Engel (1974), Levenson (1979), and Stoyva (1979).

One psychophysiological mechanism which would lead one to expect increased phasic and perhaps tonic muscle activity in LBP patients follows from the literature cited above, which indicates that many LBP patients exhibit neurotic characteristics. Recent psychophysiological research has quite consistently been in support of work by Malmo, who showed in the early 1950s that in various functions, including EMG activity, neurotic individuals respond to a variety of stressors with responses of larger magnitude and of longer duration than do normal subjects (Alexander, 1972; Goldstein, 1972). Less consistent has been support of the notion that neurotic individuals exhibit higher tonic levels of EMG and other physiological activities (Alexander, 1972; Goldstein, 1972), but the common lay notion

that neurotics are muscularly "uptight" has found some support in the application of EMG biofeedback relaxation techniques (Raskin, Johnson & Rondestvedt, 1973; Stoyva, 1979) and even in the application of verbal relaxation-induction procedures originated by Jacobson (1934, 1938).

The greatest difficulty in evaluating the literature dealing with the EMG response characteristics of neurotic individuals is that usually only a small number of muscles is monitored in any given study. If no differences are found between normals and neurotics, then the negative studies can always be dismissed by citing the principle of individual response stereotypy. This principle will be described below.

A Psychophysiological Model of Psychosomatic Etiology

Sternbach (1966) has integrated a number of well-established psychophysiological concepts attributable to the work of other researchers to provide an etiological model of psychosomatic disorders. Though very plausible in light of antecedent research, the Sternbach model has generated little investigation, possibly because the biofeedback boom started at about the same time as the model was proposed, which provided investigators an area of more easily-performed but more superficial psychosomatic studies. Stoyva (1979) has recently proposed a return to more comprehensive research testing such a model, and has reviewed the few such studies that have been reported.

Sternbach used the concept of response stereotypy, developed

by John Lacey, to explain organ specificity in the psychosomatic process. Lacey showed that, within one individual, the responses of various autonomically-innervated organs align themselves into a ranked order, or hierarchy, as regards their degrees of response, across a wide spectrum of different stressors. This ranked physiological response hierarchy is quite stable within some individuals over time, and those individuals are said to show individual response stereotypy. Different individuals show different idiosyncratic response stereotypies, that is, different autonomic functions will hold different ranked positions in the response hierarchies of these different individuals across stressors. Sternbach has hypothesized that the organs of the most responsive function of an individual's response stereotypy would be the first to break down if a long series of stressors was encountered and the individual's homeostatic mechanism was disinhibited. Such disinhibition is shown in the excessive (magnitude and duration) physiological responsiveness of neurotic individuals demonstrated by Malmo, and may result from prolonged periods of life stress. Potentially, the Sternbach model would appear to be applicable to the etiology of musculoskeletal disorders as well as autonomic disorders in that individual response stereotypy has been demonstrated in the skeletal musculature by Goldstein and her coworkers (Goldstein, Grinker, Heath, Oken & Shipman, 1964). Goldstein et al. showed that separate hierarchies may exist

within one individual as regards his autonomically innervated organs and his skeletal muscle groups.

The Sternbach model appears to be a very promising basis for investigations concerning the etiology of LBP. The literature reviewed in earlier sections of the present paper strongly suggests the hypothesis that abnormal, excessive lumbar muscle activity is responsible for both pain and disc degeneration in LBP conditions. It may be that LBP patients are individuals who have a musculoskeletal response stereotypy characterized by maximally responsive lumbar muscle groups. The many social, marital, vocational, and other pre-morbid maladjustments of LBP patients would make it probable that they would encounter frequent stressors in daily living, leading to frequent activation of the lumbar muscle groups. The known associations between LBP, neurotic features, and physiological overresponsiveness further suggest that the lumbar muscle responses of LBP patients are of an excessive magnitude and of excessive duration. Thus frequent, large magnitude, long duration back muscle activation would, on a chronic basis, lead to repeated force loadings on the lumbar discs, causing their untimely or accelerated degeneration.

Overactivity of the lumbar muscles as described above would pre-date the onset of LBP symptoms and if only on a chronic basis, would lead to degeneration of the discs. It is well known from the work of Rahe and others (e.g., Graham, 1972)

that periods of high stress occur in the six months preceding the onset of illness, stress which from the view of the present model would be expected to accelerate the degenerative process and perhaps even lead to the initiation of pain of skeletal and/or intramuscular origin. With the onset of overt disability, even greater stressors such as concerns about pain, surgery, prognosis, finances, interpersonal relationships, vocational future, and the like confront the individual, as indicated by the Casa Colina model shown in Figure 6, leading by the mechanisms described to even more lumbar muscle activity. Perhaps at some point in this etiological sequence, additional muscle spasm might also be created by the splinting reflex described above.

It would of course be extremely costly to conduct a prospective study concerning the Sternbach model in the etiology of LBP. However, the validity of a Sternbach model of LBP would require phasic lumbar muscle hyperactivity to be present in asymptomatic LBP patients early in their illness histories, and study of such a group would be the first step in testing the model. The implications of identifying such an etiological process for LBP will be more fully discussed below, but the potential of biofeedback for rectifying any muscle activity abnormalities should briefly be considered.

Biofeedback

If abnormalities of lumbar muscle activity are identified

in LBP patients, symptomatic or asymptomatic, another psychophysiological procedure, biofeedback, may be the most direct and rapid means of removing the abnormalities, thereby arresting the degenerative process and alleviating current pain.

Biofeedback is a clinical procedure through which an individual can learn to change the rate of activity in various of his physiological functions if he is given information via external sensory channels concerning the activity level of the function to be changed. This feedback is usually supplied by an electronic apparatus having a transducer to convert the relevant biological fluctuations to minute electrical fluctuations, an amplifier to increase the power of the electric fluctuations, and some type of output transducer such as an audio speaker or a panel meter to relay the amplified fluctuations to the subject by auditory or visual means.

The theoretical framework on which biofeedback is based developed in the early 1960s, when it was recognized that bodily functions are amenable to change by operant or reward conditioning rather than only by classical conditioning methods. Kimmel (1974) has provided a good historical account of the animal and human research leading to this change in theoretical perspective and of the rapidly proliferating subsequent clinical applications of biofeedback techniques.

Blanchard and Young (1974) have reviewed the biofeedback literature in a conservative manner and have concluded that

of all the physiological functions reported to have been modified by biofeedback, only with EMG activity is there strong evidence that biofeedback is effective. Those authors consider the effectiveness of EMG biofeedback associated with treatment of such disorders as tension headaches to be "soundly confirmed". In a review of biofeedback literature concerned with pain reduction, Roberts (1974) describes EMG biofeedback as the most promising of the biofeedback types.

Electromyographic feedback has been applied most frequently in the reduction of tension headaches and spasmodic torticollis. This literature has been reviewed by Blanchard and Young (1974), Roberts (1974), Miller (1974), Jessup, Neufeld & Mersky (1979), and others. These applications of EMG biofeedback appear to be more than vaguely aligned with the use of EMG biofeedback to reduce lumbar muscle tension in that they are frequently applied to spinal muscles, but in the cervical region. More closely related to the reduction of lumbar muscle tension is an application of EMG biofeedback reported by Jacobs and Fenton (1969), who used it to treat the cervical muscle spasms of neck-injured patients. Jacobs and Fenton found that neck-injured patients showed much higher EMG levels in the cervical spine area than did normal subjects and, that when simply instructed to do so, the neck-injured patients could not relax those muscles as well as could the normal subjects. These investigators demonstrated that with only ten, 15-second biofeedback

trials the neck-injured patients could bring their cervical EMG levels down to equal those of normal subjects. Inexplicably, Jacobs and Fenton did not report what effect, if any, this EMG reduction had on the pain experienced by the neck-injured patients, nor did they follow up on what EMG reduction, if any, remained even after a few hours.

The rate at which many subjects can master biofeedback training is very rapid and makes the potential use of such training for research manipulations and for clinical therapy very attractive. Many EMG biofeedback tasks can be mastered in under thirty minutes (Goldstein, 1972), and in the present author's experience (Hanna, Wilfling & McNeill, 1976), the technique can be taught to a subject with a few minutes of coaching.

Hypotheses of the Present Study

The present study was designed to investigate whether or not the etiology of LBP conforms to the psychosomatic model proposed by Sternbach (1966). Repeated overactivation of the lumbar muscles by stress would, by way of the biomechanical principles discussed in earlier parts of this paper, be expected to lead to acceleration of lumbar spine degeneration. If the Sternbach model is valid with respect to the etiology of LBP, one would expect the following hypotheses to be supported in an asymptomatic sample of individuals with a minimal history of LBP:

1) LBP subjects will show a greater EMG response of the posterior lumbar muscles to stress of a psychological or physical nature than do subjects with no history of LBP.

2) The LBP subjects will show a specific individual response stereotypy to stress, such that when the responsiveness of a number of physiological functions is compared, the posterior lumbar muscles will be the most responsive. Subjects without a history of LBP will not show a similar individual response stereotypy pattern, though individual response stereotypy patterns dominated by physiological functions other than the back muscles may be present.

3) Subjects with LBP will be more neurotic than subjects without a LBP history. This should be psychometrically demonstrable, especially with an instrument having a demonstrated association between elevated neuroticism scores and increased physiological responsiveness to stress. The Eysenck Personality Inventory would appear to be such an instrument (Eysenck & Eysenck, 1968).

4) Associated with the neuroticism will be a lack of homeostatic control, which will lead to a longer period until the physiological responses return to baseline levels in the LBP subjects as compared to the Control subjects.

Biomechanical discussions earlier in this paper explored the probable great importance of a hydraulic "balloon effect" of the abdominal contents in unloading forces bearing on the

intervertebral discs. This hydraulic effect is brought about by the abdominal oblique muscles (Bartelink, 1957), and it was thus decided to include the abdominal oblique muscles in the study of individual response stereotypy. However, procedures were also added to study activity of the abdominal oblique muscles in physical situations such as keeping the body erect or flexed. In both the psychological stress and postural situations, hypoactivity of the abdominal oblique muscles would adversely load the discs. The hypothesis was thus adopted that:

5) Hypoactivity of the abdominal oblique muscles will be seen in both the pyschological stress and postural manipulations of LBP subject.

METHOD

Subjects

Subjects, the majority of them being teachers, were chosen from students attending the 1977 summer session at the University of British Columbia. Faculty members in the Department of Psychology and the Faculty of Education were approached by the experimenter, who asked permission for brief access to their classes. At the classes, the experimenter provided the students with short explanations about LBP and about the methods and the measures of his research. It was also explained that, on a chance basis, one out of every eight subjects would receive $50 for participation in the research. The students were then requested to fill out a short screening questionnaire concerning demographic and LBP history information, regardless of whether or not they wished to participate as subjects in the study proper. Those wishing to volunteer as subjects could do so by filling in their names and telephone numbers at the end of the questionnaire.

Subjects were chosen for the experimental group on the basis that they reported having experienced limitation in their daily functioning and/or having visited a physician at least once in the past year because of LBP. Individuals who reported a gross pathological condition or substantial trauma accounting for their LBP, as well as those who had undergone low back surgery, were excluded from the study. Ten females and ten

males were thus chosen for the experimental group. They were then sex- and age-matched to within four years with volunteers for a control group who reported never having experienced LBP.

Materials

As described above, students were screened for participation in the study by use of a short screening questionnaire, included here in Appendix A. On presenting at the laboratory, the subjects completed a standard consent form (Appendix B). A short interview with regard to recent unusual or stressful events, any medication taken, and menstrual cycle information was then conducted by the experimenter with the information being recorded on a data sheet, included here in Appendix C. Subjects then completed the Eysenck Personality Inventory (Eysenck & Eysenck, 1972) and the McGill Pain Assessment Questionnaire (Melzack, 1975), samples of which are included in Appendices D and E respectively.

A Beckman Type R eight channel dynograph with rectilinear recording pens was used to record forearm electromyographic (EMG) activity, bilateral posterior low back muscle EMG activity, abdominal oblique muscle EMG activity, skin conductance (SC), heart rate (HR), respiratory rate (RR), and peripheral vasomotor (VM) activity at a chart speed of five millimeters per second. A marker channel was manually triggered by the experimenter to mark significant experimental events. All electrodes used in monitoring EMG, HR, and SC were of the Beckman

silver silver-chloride bipotential variety, each with a contact area .78 centimeter in diameter. The electrodes were attached to the subjects by using Beckman sticky collars. Hewlett Packard Redux paste was used to clean the EMG and HR monitoring sites as well as to provide a contact medium in the corresponding electrodes. A 0.5% NaCl paste was used as the contact medium in the SC electrodes. Forearm EMG activity was recorded in raw form with a Beckman Type 9852 coupler; three couplers of the same type, modified to function as accumulating devices (as described below), were used for monitoring the low back and abdominal muscle sites. Skin conductance was recorded by a Beckman Type 9844 coupler, which imposed a constant half volt across the electrodes and subjects, and provided a direct chart recording in micromhos. Heart rate was recorded by a Beckman Type 9857 cardiotachometer coupler, which provided a direct recording in beats per minute, on a beat-by-beat basis. Respiratory rate was recorded by following chest excursions directly with a pneumatic chest bellows attached to a pressure transducer, which provided electrical signals to a Beckman Type 9825 coupler. Digital VM activity was monitored by a reflectance photoplethysmograph incorporating a light-emitting diode and phototransistor, the signal being passed through a Beckman Type 9874 coupler utilizing a .03-second time constant.

Difficulty was encountered on initial attempts to monitor low back and abdominal EMG activity with conventional Beckman

equipment. The EMG signals derived from the back and abdominal muscles in the experimental paradigm were found to be quantitatively so small, in the order of several microvolts, that raw EMG traces or traces from an integrating coupler, such as the Beckman Type 9852, would not display them with fidelity. An accumulating type of EMG coupler, operating over several-second intervals was thus required. Satisfactory recording characteristics were finally obtained from a Beckman Type 9852 integrating EMG coupler, modified by an electronics design technician so as to function as an accumulative device in accordance with the following principles:

The raw EMG signal is a series of biphasic spike waveforms, the potentials of which may be directly recorded by the dynograph. An integrating coupler rectifies the biphasic waveforms and charges a condensor with the resultant energy, the instantaneous potential across the condensor being reflected by the dynograph tracing. The energy in the condensor is continuously "bled off" through a resistor, resulting in a time constant of trace decay. To convert an integrating coupler to an accumulating coupler, the bleeder resistor is removed and a timing circuit is added which, at regular intervals, shorts out or instantaneously "dumps" the storage condensor. However, a problem arises with regard to the principle that electrical energy is progressively harder to introject into a condensor with increases in the charge that the condensor is already storing. This problem can

be overcome to a large extent, however, by using a large condensor which, with maximal expected EMG inputs, will become charged to only a small fraction of its capacitance. Equipment for this study was altered in accordance with these principles.

The modified EMG couplers were tested by supplying them with inputs from a Hewlett Packard 3351A transmission test set with attenuator. Test inputs comprised various combinations of amplitudes from five to 25 microvolts in five microvolt steps, frequencies of 50, 100, 200, 300, and 400 hertz, and waveforms of spiked, sinusoidal and square varieties. These tests indicated good linearity between the energy content of the various waveforms imposed on the couplers and the height of the resultant dynograph traces.

In their final form, traces from the accumulating couplers took the shape of sawtooth waveforms, with ascending curves reflecting a buildup of stored energy, terminated by abrupt vertical drops of the traces back to constant baseline levels, corresponding to the shortings out, or "dumpings", of the storage condensor. The dumping interval was finely adjustable and highly stable with reference to the chart speed, and was chosen as two seconds. Electrical noise inherent in the dynograph contributed considerably to growth of the accumulators' sawtooth traces, because of its summation over a considerable time period. Testing of the dynograph and modified couplers over periods of hours, however, showed the inherent noise in the

three channels to be constant after a ten minute warm-up period of the equipment, though the noise levels of the three channels were quite different. Precautions were thus taken to warm up the equipment for at least one-half hour before running a subject and, in addition, reference traces without input from the subject were obtained at the beginning and end of each dynograph chart. The EMG activities of the subjects were scored as height differences above the sawtooth height resulting from inherent electrical noise alone, a scoring task which was greatly simplified by the fortunate stabilities of both the baselines of the sawtooth waveforms as well as the inherent instrument noise.

All psychophysiological recording took place with the subjects inside a soundproof, electrically shielded room. Subjects were seated on a common chrome and vinyl office chair, with a seat 16 inches above the floor, unpadded arms seven and three-quarter inches above the seat, and a slightly angled back extending 15 inches above the seat.

All instructions and stimuli presented to the subjects during the experimental session were tape recorded and reproduced by a Sony TC355 tapedeck through a loudspeaker. One of the experimental tasks required the subjects to play "Pong", a hand-eye co-ordination game similar to tennis, played on a small TV set. A Ridgewood Gamatic 7600 unit was used, set to slow speed, "autoserve", and a 40^{o} deflection angle. The Pong

display was a 12-inch black and white TV set placed four and a half feet in front of the subjects. The size of the Pong bat or paddle was changed from large to small by the experimenter at the midpoint of the six-minute task, and he was also responsible for resetting the game to zero score whenever the final score of 15 was reached.

In the course of the experiment the subjects were requested to immerse their hands in ice water, a procedure widely known as the Cold Pressor Test. The apparatus for this test was a one-gallon insulated beverage container, as is often used on picnics, which had a four-inch hole cut in its top. Standardized quantities of water and crushed ice placed in this apparatus led to an equilibrium temperature of four degrees centigrade after ten minutes, which would be maintained for several hours.

Procedure

A subject chosen for the study was telephoned and given an appointment time, at his convenience, for a two-hour session in the psychophysiology laboratory. On presenting at the laboratory, the subject was asked to pick randomly one of a group of manilla envelopes which assigned a subject number, and which also contained either a fifty dollar bill or a thank you note. The envelope was opened at the end of the experimental session. The subject was then asked to sign the consent form before the previously described interview form was completed by the experimenter. It was not found to be necessary to reject any

subject because of current LBP symptoms or recent substantial intake of medication. The subject then completed the Eysenck Personality Inventory and the McGill Pain Assessment Questionnaire. A control subject, who of course would not have experienced LBP, was asked to complete the latter questionnaire "as if" he had had LBP, drawing on his understanding and observations of the LBP experience as he had heard it described or seen it manifested in others.

Following completion of the above "paper work", the subject was given a brief orientation tour of the psychophysiology laboratory for the purpose of allaying any unnecessary apprehensions concerning the electrical equipment or procedures. He was then requested to go to a nearby washroom to wash his hands thoroughly to facilitate the recording of SC. The subject was also told that he would not have further access to such a facility for about one and one-half hours once the physiological transducers were attached.

On returning to the laboratory, the subject was taken into the experimental chamber, and the electrodes and transducers were attached. Because HR and EMG monitoring sites were located under the garments, a female laboratory assistant hooked up all female subjects, while the male experimenter hooked up all male subjects. Routine testing of the interelectrode resistances was not undertaken with the experimental subjects because of a lack of suitable equipment. However, practice

prior to the research showed that the cleansing technique used consistently resulted in interelectrode resistances of under 5,000 ohms, as measured by the available ohmmeter (which rapidly polarized the electrodes).

Lumbar EMG electrodes were attached bilaterally, three centimeters from the midline of the body, on the transverse plane between the L4 and L5 spinous processes. A second pair of electrodes was placed five centimeters superior to the previous electrodes. Vertically in-line pairs of electrodes were then connected to the dynograph input cables, resulting in the two erector spinae muscles being individually monitored. Electrodes to monitor the abdominal oblique muscles were attached parallel to the sagittal plane of the body, one-third and two-thirds of the distance between the anterior superior iliac spine and the lowest rib. The forearm EMG electrodes were attached to the nondominant forearm in the manner described by Lippold (1967). The HR signal was derived from "chest leads", with a reference and an active electrode placed on the anterior midline of the chest, and a second active electrode placed under the left axilla. The SC electrodes were fastened to the volar surfaces of the middle phalanges of the first and second fingers of the subject's nondominant hand. The photoplethysmograph used to monitor VM activity was taped to the middle phalange of the ring finger of the same hand. A pneumatic bellows was fastened around the subject's lower chest to monitor chest

excursions as a measure of RR.

After being seated, each subject was asked to keep both feet on the floor and not to shift around more than necessary for the duration of the experimental session. The rotary rheostat control for the TV Pong game was taped on the arm of the chair corresponding to the subject's dominant hand, and each subject was given a short practice session to familiarize him or her with the game and the functioning of the associated equipment. The Cold Pressor Test apparatus was placed immediately below the Pong control rheostat, and brief instructions were given to the subject concerning how to immerse his hand in it. Finally, instructions were given (and demonstrated by the experimenter) with regard to the experimental tasks involving forward flexion of the upper body and the increase of intraabdominal pressure (Valsalva manoeuvre).

The experimenter started the tape recorder and undertook final calibration of the dynograph after leaving the subject in the experimental room with instructions to relax. With starting of the tape recorder, all further instructions and stimuli presented to the subject were thus automatically timed and kept standard. A 100 db., 500 hertz tone, rising from zero to maximum loudness in its .2 second duration (TONE), was delivered to the subject after an initial 15 minutes of silence. To allow the subject to return to prestimulus psychophysiological activity levels, three and one-half minutes of silence followed

before instructions for a cognitive interference task (COUNT) were presented. The subject was told to remember three words (apples, loyalty and turquoise), and was then asked to count backwards by threes as rapidly as possible, starting from 518. After 30 seconds of counting, the subject was asked to recall the three words. Another period of silence of three and a half minutes duration followed before instructions for the Pong game (PONG) were given. The PONG task continued for a total of six minutes, with the experimenter switching the machine from large to small paddle size at half time, cued by the taped comment to the subject "Let's make it a little harder now". During the PONG task the experimenter monitored the TV playing screen through a peephole in the experimental chamber, reset the PONG master control when each game of 15 points was completed, and marked that occurrence on the dynograph chart. Five minutes of silence elapsed after the PONG task before the subject received recorded instructions to place his hand into the ice water (COLD PRESSOR). After three minutes of immersion he was instructed to remove and dry his hand. Six more minutes of silence elapsed before instructions were presented, instructing the subject to get up carefully and stand comfortably with his hands at his sides (STAND). Three and one-half minutes later the subject was instructed to perform the Valsalva maneouvre (VALSALVA) for 15 seconds ("Take a deep breath, hold it, but really blow hard - act like you are trying

to blow out but can't"). Another three and one-half minutes followed before the subject was instructed to flex his upper body about 45^{o} at the hips (FLEX) and to hold that position until occurrence of a further taped instruction to straighten up 15 seconds later. A further three minutes of silence then elapsed before the announcement was made that the experiment was over. The experimenter obtained a short record of the EMG accumulator coupler traces without input from the subject before switching the dynograph to standby mode and entering the experimental chamber.

All electrodes and transducers were removed from the subject, he was debriefed and shown his dynograph record if interested, and he was then asked to open the manilla envelope he had chosen on first entering the laboratory. A subject who found a fifty dollar bill in his envelope was congratulated and asked to sign a receipt.

Data Scoring

All psychometric tests were scored in the conventional manners suggested by their authors. The Eysenck Personality Inventory was scored, with the aid of templates, to yield Neuroticism (N), Extraversion (E), Psychoticism (P) and Lie (L) scores. The McGill Pain Assessment Questionnaire was scored with regard to adjectives used to describe pain, the Number of Words Chosen, Sensory, Affective, Evaluative, and Miscellaneous values being determined.

The dynograph recordings were handscored by a research assistant having some ten years of experience in such work, and she was kept blind with regard to the experimental conditions of the subjects. A rescoring of a random 15 percent of these records by the experimenter showed almost perfect agreement.

For purposes of scoring, all psychophysiological recordings were considered in intervals of 30 seconds. Traces from the three accumulating EMG couplers, which showed resets every two seconds, were scored for the total height of the 15 waveforms attributable to subject activity. The baseline heights of these waveforms attributable to electrical noise inherent in the equipment (discussed in Materials section) were ignored and the subject-related increments in the waveform heights were scored to the nearest one-quarter millimeter. Difficulties with electrical gain led, after scoring and preliminary analysis, to omission of data from one of the two channels of information from the back muscles. Insensitivity of this channel frequently did not allow changes in EMG activity to be discernible. Also, the raw EMG trace related to forearm activity was found not to be scoreable because of a constant equipment malfunction. Skin conductance and HR, their respective values in micromhos and beats per minute being directly available from the dynograph charts, were scored within each 30-second interval for numerical mean, maximum, and minimum values. Respiratory rate was scored to the nearest one-half cycle per minute within each 30-second

interval, by inspection of the roughly sinusoidal tracings, directly reflecting chest movements, seen on the dynograph charts. Vasomotor activity was scored to the nearest millimeter of trace height of each of the sawtooth-like waveforms displayed on the dynograph record. Scores within each scoring interval were then averaged. A great deal of missing VM data was encountered with the COLD PRESSOR experimental manipulation and the traces became unusable after the subjects stood up. Thus, no VM data is available for the COLD PRESSOR, STAND, VALSALVA and FLEX experimental manipulations.

The dynograph charts were scored for the following time periods: the last two minutes before TONE, and for minutes zero to one and two to three following it; during the cognitive interference task (COUNT) and for minutes zero to one and two to three after it; during the PONG task (twelve 30-second intervals) and for minutes one to two and three to four after it; during the COLD PRESSOR (six 30-second intervals) and minutes one to two, three to four, and five to six after it; after STANDing up, minutes zero to one and two to three were scored; during the VALSALVA manoeuvre only one ten-second interval was available for scoring, and minutes zero to one and two to three after it were scored, and, during FLEXing the upper body forward, three five-second scoring intervals were scored, with minute two to three following it. In order to make the data scored during VALSALVA and FLEX compatible for statistical analysis

purposes with all the other experimental data, which were derived from 30-second intervals, the 10-second VALSALVA values were multiplied by three and the three five-second FLEX values were summed and multiplied by two.

Statistical Analyses

Two sets of analyses were carried out using the statistics described below, the first comparing males and females, the second comparing the LBP experimental group to the control group.

Psychometric as well as "incidental" data were compared across groups using multiple t-tests. The acceptable level of significance was set as $p < .01$ because of the numerous comparisons being made. In this manner the groups were compared with respect to the Neuroticism, Extraversion, Psychoticism and Lie scores of the Eysenck Personality Inventory and the Number of Words Chosen, Sensory, Affective, Evaluative, and Miscellaneous indices of the McGill Pain Assessment Questionnaire. Other comparisons involved the height and weight of the subjects and total scores obtained during the PONG game. Some subjects withdrew their hands from the COLD PRESSOR before being instructed to do so after three minutes of immersion, because they found the pain intolerable. Between-group comparisons of the number of subjects showing such lowered pain tolerance were made using Chi Square.

All other analyses were conducted on the University of British Columbia Computer using programs available in the

Michigan Interactive Data Analysis System (MIDAS) package (Fox and Guire, 1976).

Several major hypotheses of the present study related to differences in the shapes of psychophysiological stressor response curves. Certain characteristics of such data require that considerable caution be exercised with regard to the statistical procedures used in their analysis. That is, it is highly unlikely that a series of data points sampled on a psychophysiological response curve are independent of one another because of the latencies and continuities brought about by the arousal and homeostatic mechanisms inherent in physiological activity. Because of this, the values of data points close together in time will tend to be more highly correlated than the values of data points more remote from each other in time. Therefore a covariance matrix of a series of data points taken across psychophysiological response curves will not exhibit equal values in all off-diagonal cells. Such a covariance matrix does not exhibit compound symmetry and thus statistical procedures which rely on this assumption, as for example, repeated-measures analysis of variance (Winer, 1971), should be avoided in analyses of psychophysiological response curve data. Profile analysis, described by Morrison (1976), has no requirements regarding compound symmetry and it is thus well suited to the analysis of psychophysiological data such as those of the present study.

With profile analysis, which is available in the MIDAS package, the curves of two groups of subjects to be compared are statistically examined in three independent ways (see Harris, 1975): Firstly, with regard to the parallelism hypothesis, the parallelism of the two curves is tested with Hotelling's T^2 and $F = (N_1 + N_2 - p)/ (p - 1) (N_1 + N_2 - 2) T^2$, with $(p - 1)$ and $(N_1 + N_2 - p)$ degrees of freedom, where p equals the number of data points monitored in each curve and N_1 and N_2 equal the number of subjects in the first and second groups respectively; Secondly, with regard to the levels hypothesis, the separation of the two curves is tested with a univariate t, comparing the sampled data points of the two groups, with $(N_1 + N_2 - 2)$ degrees of freedom (notation as above), and; Thirdly, with regard to the flatness hypothesis, whether or not the two curves differ significantly from straight lines is tested with Hotelling's T^2 and $F = (N_1 + N_2 - p)T^2 / (p - 1) (N_1 + N_2 - 2)$, with $(p - 1)$ and $(N_1 + N_2 - p)$ degrees of freedom (notation as above).

As a first step in testing the experimental hypothesis concerning individual response stereotypy in LBP patients, the magnitudes of all psychophysiological responses were adjusted to reflect pre-stressor baseline activity levels, as suggested by Wilder (1962) in his description of the Law of Initial Values (LIV). The LIV notes that the magnitude of a physiological response to stimulation is a function of the

prestimulus level of physiological activity. That is, the higher the prestimulus level of physiological activity the smaller will be the increase of the physiological activity resulting from a given stimulus. Following on suggestions made by Sternbach (1966), the psychophysiological response magnitudes in the present study were corrected for the effects of the LIV by a covariance procedure (Winer, 1971) available in the MIDAS package. The LIV-corrected responses of individual subjects were then converted to both rank and standard score values across the forty subjects, at each of the experimental stressors (TONE, COUNT, PONG, COLD PRESSOR, STAND, FLEX, VALSALVA). Using these two different types of values, ranks and standard scores, two different tests of the individual response stereotypy hypothesis were undertaken.

Firstly, the rank values (across forty subjects) for each subject for each of the five psychophysiological variables (abdominal oblique EMG or ABEMG, back EMG or BKEMG, HR, SC, RR; VM was omitted because of previously described missing data) were averaged across the seven experimental stressors yielding an average rank of response for each subject for each psychophysiological measure. These averaged ranks of subjects in the LBP and control groups were then compared for each of the five psychophysiological variables by use of Mann-Whitney U tests.

Secondly, the standardized response scores were used to construct profiles for each individual subject, across the five psychophysiological variables, at each experimental stressor or manipulation. The profiles of the forty subjects at each experimental manipulation were then examined for optimal, naturally-occuring subgroup profiles with a hierarchical grouping analysis (Ward, 1963) procedure available in the MIDAS package. Subgroup memberships, in terms of LBP or control group origin, were then established and examined for a predominance of LBP subjects. This method of examining psychophysiological data for the presence of individual response stereotypy has also been described by Sternbach (1966).

RESULTS

Survey Population and Subject Sample Characteristics

Three hundred and fifteen summer school students, mostly teachers from Faculty of Education courses, were asked to complete the screening questionnaire. Only two declined, and of the remaining 313 students, 111 indicated their willingness to serve as subjects.

Of the 313 individuals completing the questionnaire, 202 or 65% reported having experienced LBP. The reported incidence of LBP in the population surveyed was higher among females, with 71% of the 182 females as compared to 55% of the 131 males reporting LBP (X^2 = 9.13, df = 1,$p<.01$). Eighty-seven individuals, or 28% of the surveyed population, reported that they had restricted their activities and/or had consulted a physician because of LBP. Of those having had LBP, more females reported symptoms of such greater severity, with 36% of the females as compared to 20% of males having taken such action (X^2 = 7.08, df = 1,$p<.01$). Four of the 313 individuals surveyed had undergone low back surgery.

The 111 individuals volunteering to be subjects comprised 49 men and 62 women of whom 65% and 72%, respectively, reported LBP symptoms and 20% and 42%, respectively, reported activity restriction and/or physician contact. These volunteers were typically in their early thirties, the men having a mean age of 32.1 years (SD = 7.21), the women a mean age of 30.5 years

(SD = 7.07). The men and women volunteers did not differ significantly in age (t = 0.29, df = 38).

The forty subjects were ten pairs (one subject with a LBP history, another subject without such a history) of males and ten pairs of females, the subjects of each pair being age-matched to within four years. Mean ages of the study samples were 31.7 (SD = 4.62) and 30.9 (SD = 5.91) years for males and females respectively, an insignificant age difference (t = 0.29, df = 38).

While all Control subjects reported that they had never experienced LBP, all experimental subjects reported experiencing LBP during the past year, on multiple occasions for 17 of them. The mean duration of the last LBP episode was reported to be about 14 days. Seventeen of the subjects had limited their non-occupational activities because of LBP, though only five subjects had missed short periods of time from work (maximum 7 days) because of it. All experimental subjects had consulted a physician because of their LBP, with the exception of one subject who worked in a hospital setting and felt that such an action would be futile. At some time in the past five subjects had seen a specialist, 12 had had x-rays, 10 had taken medications, and nine had received conservative treatment (physiotherapy or chiropractic) for LBP.

Effects of Experimental Stressors

Table 1 provides a summary showing the baseline activity

TABLE 1. Baseline and Response Values of Each Psychophysiological Variable for Each Experimental Stressor[1].

	ABEMG	BKEMG	HR	SC	RR	VM
TONE	40.49 46.40	19.53 22.60	76.53 86.05*	6.41 9.39*	7.29 7.64	15.59 11.14*
COUNT	41.26 65.46*	18.80 25.27	74.68 103.66*	6.46 12.43*	7.56 8.07	14.67 8.49*
PONG	37.23 68.35	21.96 32.00	74.15 92.15*	6.90 12.05*	7.43 10.95*	14.29 8.79*
COLD PRESSOR	28.05 40.20*	21.99 24.38	76.85 88.58*	7.42 9.26*	7.25 7.97	MD
STAND	26.26 106.23*	16.47 83.22*	71.90 102.95*	6.59 9.63*	7.49 8.66	MD
VALSALVA	62.44 102.67*	21.10 31.50	72.18 97.92*	6.42 10.57*	MD	MD
FLEX	52.08 17.23*	27.15 81.20*	86.97 95.24*	7.05 8.70*	7.52 8.04*	MD

[1] Upper number in each cell indicates pre-stressor baseline value: lower number indicates maximum response value: see text concerning method of choosing these values.

* Indicates that the flatness hypothesis of profile analysis across baseline, response, and recovery portions of the psychophysiological response curve was rejected at the $p<.05$ level.

MD = Missing Data

value of each psychophysiological variable prior to each of the seven experimental stressors, the maximum response value of each psychophysiological variable following the occurrence of each stressor, and an indication of whether or not a statistically significant stressor response, or change in psychophysiological activity, occurred in each instance. Appendix F provides more detailed information concerning the number of data sampling points and statistical test values with regard to the effects of each of the seven experimental stressors on each of the six psychophysiological measures. The baseline and response values presented in Table 1 are provided with the main purpose of allowing inspection of the subject arousal levels in the course of the experiment. These values, which reflect activity during 30-second intervals, are the most extreme values shown in a number of 30-second intervals sampled during both the baseline and response portions of the psychophysiological activity curves. The actual statistical tests of the effectiveness of each of the experimental stressors in eliciting significant psychophysiological responses consisted of 42 profile analyses which examined departures from linearity of curves plotted across baseline, during-stressor, and recovery-to-baseline data points.

As reference to Table 1 and Appendix F indicates, the TONE manipulation led to significant increases in HR and SC, as well as significant VM vasoconstriction. The TONE was not

accompanied by significant changes in ABEMG, BKEMG, or RR, though all showed mean increases. The COUNT manipulation produced significant increases in ABEMG, HR and SC, as well as significant VM vasoconstriction. Some mean increases in BKEMG activity and RR resulted from the COUNT task, but again these changes were not statistically significant. The PONG manipulation was effective in eliciting significant increases in HR, SC and RR, and a significant degree of VM vasoconstriction. While the ABEMG and BKEMG measures showed substantial mean increases in activity during the PONG game, these increases were not significant. The COLD PRESSOR manipulation produced significant increases in ABEMG, HR and SC and non-significant increases in BKEMG and RR. The change from a sitting to a standing position (i.e., the STAND manipulation) resulted in significant increases in the ABEMG and BKEMG measures, as well as in HR and SC, whereas a significant change in RR did not occur. The VALSALVA manipulation produced significant increases in ABEMG, HR and SC, whereas the mean increase shown in BKEMG was not significant. Because the VALSALVA manoeuvre involves holding the breath, the RR variable was not evaluated. The FLEX manipulation produced highly significant increases in BKEMG, HR, SC and RR and a highly significant decrease in ABEMG activity. This latter decrease may be related to biomechanical considerations involving the "balloon effect" discussed previously, or may have resulted from the abdominal skin and fat bunching up over

the underlying muscle in the flexed position.

Comparison of Male and Female Subjects

An initial set of analyses was undertaken to examine the differences between males and females on the entire host of study variables including demographic, psychometric, psychophysiological-baseline, and psychophysiological-response characteristics. As would be expected, the males were significantly taller ($t = 4.53$, $df = 36$, $p<.01$) and heavier ($t = 5.37$, $df = 36$, $p<.01$) than were the females.

The sexes did not differ with regard to their descriptions of LBP on the Sensory, Affective, Motivational, Miscellaneous, or Number-of-Words-Chosen indices of the McGill Pain Assessment Questionnaire. The males and females also did not differ psychometrically with regard to the Extraversion, Neuroticism, Psychoticism or Lie scores of the Eysenck Personality Inventory. The mean scores for both sexes on these personality measures were very similar to the normal population values published by the Eysencks (1972). The Eysenck Personality Inventory scores for subjects from the present study and for the Eysencks' normal population are available for inspection in Appendix G.

As compared to the men, the females of the present study had significantly higher total scores for the six-minute PONG game (females, $\bar{X} = 90.9$; males, $\bar{X} = 71.4$; $t = 3.34$, $df = 38$, $p<.01$). Also, the females demonstrated a significantly lower pain tolerance than did the males in that more of them withdrew

their hands from the ice water before the scheduled three minutes of immersion were over (14 of 20 females withdrew, 4 of 20 males withdrew; $X^2 = 10.10$, df = 1, $p<.01$).

The psychophysiological responsiveness differences between male and female subjects were examined by 42 profile analyses, performed for each of the psychophysiological variables, at each of the seven experimental manipulations. Each one of these 42 profile analyses provided statistical tests of the parallelism, levels and flatness hypotheses as described in the Method section. A summary of significance levels for the parallelism and levels tests from the 42 profile analyses comparing males and females is presented in Table 2. In review, significance of the statistical test of the levels hypothesis indicates that the curves of the two groups are widely separated, while significance of the statistical test of the parallelism hypothesis indicates that convergent or divergent trends exist between the curves. If there are statistically significant convergent or divergent trends between the two curves, the part of the curve where these trends occur can often be determined by visual inspection of the plotted curves forming part of the MIDAS profile analysis computer printout. Sample curves from the profile analyses will be presented for inspection below.

Sex differences in baseline psychophysiological activity levels, which were then maintained across the responses (i.e., significance shown by the levels hypothesis test with a lack

TABLE 2. Profile Analyses of Group Differences (Males vs. Females) for all Psychophysiological Variables at each Stressor[1].

	ABEMG	BKEMG	HR	SC	RR	VM
TONE	.02 * .23	.12 .53	.88 .68	.00* .22	.66 .71	.45 .41
COUNT	.01 * .10	.21 .11	.54 .78	.00* .00*	.17 .14	.73 .35
PONG	.03 * .29	.25 .21	.62 .75	.00* .01*	.22 .59	.70 .13
COLD PRESSOR	.45 .22	.05 .06	.78 .37	.00* .14	.57 .84	MD
STAND	.27 .67	.32 .50	.62 .66	.00* .00*	.16 .21	MD
VALSALVA	.31 .13	.24 .31	.41 .60	.00* .00*	MD	MD
FLEX	.30 .66	.91 .04 *	.60 .39	.00* .02*	.22 .17	MD

[1] Upper numbers in cells are significance levels concerning the levels hypothesis (separation of curves): lower numbers are significance levels concerning the parallelism (of curves) hypothesis.

* $p<.05$

MD = Missing Data

of significance shown by the parallelism hypothesis test), were evident in the abdominal muscles (ABEMG) in the early phases of the experimental session (TONE, $F = 5.39$, $df = 1,36$, $p<.05$; COUNT, $F = 6.03$, $df = 1,36$, $p<.02$; PONG, $F = 4.85$, $df = 1,36$, $p<.05$). Examination of the computer-printed response curves, an example of which is reproduced in Figure 7, demonstrated that the females consistently showed the greater ABEMG activities. In addition to the profile analyses, three 2 x 2 analyses of variance (male/female versus LBP/control) were carried out using the ABEMG maximum response data for the TONE, COUNT and PONG manipulations, to determine if subjects of different sex with and without LBP responded differentially to these stressors. The interaction terms from the COUNT and PONG analyses of variance did not approach statistical significance ($p = .32$ and $p = .53$ respectively). However, the interaction term from the TONE analysis reached statistical significance ($F = 4.88$, $df = 1,34$, $p<.05$). Inspection of the means data for the TONE stressor indicated that the female LBP subjects showed a much greater ABEMG response than did the female control subjects or the males.

Significant sex differences also occurred with regard to the SC variable. Wide separations between parallel male and female response curves were found in two experimental manipulations (TONE, $F = 12.79$, $df = 1,38$, $p<.002$; COLD PRESSOR, $F = 10.87$, $df = 1,20$, $p<.005$), with the females showing the higher

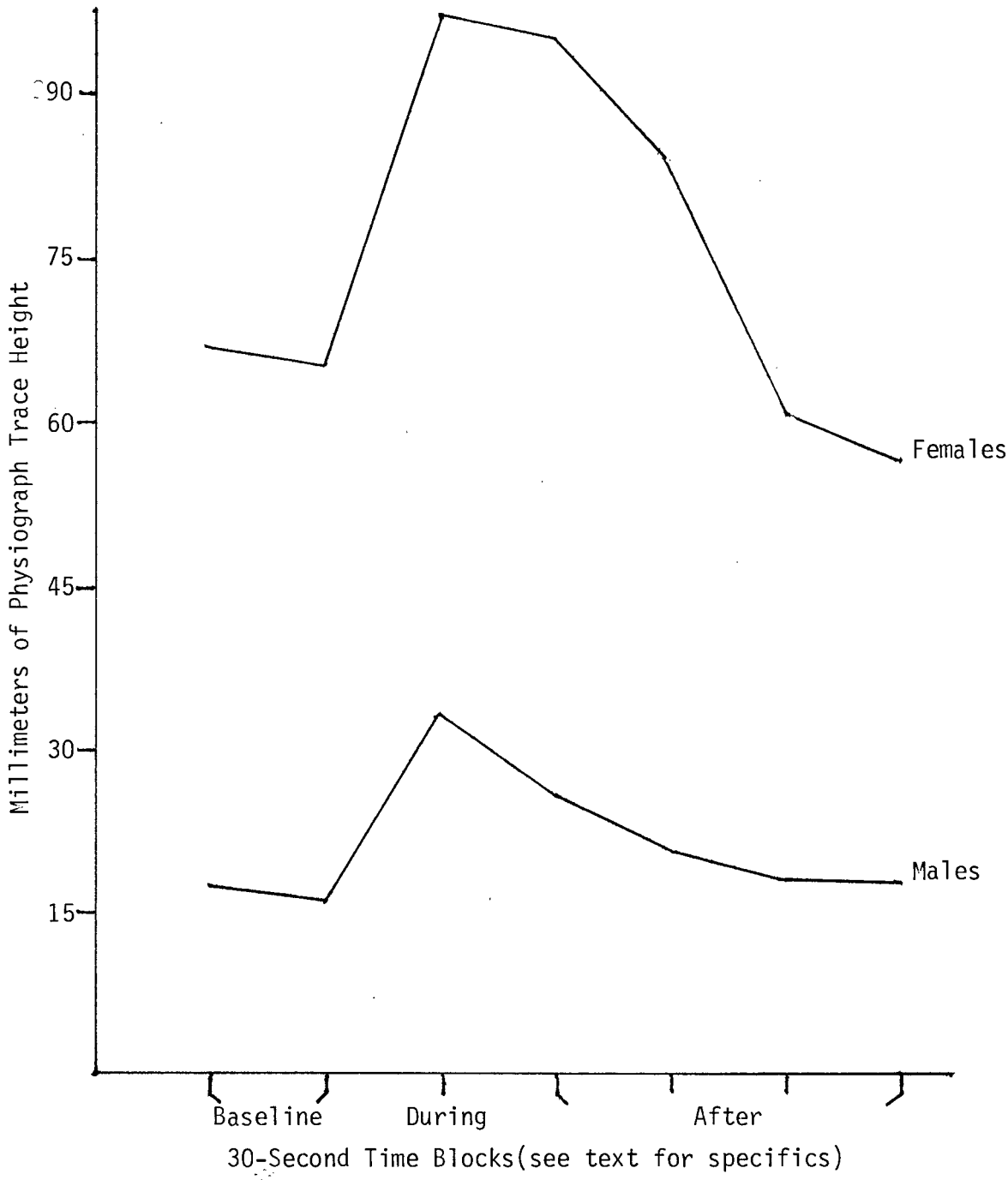


FIGURE 7. Profile Analysis Comparing Sexes with Regard to the COUNT ABEMG Response.

SC values. In the other five experimental manipulations significant non-parallelisms of the curves were in evidence (COUNT, T^2 = 35.03, F = 5.07, df = 6,33, $p<.001$; PONG, T^2 = 42.00, F = 2.81, df = 11,28, $p<.02$; STAND, T^2 = 25.12, F = 4.49, df = 5,34, $p<.005$; VALSALVA, T^2 = 25.50, F = 3.69, df = 6,33, $p<.01$; FLEX, T^2 = 14.26, F = 3.28, df = 4,35, $p<.05$), making the significant levels (or separation of curves) tests (COUNT, F = 33.18, df = 1,38, $p<.0001$; PONG, F = 28.49, df = 1,38, $p<.0001$; STAND, F = 18.78, df = 1,38, $p<.0002$; VALSALVA, F = 23.61, df = 1,38, $p<.0001$; FLEX, F = 22.16, df = 1,38, $p<.0001$) difficult to interpret. However, inspection of the computer-printed response curves, a sample of which is reproduced in Figure 8, strongly suggests that the women showed higher initial SC values and much larger responses than did the men. Three 2 x 2 analyses of variance (males/females versus LBP/controls) of the maximum SC response data from several of the experimental manipulations showed non-significant interaction effects (TONE, F = 0.88, df = 1,36; COUNT, F = 0.01, df = 1,36; PONG, F = 0.01, df = 1,36). These analyses suggest that the sex differences in SC activity were unaffected by the LBP or control group memberships of the subjects.

The analyses concerning sex differences also showed one non-parallelism of a BKEMG response curve, in the FLEX manipulation (T^2 = 11.64, F = 2.67, df = 4,34, $p<.05$), though the sexes did not differ in overall activity or separation of the

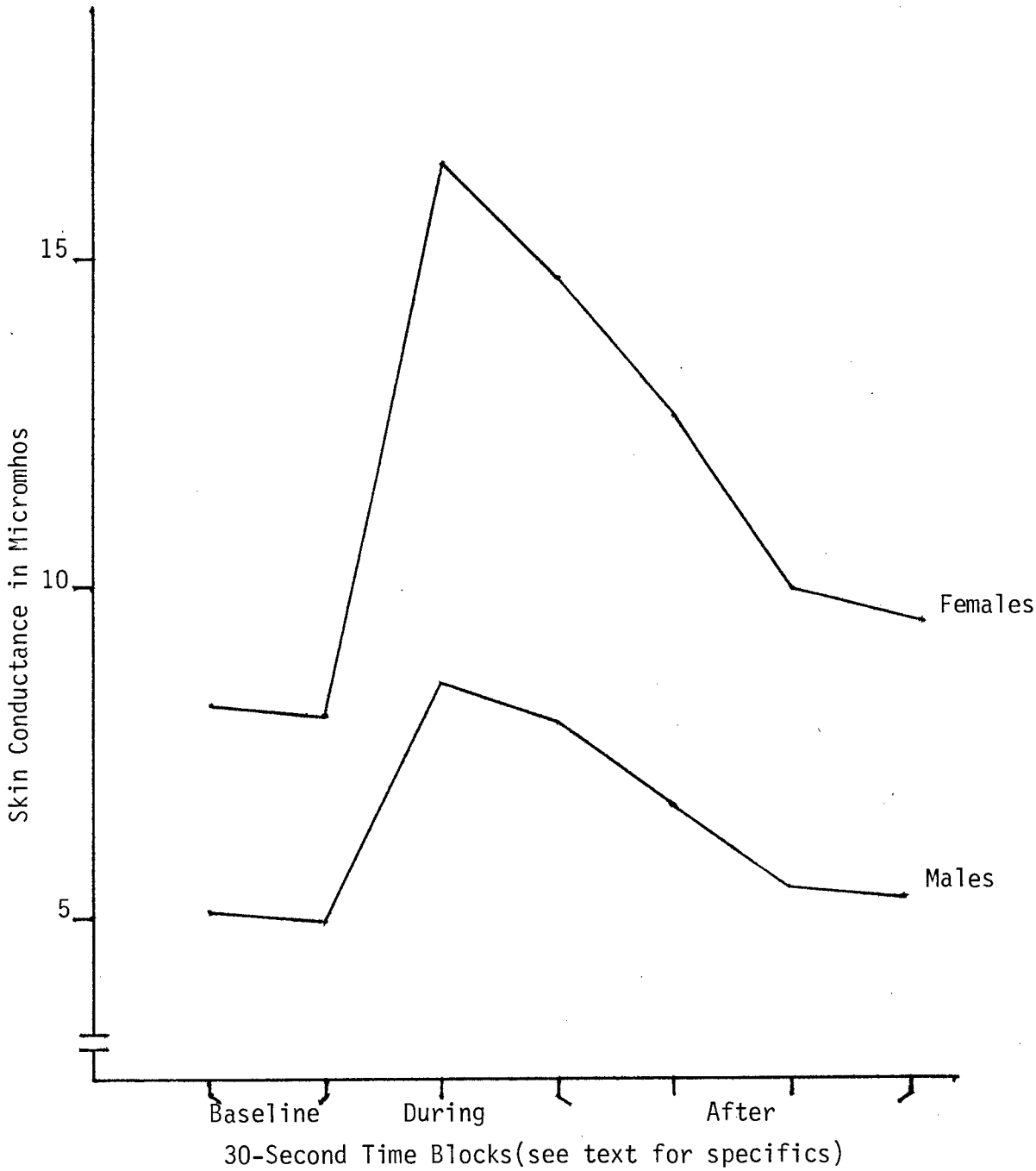


FIGURE 8. Profile Analysis Comparing Sexes with Regard to the COUNT SC Response.

curves ($F = 0.01$, $df = 1,37$). Inspection of the computer-printed BKEMG response curves for the FLEX manipulation suggests that this observation resulted from the males giving a much larger response from a lower baseline level as compared to the females.

Comparison of LBP and Control Subjects

A second set of analyses was undertaken to examine the data for differences between the LBP and Control groups with regard to all demographic, psychometric, psychophysiological-baseline, and psychophysiological-response characteristics.

The LBP and Control groups did not differ to a statistically significant degree with regard to any of the non-psychophysiological measures such as height, weight, PONG, or COLD PRESSOR performances, or in descriptions of the LBP experience on the various McGill Pain Assessment Questionnaire measures. The Control subjects, as mentioned previously, had been instructed to complete the McGill Pain Assessment Questionnaire in a manner "as if" they had had LBP. It should be specifically noted, because it bears on one of central hypotheses of the present study, that the LBP and Control subjects were psychometrically similar with regard to neuroticism as measured by the Eysenck Personality Inventory ($t = 0.57$, $df = 38$). Indeed, both the LBP and Control subjects were psychometrically very similar (with regard to all the Eysenck Personality Inventory measures) to a normal population surveyed by the Eysencks (1972). The Eysenck normal population psychometric

values, as well as mean values for the LBP and Control groups of the present study, are presented in Appendix G.

Again, profile analyses were performed to compare the LBP and Control groups with regard to the separation and parallelism of each of the six psychophysiological response curves at each of the seven experimental stressors or manipulations, yielding a total of 42 such analyses. Table 3 summarizes the significance values of statistical tests of the parallelism and levels hypotheses from these analyses.

As can be seen from Table 3, consistent differences between the LBP and Control groups emerged with regard to the BKEMG measure, these differences being in the nature of wide separations between parallel curves. These differences reached statistical significance in the cases of the PONG ($F = 5.84$, $df = 1,37$, $p<.05$), STAND ($F = 5.79$, $df = 1,36$, $p<.05$) and VALSALVA ($F = 5.68$, $df = 1,37$, $p<.05$) manipulations, and approached statistical significance for the TONE ($F = 3.29$, $df = 1,37$, $p<.08$), COUNT ($F = 4.02$, $df = 1,37$, $p<.06$), COLD PRESSOR ($F = 2.21$, $df = 1,20$, $p<.16$), and FLEX ($F = 3.29$, $df = 1,37$, $p<.08$) manipulations. Inspection of the computer-printed response curves indicated that in all cases these differences resulted from lower EMG activity levels characterizing the LBP group and higher EMG activity levels characterizing the control group. Two such curves are reproduced in Figures 9 and 10. In addition to the profile analyses, seven 2 x 2 analyses of variance

TABLE 3. Profile Analyses of Group Differences (LBP vs. Control) for all Psychophysiological Variables at each Stressor[1].

	ABEMG	BKEMB	HR	SC	RR	VM
TONE	.99 .60	.07 .48	.72 .78	.48 .11	.50 .46	.34 .36
COUNT	.83 .66	.05 .40	.42 .93	.50 .04*	.39 .54	.63 .42
PONG	.71 .80	.02 * .18	.28 .48	.44 .82	.99 .23	.75 .53
COLD PRESSOR	.30 .44	.15 .74	.94 .38	.60 .58	.91 .62	MD
STAND	.46 .74	.02* .41	.30 .66	.37 .80	.59 .93	MD
VALSALVA	.48 .59	.02* .53	.47 .51	.36 .37	MD	MD
FLEX	.45 .92	.07 .58	.29 .10	.20 .94	.84 .65	MD

[1] Upper numbers in cells are significance levels concerning the levels hypothesis (separation of curves): lower numbers are significance levels concerning the parallelism (of curves) hypothesis.

* $p \leq .05$

MD = Missing Data

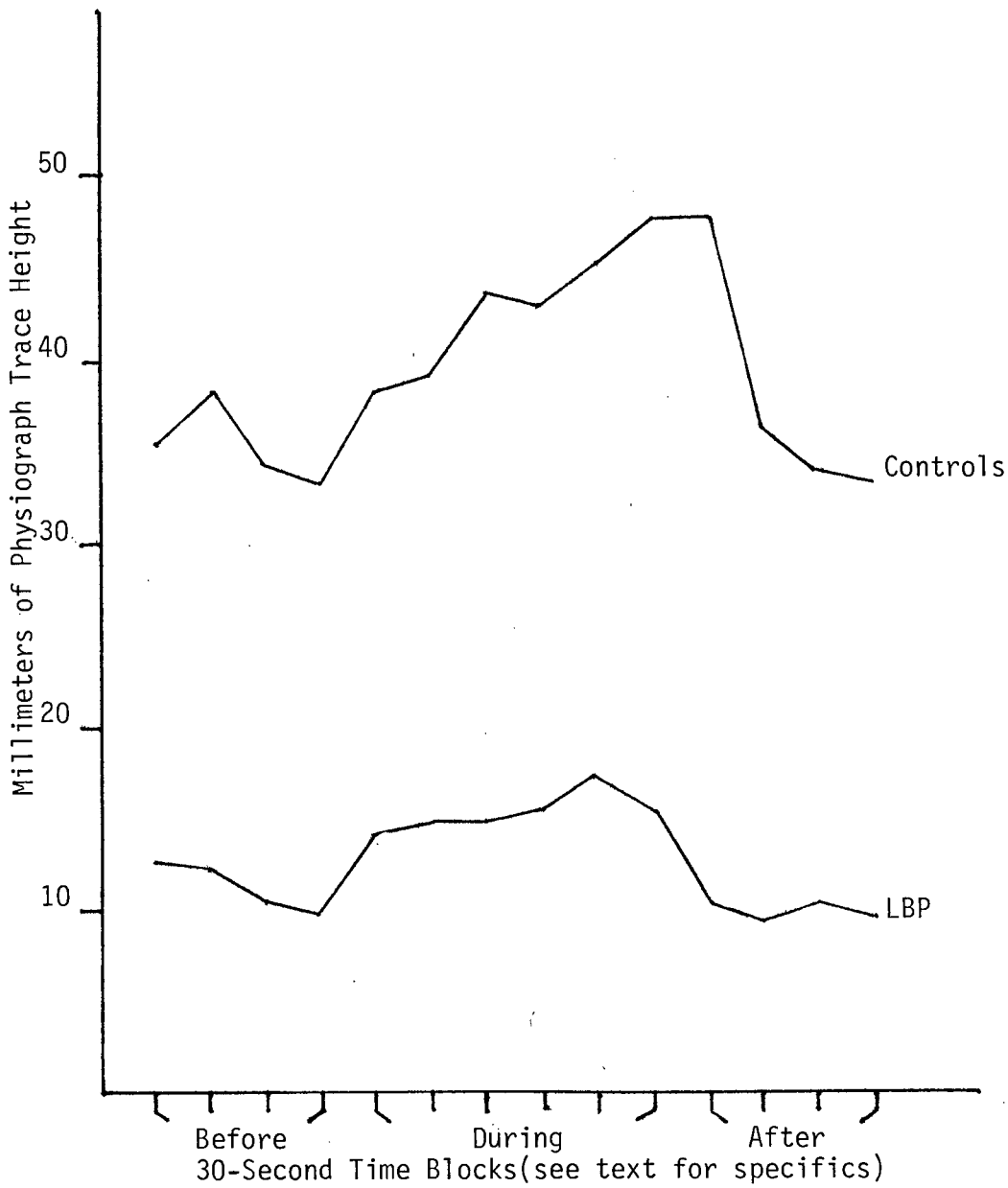


FIGURE 9. Profile Analysis Comparing LBP and Control Subjects with Regard to the PONG BKEMG Response.

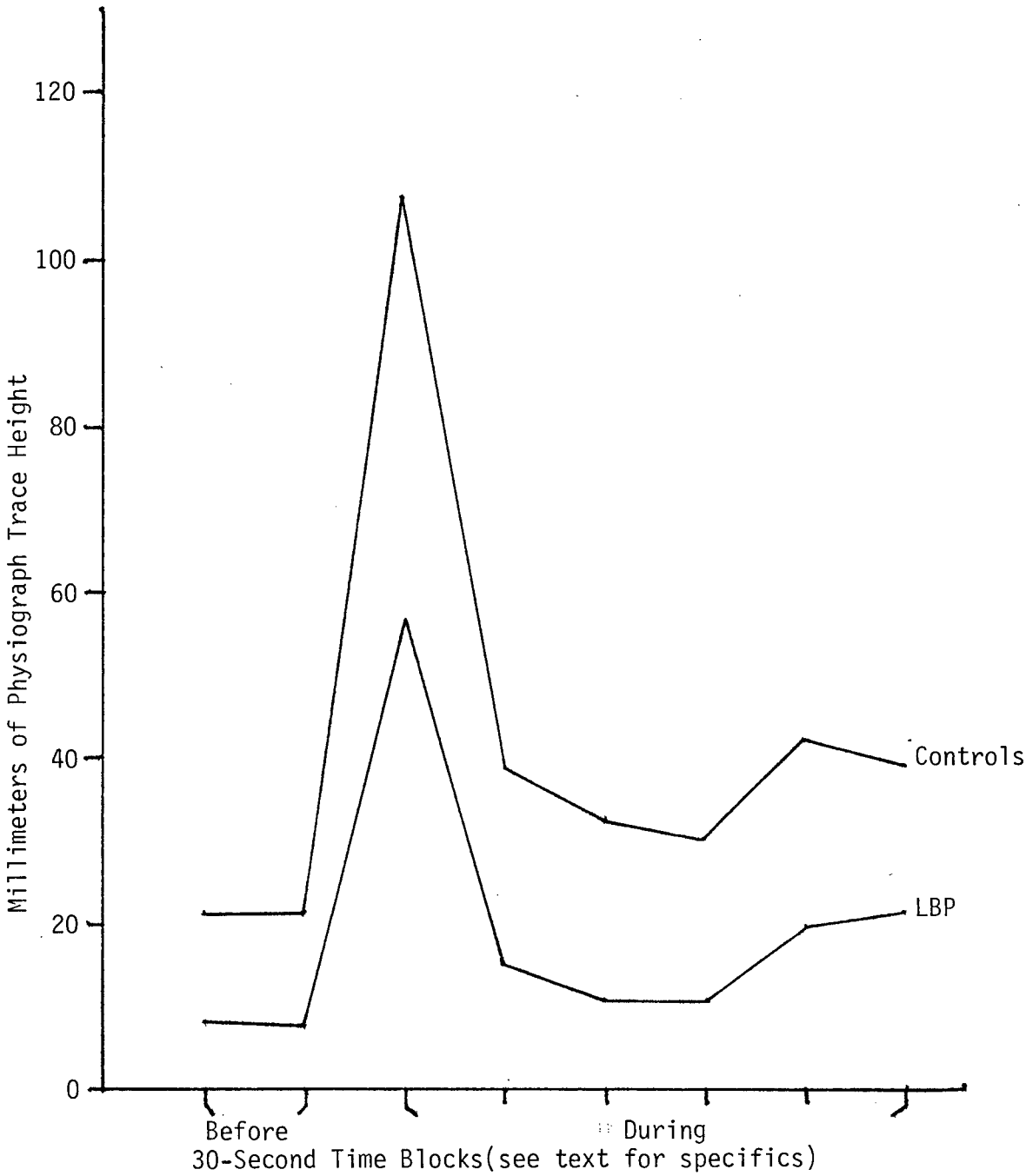


FIGURE 10. Profile Analysis Comparing LBP and Control Subjects with Regard to the STAND BKEMG Response.

(LBP/Control versus male/female) were carried out using the maximum BKEMG response data from all the experimental manipulations. The interaction terms from these seven analyses did not approach statistical significance. This would strongly suggest that, irrespective of the sex of the subjects, the low back muscles of LBP subjects show less activity than do those muscles in Control subjects.

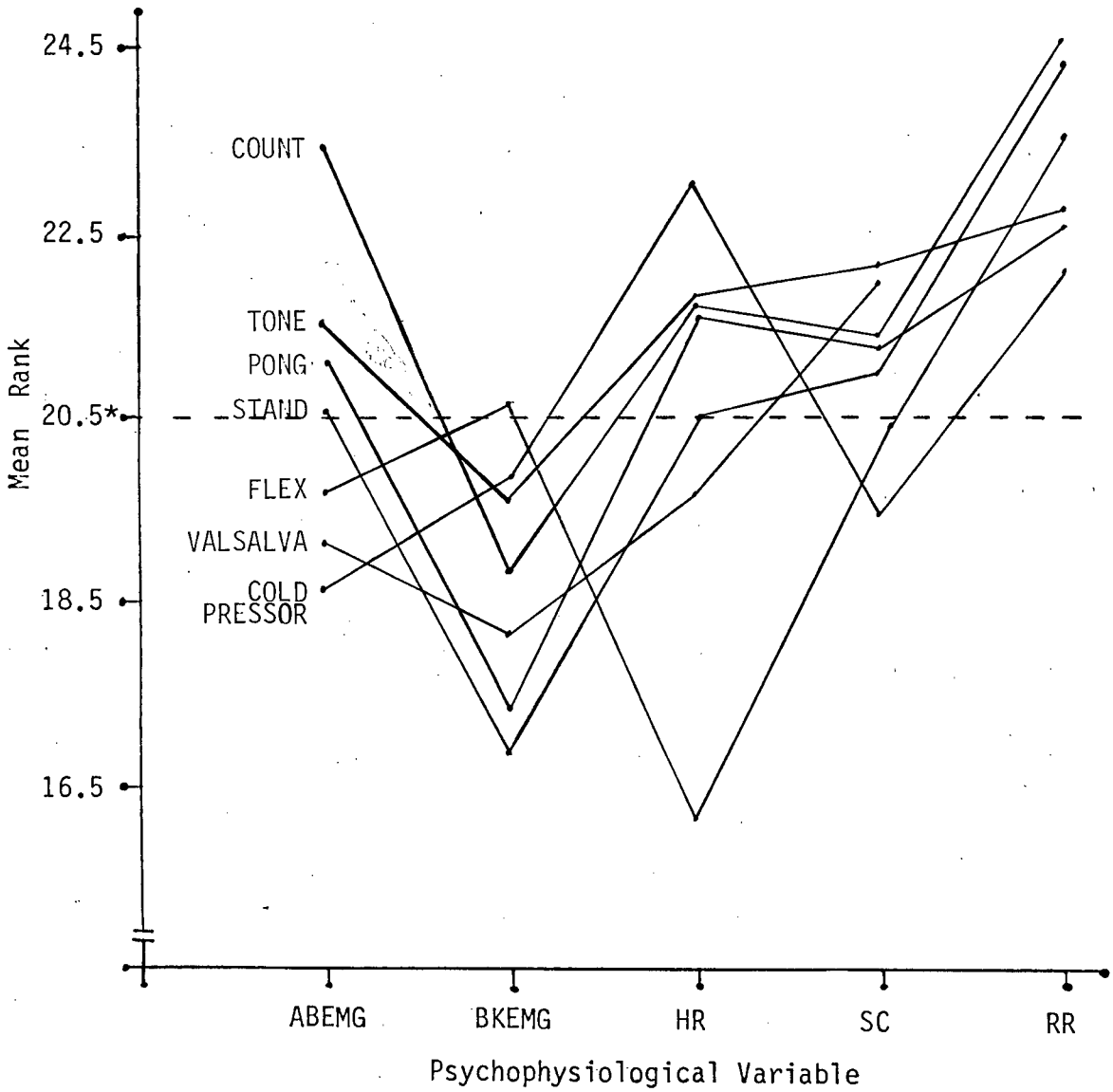
One non-parallelism, of the SC response curves of the COUNT experimental manipulation, was also evident in the comparisons of the LBP and Control subjects. While these curves were not parallel (T^2 = 16.62, F = 2.41, df = 6,33, $p<.05$) the levels hypothesis test suggests that the curves overall were not widely separated (F = 0.45). Inspection of the computer-printed curves does not make the reason for the significant observation obvious - the two curves appear parallel and close together.

Examination of Individual Response Stereotypy

As described in the Statistical Analysis section of this paper, analyses testing the individual response stereotypy hypothesis proceeded by two routes. Firstly, the baseline-corrected values for the ABEMG, BKEMG, HR, SC and RR psychophysiological response parameters were ranked across the 40 subjects at each of the seven experimental manipulations. These data are detailed in Appendix H (larger rank values indicate larger responses). The ranks for each of these psychophysiological

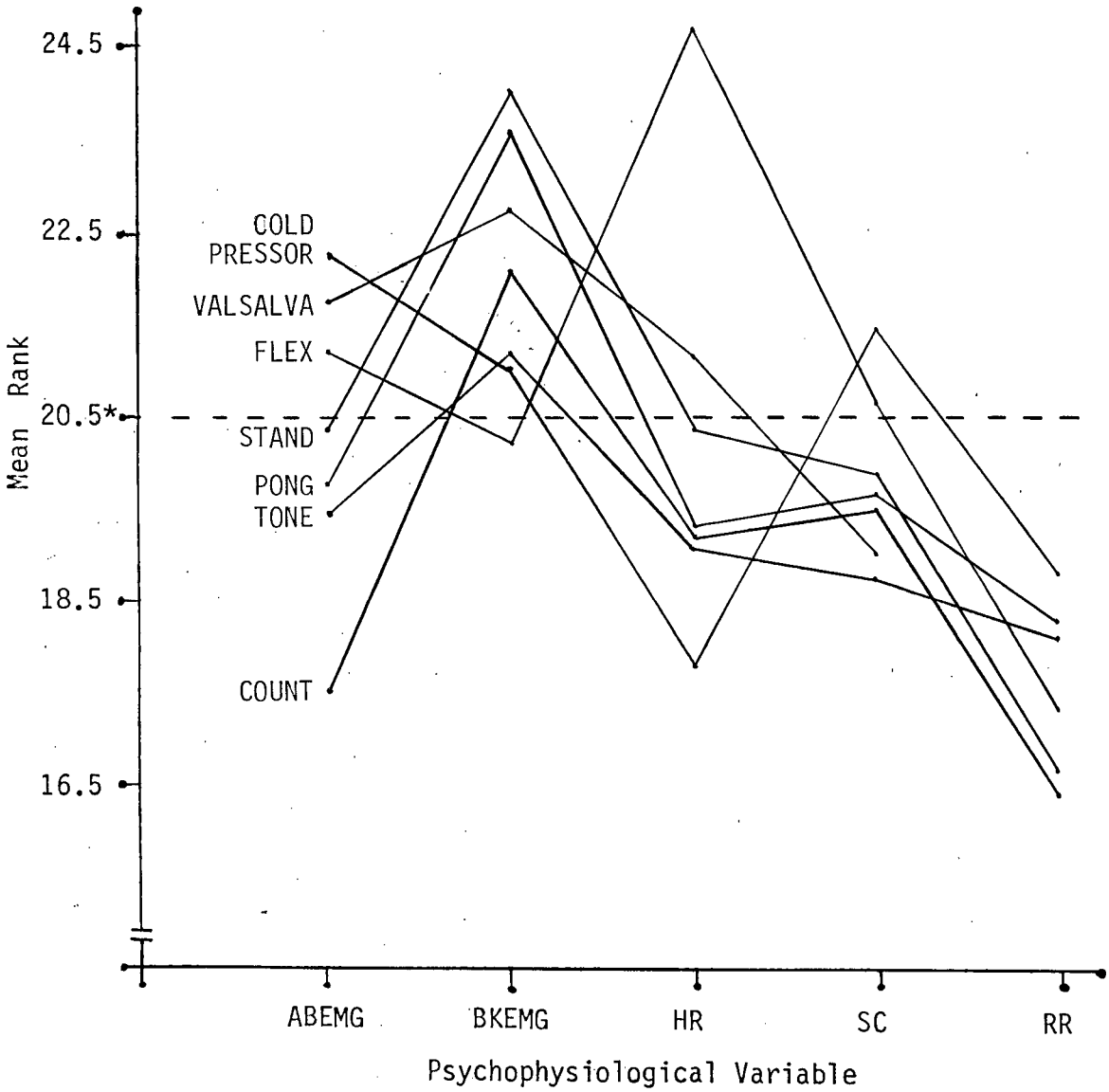
variables were then averaged across the experimental manipulations for each subject, and these averaged ranks were then re-ranked. The averaged-rank ranks are also contained in Appendix H, and graphic representations of these values for the LBP and Control groups are presented in Figures 11 and 12. When the re-ranked rank values were examined with Mann-Whitney U tests, there was no evidence of a tendency for the ABEMG, HR or SC responses of the LBP and Control groups to differ ($Z = 0.41$; $Z = 0.19$; $Z = 0.68$ respectively). The ranks of the BKEMG responses of the LBP group did show a tendency to be smaller than those of the Control group (LBP, mean rank = 17.53; CONTROL, mean rank = 22.35), but this tendency was not significant by the Mann-Whitney U test ($Z = 1.32$). The RR measure showed a statistically significant tendency for the ranks of the responses of LBP subjects to be higher than the ranks of the Control subjects (LBP, mean rank = 25.83; CONTROL, mean rank = 15.18; Mann-Whitney U test, $Z = 2.88$, $p < .05$). This suggests that the LBP subjects tended to breathe faster than the Control subjects after the occurrence of various stressors.

Secondly, evidence of individual response stereotypy was sought using hierarchical grouping analysis. Baseline-, covariance-adjusted response values for each psychophysiological measure (ABEMG, BKEMG, HR, SC, RR), at each of the seven experimental manipulations, were standardized across subjects. These standardized scores were then used to construct profiles of



* 20.5 = mean rank across LBP and Control subjects

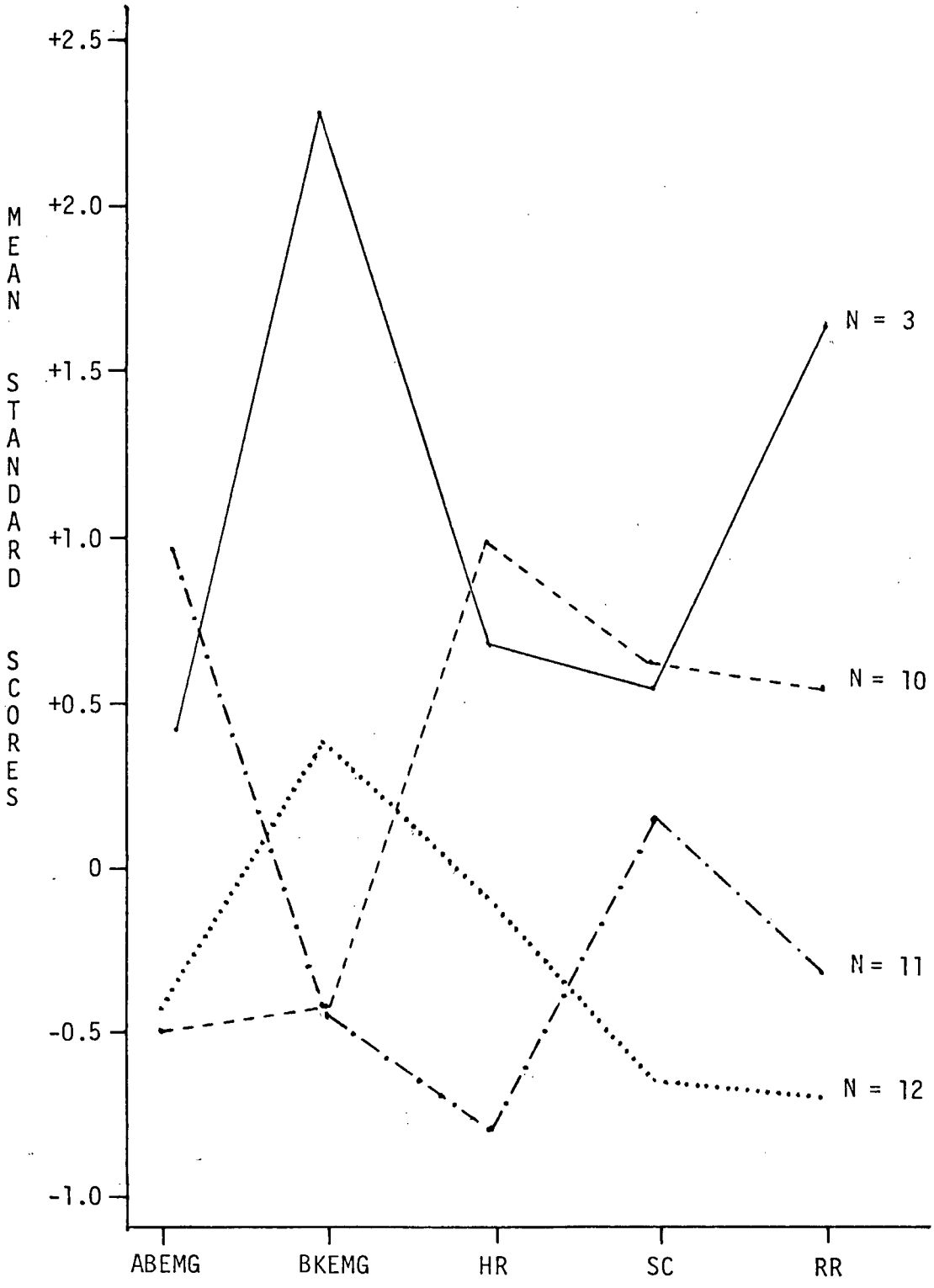
FIGURE 11 . Mean Rank Response Curves for the LBP Subjects, across Psychophysiological Variables, for all Experimental Stressors.



* 20.5 = mean rank across LBP and Control subjects

FIGURE 12. Mean Rank Response Curves for the Control Subjects, across Psychophysiological Variables, for all Experimental Stressors.

response magnitudes, across the five psychophysiological measures, for each subject at each experimental manipulation. The profiles of the 40 subjects at each experimental manipulation were then submitted to a hierarchical grouping analysis, a total of seven analyses thus being undertaken (one analysis for TONE, a second analysis for COUNT, and so forth). The naturally-occurring hierarchical subgroupings, perhaps more than one set for each of the seven experimental manipulations, were then examined with regard to subject membership (LBP versus Control subjects) and with regard to whether or not the psychophysiological response profiles were dominated by extreme values of either of the EMG measures. An example from these hierarchical grouping analyses is presented in Figure 13, wherein the mean psychophysiological response profiles of four naturally-occurring subgroups of the 40 subjects, at the STAND experimental manipulation, are presented. Table 4 provides a summary showing the naturally-occurring subgroupings of subjects at each of the experimental manipulations, the psychophysiological response extreme by which each of the mean response profiles within the subgroupings was characterized, and the overall number of subjects and the number of LBP subjects contributing to each profile. By way of example, with reference to Table 4 one can determine that, for the STAND analysis, there were 11 subjects contributing to a mean profile characterized by a very strong abdominal muscle response, five of these subjects having a



*Four subjects omitted due to missing data.

FIGURE 13. Sample of the Hierarchial Grouping Analysis Examination of Individual Response Stereotypy for the STAND Experimental Manipulation (see Table 4).

TABLE 4. Hierarchical Grouping Analysis Summary.*

STRESSOR	NO.OF GROUPS	ABEMG		BKEMG		RR		IRS OF OTHER FUNCTIONS
		HIGH	LOW	HIGH	LOW	HIGH	LOW	
TONE	5			1/2		1/1	8/19	7/13:1/1
COUNT	6	2/3		0/1			3/9	1/3:7/10:5/9
	5	2/3		0/1			4/12	7/10:5/9
PONG	6	3/4		0/1		7/12	4/11	2/5:1/1
	4			0/1		7/12	9/20	1/1
COLD PRESSOR	7	1/3		0/1	0/2		6/12	7/11:3/6:0/1
	3	1/3				3/8		13/25
STAND	6	2/5		1/3		4/5	4/12	3/5:3/6
	4**	5/11		1/3			4/12	7/10
VALSALVA	5	2/5	11/21	0/3		2/2		3/7
	4		11/21	0/3		2/2		5/12
FLEX	7	0/1		0/1		1/3		1/7:1/2:7/12:7/11
	4	0/1				1/4	8/19	8/13

*Fraction-like numbers in above Table denote the number of LBP subjects by the numerator and the total number of subjects by the denominator in each subgroup.

**See Figure 11 for graphic representation of this subgrouping.

history of LBP. It is also this subgroup of 11 subjects who compose one of the curves plotted in Figure 13.

Examination of Table 4 again provides no support for the hypothesis that EMG-dominated individual response stereotypies characterize LBP subjects, but that a RR-based stereotypy does. Again, it appears that LBP subjects tended to breathe faster after the occurrence of stressors than did the Control subjects.

DISCUSSION

The population of summer school students surveyed seems to be quite representative of the general population in the incidence of reported LBP complaints. The LBP literature generally shows the incidence of LBP to be the same across sexes (Brown, 1977; Frymoyer & Pope, 1978), though some studies have indicated a greater incidence of back complaints in women (Dillane et al., 1966). The number of individuals in the present study reporting limitation by their LBP is perhaps somewhat lower than that suggested by the maxim usually cited, "Two-thirds of people have suffered from it, one-third have been disabled by it", but this may be related to the young age of the sample. The third and fourth decades of life have been identified as the times of peak occurrence of LBP (Hult, 1954; Nachemson, 1975), and it is of course more common to have been disabled by LBP later in that period. The present study's sample of subjects (teachers) is atypical demographically, because the LBP literature usually involves samples of manual workers. However, as noted in the Introduction, there is no clear relationship between the incidence of LBP and the heaviness of work performed. However, it is often believed in clinical settings that equivalent LBP symptoms are more disabling for manual workers than for more sedentary workers (Hirsch, 1966).

Initial phases of analysis revealed sex differences in height, weight, COLD PRESSOR performance, total PONG score,

ABEMG activity, and SC. Demonstration of mean height and weight differences between men and women adds little to the scientific fund of knowledge.

Significantly fewer women than men tolerated the three-minute hand-immersion time in the COLD PRESSOR ice water bath, indicating that the women tended to have a lower pain tolerance than the men. This finding is consistent with current knowledge, based on recent research and reviews of the literature (Notermans & Tophoff, 1975; Woodrow, Friedman, Siegelaub & Collen, 1975). Perhaps the lower pain tolerance shown by women accounts for another observation in the present study; that is, that the women more frequently restricted their activities and/or sought medical attention because of LBP than the men did.

The total PONG scores were found to be significantly lower for the men than for the women, indicating that the men were more proficient at this hand-eye coordination game than were the women. Though these video games are becoming quite common, casual observation during the initial practice sessions left little doubt in the experimenter's mind that the women frequently had negative attitudes and expectancies about the gadgetry, whereas the men frequently were delighted at the prospect of playing the game and often acknowledged previous experience with very similar equipment. These attitude and practice differences, though not formally documented, are held to be an adequate explanation for the observed differences in performance.

With regard to psychophysiological parameters, the most consistent differences between sexes occurred with regard to SC, with the women seemingly showing higher initial values and greater responsiveness than the men. While there exist widely discrepant reports concerning sex differences in electrodermal activity, higher tonic SC levels have previously been observed in males (Ketterer & Smith, 1977; Kopacz & Smith, 1971). A greater responsiveness from such levels of males has also been observed (Kopacz & Smith, 1971), though an even greater variability in reports exists in this regard along with the identification of all manner of mediating influences from variables such as type of task, level of task stressfulness or difficulty, handedness of subjects, etc. In the present research, however, there may be another explanation for the observed differences in tonic SC levels. As described previously, for ethical reasons all male subjects had their electrodes attached by the male experimenter while all female subjects had theirs attached by a female laboratory assistant. Technique in attaching the SC electrodes, in conjunction with likely differences between subjects of the two sexes in available areas and curvatures of the attachment sites, may have led to measured SC differences by way of systematic electrode contact differences. In other analyses, there were no indications of SC differences related to LBP or Control group membership, or of an interaction of sex of the subjects with such group membership.

The greater abdominal EMG activity shown by women in the initial phases of the research session is more difficult to account for in that such differences have not routinely been shown for EMG measures. Where sex differences in EMG activity have been found, they appeared to be directly related to gross strength differences (Goldstein, 1972). The abdominal oblique muscles are, of course, also an unusual site for psychophysiological monitoring. However, the sex differences in abdominal EMG activity were evident only in the early part of the experimental session, suggesting that there may have been some type of habituation phenomenon operating. Perhaps the women sat more stiffly or primly initially than did the men. In other analyses performed on the ABEMG data, there appeared to be no systematic differences related to LBP or Control group status, except for one interaction between sex of the subjects and LBP status (LBP females showed higher values than other groups). This interaction occurred in analyses of data related to the first experimental stressor and, standing in isolation among other insignificant results, cannot be meaningfully interpreted.

Turning to the comparisons between the LBP and control groups, the first notable observation is the lack of significant psychometric differences with regard to the Eysenck Personality Inventory measures. Support of the Sternbach model of psychosomatic etiology tested in the present research would have required the LBP group to show higher neuroticism scores than

the control group or the normal population. In fact, both study groups were found to have slightly lower scores than those reported for a normal population by the Eysencks (1972). Beyond not supporting the Sternbach model, this observation of normal neuroticism scores characterizing a LBP population is also counter to reports in the literature, reviewed in the Introduction section, describing abnormal neuroticism scores in LBP patients. The literature reviewed, however, usually concerned clinical populations of individuals who were complaining of LBP and actively seeking professional help, often surgery, repeat surgery, or alternatives to failed surgery. It may well be that such clinical populations represent a self-selected subclass of people with LBP who continue to seek help, while non-neurotic people with LBP may tend to consult physicians a few times and then tolerate their symptoms. Alternatively perhaps, as some have suggested (Caldwell & Chase, 1977; Mersky & Boyd, 1978; Sternbach, 1977), the protracted experience of significant LBP symptoms may give rise to the neuroticism features seen in clinical populations.

Similarly, the absence of group differences with regard to pain tolerance is of interest in that observations from former research (Wilfling, 1973) with a clinical population would have led to expectations of such differences. Again, however, self-selection of a clinical population, or developing intolerance to pain with protracted experience of it, may explain the discrepancies between these research findings.

Though not bearing on a central hypothesis of the present research, it is interesting to note the lack of significant differences in the subjective descriptions of the LBP experience, as documented by the McGill Pain Assessment Questionnaire, between those who have personally experienced LBP and those who have not. In that LBP is so very common in the population, it is possible that the control subjects of the present study had observed many individuals with LBP, had heard their descriptions of the experience, and were thus able to describe LBP accurately on the questionnaire.

The psychophysiological parameters of the present study present the most interesting findings. The total absence of group differences with regard to initial baseline and response magnitude and duration characteristics of the HR, SC and VM variables certainly suggests that generalized psychophysiological differences do not differentiate individuals with a history of LBP from normal subjects. Furthermore, the lack of group differences with regard to the ABEMG activity suggests that generalized skeletal muscle activity abnormalities do not characterize individuals with a history of LBP.

The central hypothesis of the present research relates to activity of the posterior lumbar muscles. On the basis of the Sternbach model, it was hypothesized that these muscles in LBP subjects would hyper-respond to any stress, both in greater magnitude and for a longer duration before returning

to baseline values, as compared to these muscles in normal subjects. The profile analyses performed on data from the present research, however, provide no support for this hypothesis. To the contrary, the subjects of the LBP group consistently showed less baseline EMG activity in the critical anatomical region as compared to normals, with this inferiority being maintained after stress, with no gains or losses in magnitude or duration of response being shown in comparison to the response curve shapes of the normal control subjects. That is, the baseline, response, and recovery portions of the curves for the LBP and control groups were parallel but widely separated, often to a statistically significant degree.

It is difficult to believe that the observed differences between the LBP and Control groups with regard to BKEMG activity could be due to systematic biases. The experimenter's subconscious desire to support his hypothesis, if subtly manifested by quality of skin preparation for electrode attachment, by motivation-inducing differences in initial instructions, or by any similar procedural difference, would have biased the results in the opposite direction. Furthermore, the experimental conditions were standardized by all instructions and stimuli occurring during the experimental session being tape recorded, electrodes being placed by measurement from body landmarks, all physiograph records being scored by a disinterested technician blind to the experimental conditions, and so forth.

Examination of the hypothesis concerning the presence of a posterior lumbar muscle individual response stereotypy in LBP subjects led to rather meagre observations, as might have been expected by the consistent parallelism seen between the response curves of the two groups in the earlier profile analyses. After adjustment of the response magnitudes for the effects of baseline differences had been made, there was a weak (not statistically significant) indication, in the analysis of ranked responses, that the LBP subjects in fact responded less from their baseline levels than did the control subjects from theirs.

The findings of a significant tendency for LBP subjects to show greater increases in RR in response to the various experimental manipulations than the Control subjects is both methodologically and theoretically interesting. From the methodological point of view, the lack of significant separations of the LBP and Control group RR curves, as evidenced in the profile analyses, makes the appearance of significant differences in the individual response stereotypy (IRS) analyses surprising. However, response data for the IRS analyses were derived by subtracting the baseline activity value from the maximal response value and correcting the response for the LIV, whereas the data for the profile analyses consisted of multiple uncorrected activity values across before-, during-, and after-the-stressor parts of the response curves. From the theoretical

point of view, the finding that LBP subjects tend to breathe faster than Control subjects during environmental events or motor tasks is interesting in light of the biomechanical "balloon effect" described in the Introduction. To the degree that the lumbar spine is unloaded and protected by an increase in intra-abdominal pressure (the balloon effect), and this increase is interfered with or prohibited by breathing, breathing during the occurrence of environmental events or movement could increase the risk of injury to the lumbar spine.

A second way of approaching the issue of individual response stereotypy and its importance to the psychosomatic process relates more closely to the Sternbach model and addresses itself to the question of "organ specificity" of the psychosomatic process. Other conditions in the Sternbach model being satisfied, it would be the most psychophysiologically responsive organ or system of an individual's body that would sustain damage with repeated activation. This would account, for example, for why one person develops ulcers while another develops cardiovascular problems in response to repeated or prolonged stress. Pathophysiological processes stemming from hypoactive physiological systems are also well recognized, and it would thus seem advisable to examine individual response stereotypy patterns with regard both to the most and the least responsive psychophysiological functions. In this regard, of course, no support was found for either of the posterior lumbar muscle individual

response stereotypy patterns (very high or very low activity) in the LBP subjects, and the abdominal muscles also did not contribute to a LBP individual response stereotypy. However, in the results of this analysis there also appeared to be evidence of IRS, with regard to the RR variable. That is, of those subjects that showed the least increase in RR following stressors or motor tasks, a disproportionately low number belonged to the LBP group. As noted above, these RR response differences would interfere with the hydraulics of the abdominal "balloon effect" and would leave the spine poorly supported and protected against trauma.

The hypoactivity of the posterior lumbar muscles of LBP subjects, as described above, in all likelihood contributes further (in addition to the effects of the decreased "balloon effect") to poor stabilization and protection of the lumbar spine. By way of a number of well established biomechanical principles, hypoactivity of the posterior muscles would be expected to lead to destructive forces acting on the lumbar discs. The resultant pathophysiological process is probably very much more damaging than the compression-based process initially hypothesized in this research.

Briefly, the spine can be likened to a mast, rod, or beam composed of a stack of poorly joined sections (the vertebrae) which are inherently unstable or free to move in relationship to each other. Linear rigidity and weight-carrying capacity

is achieved in such a mast by guying it at multiple levels, as is done with a tall antenna mast. Just as the guy wires of an antenna allow it to remain erect when it could not do so of its own integrity, so the muscles surrounding the spine guy it and hold it erect. Such a biomechanical model of the spine has long been described (Asmussen & Klausen, 1962; Farfan et al., 1970; Parke & Schiff, 1971; White & Panjabi, 1978). In addition to the stabilizing effect of this guying, the preload placed on the spine actually stiffens it (White & Panjabi, 1978) or enhances its "beam strength" (Parke & Schiff, 1971) and in the process the articular processes are pushed together, protecting the spine from excessive rotation (Farfan et al., 1970).

Just as loosening the guy wires of an antenna mast would allow curvatures to develop in its length, ultimately leading to buckling and to the introduction of torsional and shear forces between the individual sections, so too would one expect similar forces to be exerted on poorly stabilized spines, such as those which characterized the LBP subjects in the present study. The poor stabilization would result in torsional and shear forces on the discs, which are much more destructive and likely to produce eventual degeneration of the discs than are the compressive forces implicated in the original hypotheses of this study (Farfan et al., 1970; Troup, 1966). Numerous researchers (see Farfan et al., 1970; Frymoyer & Pope, 1978; White & Panjabi, 1978; Wiltse, 1971), have shown the intervertebral disc to

be quite resistant to compression but to be very easily damaged by torsion or shear, and Wickstrom (1978) and White & Panjabi (1978) have discussed how torsional or shear forces can directly cause disruption and tearing of the long, stringy, organic molecules comprising the annulus fibrosus of the disc. The probable importance of torsional and shear forces is also evident on a clinical basis in that most acute episodes of LBP treated at Compensation Boards appear to be initiated by twisting/lifting movements (Brown, 1977). The introduction of torsional and shear forces has also been discussed in relation to the poorly stabilized spine (Troup, 1977) or a spine left poorly protected by fatigue (Brown, 1977) or sudden unexpected physical effort (Magora, 1973). Thus, a series of shearing mini-traumas to the disc may well be responsible for the accumulation of small fissures of the annulus fibrosus, fissures which lead to its gradual weakening, deterioration, and ultimate disruption by a minimal "final straw" force (Farfan et al., 1970; Ritchie & Fahrni, 1970; Wickstrom, 1978). Before the ultimate disruption of the disc, leakages of intradiscal materials through the small fissures resulting from mini-traumas can give rise to local inflammation and periods of LBP (Brown, 1971; Hirsch, 1966; Nachemson, 1975; White & Panjabi, 1978).

Another manner in which the observations of the present study seem to be important relates to Farfan's (1975) demonstration that there must exist a dynamic mechanical balancing

between the abdomen's oblique and flexor muscles and the posterior muscles of the back. If not, destructive shear forces will be exerted on the discs in lifting and even in the course of maintaining posture. It is pertinent to note that, while the LBP and Control subjects showed equivalent abdominal muscle activity, they differed with regard to their back muscle activities. Of the two groups, it is most probably the LBP subjects who are unbalanced or, in the vernacular of the model cited earlier, "have their guy wires slack".

Yet another pathophysiological condition involving the intervertebral discs, a condition which may follow from hypoactive posterior lumbar muscles, relates to nutrition of the discs. In adult life the discs are not vascularized, and it is thought that they acquire the nutrients to maintain their integrity by fluid diffusion through the vertebral endplates. This fluid movement is promoted by a mechanical pumping action which comes from cyclic loading and unloading forces on the disc, a decrease in which would, in all probability, lead to accelerated degeneration due to nutritional deficits (Nachemson, 1975). Since the LBP subjects of this study do not appear to be loading their discs as much as the normal subjects, they may be decreasing this pumping action and nutritional process.

Possible Origins of the Observed Psychophysiological Anomalies of LBP Subjects

Stated in the most extreme and simplified version, the

RR IRS characterizing LBP subjects would be akin to not holding the breath while lifting. This would of course subject the spine to overloads and damaging compression, torsion, and shear forces. Not restricting the breathing during the occurrence of environmental stressors and light motor tasks would also leave an individual's spine unprotected against unexpected heavy loads that may well follow on such stressors and modest tasks. How the behaviour of breath-holding during lifting is acquired is uncertain, but it may be learned in that overt instructions to do so are commonplace. What is even less certain is how the behaviour of restricting RR, as an anticipatory biomechanically protective response, might be acquired.

Why the LBP subjects of the present study should have had hypoactive posterior lumbar muscles cannot be answered on the basis of the study, but the hypoactivity may simply be one of those physiological individual differences, as is individual response stereotypy, which is perhaps related to early learning or genetic endowment (Roessler & Engel, 1974). The finding may have other explanations, however, which could lead to interesting and productive questions for further research.

One possibility might be that even a minimal history of LBP, such as that which had been experienced by the subjects of this study, may promote learning during symptomatic periods of subtle, pain relieving postural positions associated with posterior muscle laxity, postures which are then maintained

during asymptomatic periods. Fordyce (1974) has described how learning of disturbed posture or gait can take place during periods of pain and, because it is instrumental in reducing or avoiding the pain, how the learned posture or gait may be maintained long after the organic lesion has resolved. Thus, a study of habitual postures of LBP and normal subjects would be most interesting.

A second possibility is that the back muscle hypoactivity observed in the LBP subjects of the present study may have resulted from partial denervation of the posterior lumbar muscles which are supplied at segmental levels by the posterior primary rami (Mack, 1950). Such partial denervation has been identified in post-surgery LBP patients[9] (Larson, 1975; Mack, 1950) but has been attributed to the effects of surgery. Perhaps, however, the denervated condition predates surgery and is of etiological significance to LBP. This possibility would appear to warrant investigation by diagnostic, qualitative EMG examination of a group of subjects with a minimal history of LBP, such as the group involved in the present study.

Implications of the Present Findings for LBP Therapies and Further Research

The unexpected finding of an IRS involving the RR variable

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9. McCracken, William. Medical Director, Ontario WCB. Personal discussion concerning recently completed research, May 1979.

has rather direct implications for the development of LBP, as described previously. To protect the spine from injury and to unload the discs during physical effort, it would obviously be desirable to teach individuals to hold their breaths and activate the abdominal hydraulics of the "balloon effect" at the appropriate times. The appropriate times would probably include not only times of physical effort, such as lifting but also times following immediately on environmental stressors, which may signal the subsequent demand for rapid and extreme physical effort or responses.

Many treatment centres, such as the BCWCB Clinic, include educational programs concerning back care and lifting techniques in the overall therapy for LBP, but these programs concern themselves mainly with maintaining muscle strength and appropriate postures. It would appear that much could be gained by also attending to breathing habits of the LBP patients in these programs. Perhaps the desired breath-holding could be accomplished in these programs by simple instructions and practice for the patients, in that RR is easily controlled voluntarily. Alternatively, even the very crudest of physiological monitoring or biofeedback equipment (i.e., a liquid-filled surgical tube encircling the chest, connected to a makeshift manometer) could be used to monitor the patients' RR behaviours during various stressors and motor tasks, thus incorporating a biofeedback paradigm into this training.

Whatever the cause(s) of the low baseline back EMG activity levels shown by the LBP subjects of this study, they were capable of substantial EMG responses. Furthermore, researchers using maximal tests of gross back and abdominal muscle strength have not found significant differences between LBP patients and normal subjects (Nachemson & Lindh, 1969; Nachemson, 1975). It would thus seem that LBP subjects probably have normal back muscle capacity or strength available, but simply are not using it. Because it would in all probability be biomechanically beneficial to individuals with a LBP history to stabilize their spines more during the everyday activities of maintaining posture, moving around, and performing work, an interesting idea presents itself: It should be possible to increase substantially the EMG activity level of the posterior lumbar muscles, both during rest and with activity, in these individuals by using a neuromuscular re-education biofeedback technique (Inglis, Campbell & Donald, 1976). What prophylactic value such biofeedback training in the earliest stages of a LBP history would have for prevention of further LBP would certainly be an interesting topic for study. However, in light of the findings and biomechanical analyses presented in this paper, there certainly should be considerable caution exercised in applying a poorly-reasoned LBP treatment consisting of biofeedback reduction of posterior lumbar muscle activity. Such treatment is being undertaken by several individuals locally and has been reported

by others elsewhere (Kravitz, Moore, Glaros & Stauffer, 1978; Malpe & Yue, 1979). Indeed, preliminary clinical research along these lines (Douglas, Crockett, Wilfling, Craig & Wing, 1979) included a LBP patient whose symptoms increased with lumbar muscle EMG reduction. This observation, which is consistent with the findings and biomechanical analyses of the present study, is discrepant with the reports of Kravitz, et al. (1978) and Malpe and Yue (1979), who reported LBP relief as the result of an EMG biofeedback reduction procedure. Steger (1979) has reported that, in his clinical experience, very few LBP patients exhibit abnormally high EMG values of the posterior back muscles, but those that do respond well to an EMG biofeedback reduction procedure. Steger did not comment on whether or not he has observed unusually low posterior back muscle EMG activity in any of his patients. Perhaps the somewhat summary and universal clinical observation, "Some get Better, Some get Worse", of psychologists at the Ontario WCB[10] who have also used EMG biofeedback to reduce posterior muscle activities in LBP patients, most accurately reflects the current status of knowledge with regard to such treatment.

Perhaps, as also seems universally true, there is more than one possible mechanism accounting for any one presenting symptom and, in fact, perhaps both increased and decreased

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10. Doxey, N. Personal communication, January 1979.

posterior muscle tension may potentially give rise to LBP. For example, a biofeedback reduction of the muscle spasm which seems undoubtedly present in some acute LBP patients may give relief of intramuscular pain, whereas a biofeedback increase of posterior back muscle activity may help to stabilize the spines of LBP patients who do not have spasm or acute symptoms, thus preventing the pain from shear-induced mini-lesions. Interesting and potentially valuable questions such as these might be asked in further research. Answers to such questions might prevent future grief resulting from treating some patients the wrong way, and allow benefit to all patients by treating them differently but appropriately.

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APPENDICES

APPENDIX A

Screening Questionnaire

(Low Back Pain Survey)

LOW BACK PAIN SURVEY

This questionnaire is about low back pain. That is, pain at or near the spine, in the area from the beltline down to the upper buttocks. The pain can be of different types, such as feelings of tightness or cramping, dull aches, sharp, searing or cutting, etc.

The answers to this questionnaire, as all research data, are strictly confidential.

Age_____ Sex_____ Occupation_____ Have you filled out this questionnaire before? _____

Have you ever had low back pain? _____
How many times have you had low back pain in the past year? _____
When did you last have low back pain, and how long did it last? _____

Have you ever had to limit your non-work activities because of low back pain? _____
During the past year?_____
What extent of restriction? _____

Have you ever missed work because of low back pain? _____
During the past year? (how many days) _____

Have you ever seen a Doctor because of low back pain? _____
During the past year? (How often) _____
Have you ever seen a specialist about low back pain? _____

Have you ever taken medication for low back pain? _____What kind?_____
Have you ever had x-rays of your low back? _____
Have you ever had physiotherapy or chiropractic treatment for low back pain? _____
Have you ever had a back operation? _____
Are you presently receiving treatment for low back pain? _____

Have you ever seriously injured your low back?_____ How?_____
_____

What sort of things make you get low back pain? _____
_____

Do you think that muscle tension is involved in your low back pain? _____
How? _____

If you would like to be a subject in this low back pain research --- people without back pain are also needed for study --- then please fill in the information blanks below. This is not a final consent to be a subject - before giving that, I'll tell you everything about the study and answer any questions.

Name (Please print): _____

Summer address: _____

Telephone or way to contact: _____

APPENDIX B

Subject Consent Form

Basic Rights and Privileges of Volunteer Subjects

Any person who volunteers to participate in experiments conducted by full or part-time members of the faculty of the Department of Psychology at the University of British Columbia, by their employees, or by the graduate and undergraduate students working under the direction of faculty members of the above-named Department, is entitled to the following rights and privileges.

1. The subject may terminate and withdraw from the experiment at any time without being accountable for the reasons for such an action.
2. The subject shall be informed, prior to the beginning of an experiment, of the maximum length of time the experiment might take and of the general nature of the experiment.
3. The subject shall be informed, prior to the beginning of an experiment, of the nature and function of any mechanical and electric equipment which is to be used in the experiment. In cases where the subject is in direct contact with such equipment, he shall be informed of the safety measures designed to protect him from physical injury, regardless of how slight the possibility of such injury is.
4. The subject shall be informed prior to the beginning of an experiment, of the aspects of his behavior that are to be observed and recorded and how this is to be done.
5. Any behavioral record that is obtained during the course of the experiment is confidential. Any behavioral records that are made public through either journal papers or books, public addresses, research colloquia, or classroom presentations for teaching purposes, shall be anonymous.
6. The subject shall be offered, at the end of an experiment, a complete explanation of the purpose of the experiment, either orally by the experimenter or, at the option of the experimenter, in writing. The subject shall also have the opportunity to ask questions pertaining to the experiment and shall be entitled to have these questions answered.
7. The subject has the right to inform the Chairman of the Departmental Committee on Research with Human Subjects of any perceived violations of, or questions about, the aforementioned rights and privileges.

TITLE OF STUDY: _____

DATE: _____

I have read the above statement of my rights as a volunteer subject, understand the conditions of this experiment and am participating voluntarily.

SIGNED: _____

APPENDIX C

Laboratory Interview Form

LABORATORY INTERVIEW FORM

S#_____ Sex_____ Handedness_____ Date_____ Time _____

Any diagnosed LB path? _____

Description of LBP _____

_____

Any medications 24 hrs. _____

Unusual activity 24 hrs. _____

History of major med. probs. _____

_____

Current med. probs. _____

_____

Periodicity, day _____

APPENDIX D

Eysenck Personality Inventory

PERSONALITY INVENTORY

Name _____ Age_____ Sex_____

Occupation _____

INSTRUCTIONS

Please answer each question by putting a circle around the "YES" or the "NO" following the question. There are no right or wrong answers, and no trick questions. Work quickly and do not think too long about the exact meaning of the question.

REMEMBER TO ANSWER EACH QUESTION

1.	Does your mood often go up and down?	YES	NO
2.	Are you a talkative person?	YES	NO
3.	Have you ever taken the credit for something you knew someone else had really done?	YES	NO
4.	Do most things taste the same to you?	YES	NO
5.	Do you ever feel 'just miserable' for no good reason?	YES	NO
6.	Can you usually let yourself go and enjoy yourself a lot at a fun party?	YES	NO
7.	Were you ever greedy by helping yourself to more than your share of anything?	YES	NO
8.	Would it upset you a lot to see a child or an animal suffer?	YES	NO
9.	Do you often worry about things you should not have done or said?	YES	NO
10.	Do you have many different hobbies?	YES	NO
11.	If you say you will do something do you always keep your promise no matter how inconvenient it might be?	YES	NO
12.	Do you think that marriage is old-fashioned and should be done away with?	YES	NO
13.	Are your feelings rather easily hurt?	YES	NO
14.	Do you like going out a lot?	YES	NO
15.	Have you ever blamed anyone for doing something you knew was really your fault?	YES	NO
16.	Do you love your mother?	YES	NO
17.	Are you an irritable person?	YES	NO
18.	Do you have many friends?	YES	NO
19.	Are <u>all</u> your habits good and desirable ones?	YES	NO
20.	Do you enjoy hurting people you love?	YES	NO

21.	Are you often troubled about feelings of guilt?	YES	NO
22.	Do you hate being in a crowd who play harmless jokes on one another?	YES	NO
23.	Have you ever taken anything (even a pin or a button) that belonged to someone else?	YES	NO
24.	Can you easily understand the way people feel when they tell you their troubles?	YES	NO
25.	Would you call yourself tense or highly strung?	YES	NO
26.	Are you rather lively?	YES	NO
27.	Do you sometimes talk about things you know nothing about?..	YES	NO
28.	Would you like to think that other people are afaid of you?.	YES	NO
29.	Do you worry about awful things that might happen?	YES	NO
30.	Can you easily get some life into a rather dull party?	YES	NO
31.	Do you always say you are sorry when you have been rude? ...	YES	NO
32.	Would you take drugs which may have strange or dangerous effects?	YES	NO
33.	Would you call yourself a nervous person?	YES	NO
34.	Do you prefer reading to meeting people?	YES	NO
35.	Have you ever broken or lost something which belonged to someone else?	YES	NO
36.	Do you enjoy practical jokes which sometimes hurt people? ..	YES	NO
37.	Do you worry about your health?	YES	NO
38.	Are you mostly quiet when you are with other people?	YES	NO
39.	Do you sometimes boast a little?	YES	NO
40.	Is your mother a good person?	YES	NO
41.	Do you suffer from sleeplessness?	YES	NO
42.	Do you like having long chats on the telephone?	YES	NO
43.	Have you ever said anything nasty or bad about anyone?	YES	NO
44.	Have you always been known as a loner?	YES	NO
45.	Do you sometimes sulk?	YES	NO
46.	Would you rather plan things than do things?	YES	NO
47.	As a child were you ever cheeky to your parents?	YES	NO
48.	Do your friendships break up easily without it being your fault?	YES	NO
49.	Do you often feel life is very dull?	YES	NO
50.	Do you often take on more activities than you have time for?	YES	NO

51. Do you always wash before a meal? ..... YES NO
52. Would you feel very sorry for an animal caught in a trap? .. YES NO
53. Have you often felt listless or tired for no good reason? .. YES NO
54. Do you like telling jokes and telling funny stories to your friends? ..... YES NO
55. Have you ever cheated at a game? ..... YES NO
56. Are you always specially careful with other people's things? YES NO
57. Do you often feel fed up? ..... YES NO
58. Do you like mixing with people? ..... YES NO
59. Have you ever taken advantage of someone? ..... YES NO
60. When you are in a crowd, do you worry about catching germs?. YES NO
61. Are you touchy about some things? ..... YES NO
62. Do you nearly always have a 'ready answer' when people talk to you? ..... YES NO
63. Are you always polite even to unpleasant people? ..... YES NO
64. Do you try not to be rude to people? ..... YES NO
65. Are you sometimes bubbling over with energy and sometimes very sluggish? ..... YES NO
66. Would you call yourself happy-go-lucky? ..... YES NO
67. Have you ever insisted on having your own way? ..... YES NO
68. Do you sometimes get cross? ..... YES NO
69. Do you worry too long after an embarrassing experience? ..... YES NO
70. Do you mind selling things or asking people for money for some good cause? ..... YES NO
71. Would you dodge paying taxes if you were sure you would never be found? ..... YES NO
72. Have you ever told a lie? ..... YES NO
73. Do you suffer from 'nerves'? ..... YES NO
74. Do you prefer to have few but special friends? ..... YES NO
75. Have you ever deliberately said something to hurt someone's feelings? ..... YES NO
76. Do good manners and cleanliness matter much to you? ..... YES NO
77. Are you easily hurt when people find fault with you or the work you do? ..... YES NO
78. Do you often do things on the spur of the moment? ..... YES NO
79. Do you always practice what you preach? ..... YES NO
80. Did you mind filling in this questionnaire? ..... YES NO

APPENDIX E

McGill Pain Assessment Questionnaire

PAIN QUESTIONNAIRE

Patient's name _____ Age _____

File No. _____ Date _____

Clinical category (e.g., cardiac, neurological, etc.):

_____

Diagnosis: _____

_____

_____

Analgesic (if already administered):

1. Type _____
2. Dosage _____
3. Time given in relation to this test _____

Patient's intelligence: Circle number that represents best estimate

1 (low) 2 3 4 5 (high)

****

This questionnaire has been designed to tell us more about your pain.

Four major questions we ask are:

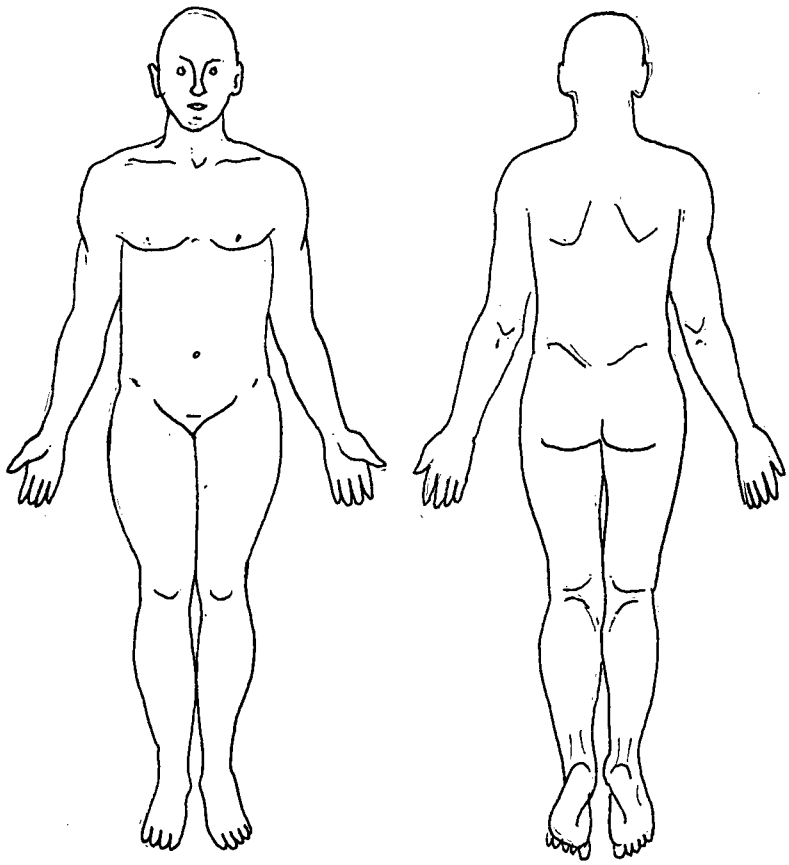
1. Where is your pain?
2. What does it feel like?
3. How does it change with time?
4. How strong is it?

It is important that you tell us how your pain feels now.

Please follow the instructions at the beginning of each part.

Where is your Pain?

Please mark, on the drawings below, the areas where you feel pain. Put E if external, or I if internal, near the areas which you mark. Put EI if both external and internal. ALSO: If you have one or more areas which can trigger your pain when pressure is applied to them, mark each with an X.



Comments:

Part 2. What Does Your Pain Feel Like?

Some of the words below describe your present pain. Circle ONLY those words that best describe it. Leave out any category that is not suitable. Use only a single word in each appropriate category -- the one that applies best.

1	2	3	4
Flickering	Jumping	Pricking	Sharp
Quivering	Flashing	Boring	Cutting
Pulsing	Shooting	Drilling	Lacerating
Throbbing		Stabbing	
Beating		Lancinating	
Pounding			
5	**6**	**7**	**8**
Pinching	Tugging	Hot	Tingling
Pressing	Pulling	Burning	Itchy
Gnawing	Wrenching	Scalding	Smarting
Cramping		Searing	Stinging
Crushing			
9	**10**	**11**	**12**
Dull	Tender	Tiring	Sickening
Sore	Taut	Exhausting	Suffocating
Hurting	Rasping		
Aching	Splitting		
Heavy			
13	**14**	**15**	**16**
Fearful	Punishing	Wretched	Annoying
Frightful	Gruelling	Blinding	Troublesome
Terrifying	Cruel		Miserable
	Vicious		Intense
	Killing		Unbearable
17	**18**	**19**	**20**
Spreading	Tight	Cool	Nagging
Radiating	Numb	Cold	Nauseating
Penetrating	Drawing	Freezing	Agonizing
Piercing	Squeezing		Dreadful
	Tearing		Torturing

Part 3. How Does Your Pain Change With Time?

1. Which word or words would you use to describe the pattern of your pain?

1	2	3
Continuous	Rhythmic	Brief
Steady	Periodic	Momentary
Constant	Intermittent	Transient

2. What kind of things relieve your pain?

3. What kinds of things increase your pain?

Part 4. How Strong Is Your Pain?

People agree that the following 5 words represent pain of increasing intensity. They are:

1	2	3	4	5
Mild	Discomforting	Distressing	Horrible	Excruciating

1. Which word describes your pain right now? _____

2. Which word describes it at its worse? _____

3. Which word describes it when it is least? _____

4. Which word describes the worst toothache you ever had? _____

5. Which word describes the worst headache you ever had? _____

6. Which word describes the worst stomach-ache you ever had? _____

Appendix F

Psychophysiological Baselines and Stressor Effects Data

Physiological Variable	Data Points Before Stressor*	Data Points During Stressor*	Data Points After Stressor*	T-Square	F Stat.	df.	Sign.
ABEMG	2	0	4	8.00	1.42	5,32	.243
BKEMG	2	0	4	7.90	1.41	5,33	.246
HR	2	0	4	158.17	28.21	5,33	.000
SC	2	0	4	63.76	11.41	5,34	.000
RR	2	0	4	5.71	1.02	5,34	.421
VM	2	0	4	39.88	6.84	5,24	.000

* All data scored in 30-second intervals unless otherwise noted.

MD = Missing Data.

TONE DATA

Physiological Variable	Data Points Before Stressor*	Data Points During Stressor*	Data Points After Stressor*	T-Square	F Stat.	df.	Sign.
ABEMG	2	1	4	37.87	5.44	6,31	.001
BKEMG	2	1	4	12.79	1.84	6,32	.122
HR	2	1	4	355.93	50.85	6,30	.000
SC	2	1	4	156.48	22.65	6,33	.000
RR	2	1	4	11.77	1.70	6,33	.151
VM	2	1	4	44.62	6.01	6,21	.000

* All data scored in 30-second intervals unless otherwise noted.

MD = Missing Data.

COUNT DATA

Physiological Variable	Data Points Before Stressor*	Data Points During Stressor*	Data Points After Stressor*	T-Square	F Stat.	df.	Sign.
ABEMG	2	6	4	25.72	1.69	11,26	.132
BKEMG	2	6	4	24.77	1.64	11,27	.143
HR	2	6	4	288.89	18.97	11,26	.000
SC	2	6	4	163.34	10.94	11,28	.000
RR	2	6	4	205.85	13.78	11,28	.000
VM	2	6	4	109.40	5.97	11,15	.001

* All data scored in 30-second intervals unless otherwise noted.

MD = Missing Data.

PONG DATA

Physiological Variable	Data Points Before Stressor*	Data Points During Stressor*	Data Points After Stressor*	T-Square	F Stat.	df.	Sign.
ABEMG	2	3	3	51.11	5.11	7,14	.005
BKEMG	2	3	3	10.42	1.04	7,14	.446
HR	2	3	3	92.22	9.01	7,13	.000
SC	2	3	3	47.65	4.76	7,14	.006
RR	2	3	3	23.36	2.34	7,14	.084
VM	MD	MD	MD	MD	MD	MD	MD

* All data scored in 30-second intervals unless otherwise noted.

MD = Missing Data.

COLD PRESSOR DATA

Physiological Variable	Data Points Before Stressor*	Data Points During Stressor*	Data Points After Stressor*	T-Square	F Stat.	df.	Sign.
ABEMG	2	0	4	88.49	15.78	5,33	.000
BKEMG	2	0	4	51.98	9.24	5,32	.000
HR	2	0	4	778.79	138.45	5,32	.000
SC	2	0	4	65.59	11.74	5,34	.000
RR	2	0	4	14.22	2.54	5,34	.056
VM	MD	MD	MD	MD	MD	MD	MD

* All data scored in 30-second intervals unless otherwise noted.

MD = Missing Data.

STAND DATA

Physiological Variable	Data Points Before Stressor*	Data Points During Stressor*	Data Points After Stressor*	T-Square	F Stat.	df.	Sign.
ABEMG	2	1	4	72.28	10.46	6,33	.000
BKEMG	2	1	4	12.11	1.75	6,32	.143
HR	2	1	4	387.88	55.67	6,31	.000
SC	2	1	4	100.32	14.52	6,33	.000
RR	MD	MD	MD	MD	MD	MD	MD
VM	MD	MD	MD	MD	MD	MD	MD

* All data scored in 30-second intervals unless otherwise noted.

MD = Missing Data.

VALSALVA DATA

Physiological Variable	Data Points Before Stressor*	Data Points During Stressor*	Data Points After Stressor*	T-Square	F Stat.	df.	Sign.
ABEMG	2	1	2	30.20	6.95	4,35	.000
BKEMG	2	1	2	22.55	5.18	4,34	.002
HR	2	1	2	253.53	58.10	4,33	.000
SC	2	1	2	75.94	17.49	4,35	.000
RR	2	1	2	19.25	4.42	4,34	.006
VM	MD	MD	MD	MD	MD	MD	MD

* All data scored in 30-second intervals unless otherwise noted.

MD = Missing Data.

APPENDIX G

Eysenck Personality Inventory Data. Normative Values and Mean Values for Groups of Subjects in the Present Study.

Data Source	Group	Eysenck Personality Inventory Scales			
		Extraversion	Neuroticism	Psychoticism	Lie
Eysenck	Population	12.55	10.95	2.16	7.29
Normal	Males	12.67	9.59	2.74	6.74
Population	Females	12.43	12.31	1.57	7.84
Subjects	Males	11.60	9.95	1.20	5.80
of the	Females	12.35	8.60	1.20	7.60
Present	LBP	12.55	8.85	1.35	6.00
Study	Non-LBP	11.40	9.70	1.05	7.40

APPENDIX H

Individual Response Stereotypy Rank Data

LBP SUBJECTS

SUBJ. NO.	TONE	COUNT	PONG	COLD PRESS.	STAND	VALS.	FLEX	MEAN RANK
1	22	17	33	36	11	35	16	29
2	39	24	31	2	17	25	5	23
3	25	20	13	9	15	14	24	10
4	27	16	10	16	20	34	19	22
6	26	32	36	18	25	22	30	36
13	5	35	21	10	26	28	7	17
16	-	-	-	-	-	10	26	-
17	9	23	35	37	32	31	39	38
18	36	36	3	21	16	20	31	28
19	20	19	19	6	30	17	13	13
21	3	2	2	7	6	4	9	1
25	6	21	22	15	12	16	22	9
26	34	25	29	20	4	21	1	19
29	19	22	9	27	31	8	21	20
31	8	5	32	30	37	6	4	12
33	11	14	24	25	19	13	20	14
35	28	11	8	8	9	11	34	8
38	17	34	38	33	21	24	35	37
39	37	38	17	23	34	7	2	26
40	38	31	1	13	27	38	37	32.5

CONTROL SUBJECTS

SUBJ. NO.	TONE	COUNT	PONG	COLD PRESS.	STAND	VALS.	FLEX	MEAN RANK
5	23	27	15	35	22	9	15	24
7	24	13	26	28	8	33	28	27
8	7	18	25	26	14	32	18	21
9	32	-	-	38	39	2	38	39
10	31	26	34	29	28	37	3	35
11	18	37	14	19	35	23	36	31
12	12	6	5	11	10	27	11	3
14	35	3	11	1	1	29	8	4
15	33	30	30	32	13	15	33	34
20	21	12	28	14	7	39	12	18
22	14	7	16	5	23	40	25	16
23	29	29	23	22	18	26	6	25
24	15	10	12	24	2	12	32	7
27	10	9	27	31	24	30	40	30
28	4	8	4	17	3	18	23	2
30	16	15	7	12	29	36	14	15
32	13	4	6	3	33	19	17	5.5
34	30	28	18	39	38	3	29	32.5
36	2	1	37	34	36	1	10	11
37	1	33	20	4	5	5	27	5.5

- = Missing Data

ABEMG RANKED RESPONSE DATA.

LBP SUBJECTS

SUBJ. NO.	TONE	COUNT	PONG	COLD PRESS.	STAND	VALS.	FLEX	MEAN RANK
1	26	15.5	11	32	23	10	27	22
2	38	27	22	5	9	35	7	21
3	21	15.5	9	31	18	15	9	11
4	32	30	26	28	15	8	29	28
6	21	15.5	3.5	19.5	5	9	4	5.5
13	33	32	24	35	19	12	30	31
16	3	7	14	7	6	23	3	2
17	14	26	35	30	29	26	34	33.5
18	13	29	17	24	7	13	6	9
19	21	15.5	3.5	19.5	3	4.5	1.5	4
21	21	15.5	7	22	14	32	26	18
25	17	10	29	13	35	2	25	15
26	10	28	16	9	22	3	38	13
29	31	37	33	38	26	14	23	37
31	37	9	27	23	38	36	36	38
33	4	3	15	8	2	29	22	7
35	21	15.5	8	25	24	21	28	20
38	-	-	-	-	-	-	-	-
39	6	4	10	4	10	20	35	8
40	5	24	20	6	17	34	11	10

CONTROL SUBJECTS

SUBJ. NO.	TONE	COUNT	PONG	COLD PRESS.	STAND	VALS.	FLEX	MEAN RANK
5	7.5	39	37	36	4	4.5	1.5	14
7	12	8	1	17	34	38	24	16
8	21	15.5	3.5	19.5	28	27	33	24
9	30	25	30	29	12	6	20	26
10	36	35	39	3	39	30	12	33.5
11	35	31	28	14	33	7	18	27
12	9	6	38	33	30	1	32	25
14	29	11	13	16	27	25	15	17
15	7.5	5	6	10	20	19	10	5.5
20	28	34	32	34	31	17	16	32
22	16	21	32	15	36	37	39	35
23	11	36	21	12	25	24	17	23
24	21	15.5	3.5	19.5	37	31	14	19
27	34	20	25	27	21	39	31	36
28	15	38	36	37	8	28	21	30
30	17	22	12	26	32	18	37	29
32	39	33	34	39	11	33	19	39
34	25	23	23	11	13	16	8	12
36	2	2	19	1	1	11	13	1
37	1	1	18	2	16	22	5	3

- = Missing Data

BKEMG RANKED RESPONSE DATA.

LBP SUBJECTS

SUBJ. NO.	TONE	COUNT	PONG	COLD PRESS.	STAND	VALS.	FLEX	MEAN RANK
1	12	30	24	9.5	10	-	12	11
2	28	13	7	20	27	-	22	24
3	6	22	14	23.5	37	-	9	15.5
4	19	26	38	14.5	12.5	-	18.5	25
6	13.5	14	19	9.5	25	-	34	17
13	11	39	39	36	15	-	32	34
16	29.5	32	4.5	40	26	-	28	33
17	25	27	23	30	31.5	-	39	36.5
18	17	23	33	11	34	-	6	21
19	20	35	17	3	31.5	-	31	28
21	36	40	25	16	6	-	21	31
25	29.5	33	8	22	1	-	15	13
26	32	25	29	27	39	-	23	35
29	37	15	13	39	11	-	3	18
31	9	10.5	1	23.5	40	-	-	7.5
33	22	5.5	18	32	21	-	29	23
35	40	16	37	33.5	35	-	25	40
38	15	34	40	31	33	-	30	39
39	38	36	34	18	24	-	27	38
40	18	17.5	31	5	29	-	36	27

CONTROL SUBJECTS

SUBJ. NO.	TONE	COUNT	PONG	COLD PRESS.	STAND	VALS.	FLEX	MEAN RANK
5	13.5	12	35	1	4	-	13	5
7	4.5	21	15	38	5	-	37	19
8	24	10.5	32	33.5	7.5	-	4	15.5
9	27	29	6	37	3	-	7.5	14
10	2	2	27	28	28	-	16	12
11	33	38	26	14.5	16	-	14	30
12	4.5	5.5	12	7	17	-	38	7.5
14	21	1	10	21	20	-	20	9.5
15	26	31	36	25.5	12.5	-	18.5	32
20	39	24	22	12	18	-	10	22
22	16	37	30	25.5	19	-	11	29
23	1	4	9	17	9	-	5	1
24	35	17.5	21	29	38	-	35	36.5
27	23	8	3	3	2	-	7.5	2
28	10	7	2	19	30	-	2	3
30	7	9	20	8	14	-	22	6
32	34	28	16	35	7.5	-	1	20
34	8	3	4.5	6	23	-	26	4
36	3	20	11	13	22	-	24	9.5
37	31	19	28	3	36	-	17	26

- = Missing Data

RR RANKED RESPONSE DATA.