

THE VALUE OF CHIROPRACTIC
MANIPULATIVE TREATMENT
IN THE MANAGEMENT OF
INSULIN-DEPENDENT DIABETES
MELLITUS

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compliance with the requirements for the
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BY

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"I, Warren Stephen Long, declare that this work is my own
work, both in conception and execution."

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ABSTRACT

Subjects who were previously diagnosed as having insulin dependent diabetes mellitus (i.d.d.m.) were treated in a single blind, randomised, placebo controlled trial. The objective of this study was to evaluate the efficacy of chiropractic treatment in the management of i.d.d.m.

The sample of 21 subjects was randomly divided into an experimental group of 11 subjects and a control group of 10 subjects. At the initial consultation, a case history and physical examination was performed on each subject and an informed consent form, screening questionnaire and subjective health status questionnaire (S.H.S.Q.) were completed by each subject. Each subject was required to be in possession of a glucometer. The subjects were treated once a week for a period of four months. Glycosylated haemoglobin readings were taken before the commencement of treatment, after two months and at the end of the study. Venous glucose readings were taken before treatment was started and for every two weeks thereafter.

Subjects in the experimental group received chiropractic manipulation of any subluxations detected within the vertebral levels of C0 - C3 and T7 - T12. Subjects in the control group received vacotron suction pads from an interferential unit over the transpyloric plane with the milliamperage set at zero.

Objective measurements for each subject included glycosylated haemoglobin readings and venous glucose readings which were obtained from fortnightly blood tests. Other objective measurements were the capillary glucose readings and units of insulin injected which were recorded by the subjects on a daily basis. The subjective measurements consisted of numerical responses to a subjective health status questionnaire which was completed prior to the initial treatment, after two months and at the final consultation.

The Wilcoxon Signed Rank test was used to analyse the glycosylated haemoglobin, venous glucose and S.H.S.Q. responses within each group. Time series analysis was used to analyse the capillary glucose readings and the units of insulin injected. Comparisons between the experimental and control groups were drawn by using the Mann Whitney U-test.

The results indicated that the only significant difference obtained was a decrease in the control group's glycosylated haemoglobin readings. There was a significant difference between the second and third glycosylated haemoglobin readings ($p = 0.037$) and between the first and third glycosylated haemoglobin readings ($p = 0.022$). There was no significant difference between the first and second glycoylated haemoglobin readings. This resulted in all of the hypotheses having to be rejected. It can therefore be concluded that chiropractic manipulation does not seem to play a significant role in the management of i.d.d.m.

UITTREKSEL

Proefpersone wat reeds gediagnoseer is met insulien afhanklike diabetes mellitus (i.a.d.m.) is in n enkel blinde, willekeurige, plasebo beheerde eksperiment behandel. Die doel van die eksperiment was om die effektiwiteit van chiropraktiese behandeling in die beheer van i.a.d.m. te bepaal.

Die groep van 21 proefpersone is willekeurig verdeel in n eksperimentele groep van 11 en n kontrole groep van 10. By die eerste konsultasie is n deeglike gevallestudie asook n fisiese ondersoek gedoen. Die proefpersone was ook gevra om n toestemmingsvorm en n subjektiewe gesondheidsstatus vraelys in te vul. Die proefpersone moes in besit wees van n glukometer. Die proefpersone is een keer per week behandel vir n periode van vier maande. Bepalings van glukosidiese hemoglobien was gedoen voer die aanvang van die behandeling, na twee maande en aan die einde van die behandelingsperiode. Veneuse glukose bepaling is gedoen voor die aanvang van die behandeling en daarna twee weekliks.

Proefpersone in die eksperimentele groep het chiropraktiese manipulasie van enige sublaksasie van vertebrale vlakke C0 - C3 en T7 - T12 ontvang. Proefpersone in die kontrole groep was oor die transpiloriese vlak behandel met vacotron suigkussings van n interferensiele eenheid teen zero milliampere. Objektiewe bepaling vir elke proefpersone het ingesluit glukosidiese hemoglobien en veneuse glukose metings wat wat elke twee weke gedoen was.

Kapillere bloedglukose en die hoeveelheid eenhede insulien wat ingespuut was, was op n daaglikse basis genoteer. Subjektiewe bepalings het bestaan uit antwoorde in die gesondheidsstatus vraelys wat op n numeriese skaal berus. Hierdie vraelys was inisieel, na twee maande en aan die einde van die behandelingsperiode ingevul.

Die glukosidiese hemglobien, veneuse bloedglukose en die vraelys resultate was deur middel van Wilcoxon se rangorde teken toets geanaliseer. Kapillere bloedglukose en die eenhede insulien per dag was met n tydreeks analyse geanaliseer. Die Mann Whitney U-toets was gebruik om die eksperimentele en die kontrole groepe met mekaar te vergelyk.

Daar was n beduidende verskil in die tweede en derde glukosidiese hemoglobien waardes ($p = 0.037$) asook tussen die eerste en derde glukosidiese hemoglobien waardes. Daar was geen beduidende verskil tussen die eerste en tweede waardes nie. Die gevolg is dat die hipotese verwerp moet word. Die gevolgtrekking kan dus gemaak word dat chiropraktiese manipulasie skynbaar nie n beduidende rol speel by die beheer van insulien afhanklike diabetes mellitus nie.

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LIST OF ABBREVIATIONS

- i) i.d.d.m.: Insulin-dependent diabetes mellitus.
- ii) IFC: Interferential current.
- iii) HbA1c: Glycoslated haemoglobin.
- iv) S.O.A.P.: Subjective objective assessment plan.
- v) n.i.d.d.m.: non-insulin-dependent diabetes mellitus.
- vi) S.H.S.Q.: Subjective Health Status Questionnaire.

CHAPTER 1

1. THE PROBLEM AND ITS SETTING

1.1 Problem Statement

The purpose of this placebo controlled study is to determine the effects of manipulation of the upper cervical and lower thoracic vertebrae on insulin-dependent diabetics in terms of glycosylated haemoglobin levels, venous and capillary glucose levels and units of insulin administered, as well as the subjects' subjective perception of their health, in order to determine what contribution such intervention makes in the management of insulin-dependent diabetes mellitus (i.d.d.m.).

1.2 SUB-PROBLEMS

1.2.1 Sub-problem 1

The first sub-problem is to evaluate the effectiveness of upper cervical and lower thoracic manipulation on the experimental group's glycosylated haemoglobin levels, venous and capillary glucose levels, and units of insulin administered, as well as their subjective perception of their health, in order to establish the extent of the relationship between these variables and spinal manipulation.

1.2.2 Sub-problem 2

The second sub-problem is to evaluate the effectiveness of de-tuned interferential current over the transpyloric plane on the control group's glycosylated haemoglobin levels, venous and capillary glucose levels, and units of insulin administered, as well as their subjective perception of their health, in order to establish the extent of the relationship between these variables and the placebo "treatment".

1.2.3 Sub-problem 3

The third sub-problem is to integrate the results obtained from the experimental and control groups in order to determine what contribution chiropractic manipulation of the upper cervical and lower thoracic vertebrae makes in the management of insulin-dependent diabetes mellitus (i.d.d.m.).

1.3 HYPOTHESES

1.3.1 It is hypothesized that manipulation of the upper cervical and lower thoracic vertebrae will tend to normalize the experimental group's glycosylated haemoglobin and blood glucose levels, decrease their units of insulin administered and improve their subjective health perception by having a homeostatic effect on the pancreas via the autonomic nervous system.

1.3.2 It is hypothesized that the placebo administered to the control group will have little or no effect on their glycosylated haemoglobin levels, blood glucose readings or units of insulin injected, but may have a beneficial effect on their subjective health status.

1.3.3 It is hypothesized that these specific chiropractic manipulations will provide a significant contribution to the management of i.d.d.m.

1.4. DELIMITATIONS

- i) This study will be limited to the treatment of insulin-dependent diabetics only.
- ii) Only subjects whose diabetes has stabilized will be considered for this study; that is, those patients who have not been admitted to hospital for poor diabetic control during the past three months.
- iii) None of the subjects participating in this study are to have had chiropractic treatment within the last year.
- iv) Blood samples taken from the subjects will only be tested for glucose and glycosylated haemoglobin levels.

- v) Only subjects with their own glucometers will be accepted into the study.

1.5 ASSUMPTIONS

- i) It is assumed that owing to the prevalence of i.d.d.m., a large enough sample size will be obtained (viz., 20 subjects).
- ii) It is assumed that we, as chiropractic interns/residents, have been adequately trained in both the detection and correction of spinal joint dysfunctions (SJD).
- iii) It is assumed that spinal joint dysfunctions can influence the function of the pancreas via the autonomic nervous system.
- iv) It is assumed that chiropractic manipulative techniques are effective in the normalization of spinal joint dysfunctions.
- v) It is assumed that all subjects will co-operate when it comes to writing up daily readings of their blood glucose and units of insulin administered.
- vi) It is assumed that the subjects' diet and exercise will not be altered during the treatment period so as to ensure that these variables remain as constant as possible.

vii) It is assumed that subjects will be compliant with regard to keeping scheduled appointments.

viii) It is assumed that the laboratory tests used for blood glucose and glycosylated haemoglobin levels are both reliable and valid.

1.6 DEFINITION OF TERMS OR ABBREVIATIONS

i) Manipulation:

A short lever, specific, high velocity, low amplitude controlled forceful thrust by hand directed at specific spinal articulations.

ii) Spinal joint dysfunction (SJD)/vertebral subluxation / fixation:

These have one or more of the following characteristics, viz.: alteration of the normal dynamics, anatomical or physiological relationships of contiguous articular structures, lack of joint play, palpable soft tissue changes such as muscle hypertonicity or imbalance, as well as local tenderness.

iii) Osteopathic lesion:

This is the same as a spinal joint dysfunction.

iv) Interferential current:

An electrical stimulation that utilises two medium frequency currents that are identical in frequency but differ in amplitude.

v) Insulin-dependent diabetes mellitus (i.d.d.m.):

A syndrome resulting from a variable interaction of hereditary and environmental factors, and characterised by abnormal insulin secretion and inappropriately elevated blood glucose levels, i.e. a fasting plasma glucose concentration of > 140 mg/dL (7.8 mmol/L). I.d.d.m. includes a variety of end organ complications including nephropathy, retinopathy, neuropathy and accelerated atherosclerosis.

vi) Ketoacidosis:

Ketoacidosis occurs when there is insufficient insulin to meet the body's needs and the deficiency is allowed to continue uncorrected. It is characterised by air hunger (an attempt to compensate for metabolic acidosis), dry skin, flushed face, subnormal temperature, fruity acetone odour on the breath, nausea and vomiting, abdominal tenderness, extreme thirst and dry mucous membranes, reflecting water depletion, weight loss, glucosuria, ketonuria, hyperglycaemia and metabolic acidosis in the blood.

vii) Microvascular disease:

This occurs when there is an abnormality of the capillary basement membranes characterized by added layers and consequent increased thickness of the lamina. This is found in the capillary beds of skin and muscle. Clinically, the most important sites of microvascular involvement are the retina and renal glomeruli as well as the renal medulla, nervous system, pancreas and heart.

viii) Macrovascular disease:

Atherosclerosis involving primarily the medium-size and larger arteries in diabetics. The peroneal, anterior and posterior tibial and digital arteries of the legs are usually involved.

ix) Neuropathy:

Segmental injury to nerves, associated with demyelination and Schwann cell degeneration, involving sensory and motor peripheral nerves, nerve roots, spinal cord, and the autonomic nervous system. Affected nerves show basal lamina thickening similar to the capillary abnormalities. Clinical manifestations include sensory disturbances, plantar ulcers, trophic skin lesions and ulcers, autonomic neuropathy (anhidrosis, vasodilation, oedema, erythema, atrophy of skin), Charcot joints and painless degenerative arthropathy.

x) Objective health status:

Daily logs that contain readings of the subject's capillary blood glucose levels as well as the units of insulin injected.

xi) Subjective perception:

Responses to questions asked in the subjective health status questionnaire (Appendix 4).

xii) Transpyloric plane:

A horizontal plane passing through the first lumbar vertebral body, the pylorus of the stomach, the body of the pancreas, the renal plexus of the left kidney and the middle segment of the right kidney.

1.7 IMPORTANCE OF THE STUDY

a) Immediate background to the problem

The idea for proposing such a study arose from anecdotal evidence provided by practising chiropractors who claim to have "cured" insulin-dependent diabetics or significantly influenced the course of i.d.d.m. through spinal manipulative therapy. On reviewing the available literature, no direct scientific evidence on the chiropractic treatment of i.d.d.m. could be found to either substantiate or refute these claims.

b) Need for solution to the problem

Numerous research studies have already established the benefits of chiropractic treatment in the management of neuromusculoskeletal disorders. These include low back pain (Bronfort, 1986; Cherkin and MacCormack, 1989; Meade et al. 1990; Waagen et al. 1986), cervical pain (Carrick, 1983) and cervical spine dysfunction (Hviid, 1971). Studies on the effectiveness of chiropractic treatment of headaches (Vernon,

1982) and migraine (Parker, 1978) have also been performed. Literature on the chiropractic treatment of other spine related disorders include those on asthma (Jamison et al. 1986; Nilsson and Christiansen, 1988), hypertension (McNight and DeBoer, 1988; Yates et al. 1988), infantile colic (Klougart et al. 1989) and nocturnal enuresis (Leboeuf et al. 1991).

There is no available evidence regarding the chiropractic treatment of organic disorders such as i.d.d.m. The rationale for spinal intervention in the management of visceral conditions is based upon empiricism (Jamison et al. 1992). Evidence in favour of the chiropractic treatment of organic disorders is of an empirical nature which highlights the need for more controlled studies in the future.

c) Benefits from the solution to the problem

- i) The results of this study may contribute to determining the effectiveness of chiropractic manipulation in the management of i.d.d.m.
- ii) Certain health benefits would be apparent if the treatment was demonstrated to be effective; for example:
 - a) Chiropractic treatment is less invasive than medical treatment.

- b) Decreased tissue scarring at site of injection if, for instance, as a result of the treatment, the patient's number of injections were reduced.

 - c) A possible reduction in the complications of i.d.d.m. such as macrovascular disease, nephropathy, retinopathy, neuropathy, cataracts and the loss of connective tissue mobility affecting joints and the skin.
- iii) If the results of this study proved favourable, there would be certain financial benefits for the diabetic patients in that their medical costs could be reduced. These costs involve the purchasing of insulin, doctors' bills, and possibly hospital costs. However, these would have to be offset by the cost of the chiropractic care, the frequency and duration of which would still have to be determined.

With regard to (c) above, to date few studies have established a clear link between tight glycaemic control and the prevention of the microvascular and macrovascular complications of diabetes. That may change, however, when the Diabetes Control and Complications Trial (DCCT), which was expected to be completed in 1993, is published.

CHAPTER 2

2. REVIEW OF THE RELATED LITERATURE

2.1 INTRODUCTION

The following literature review will provide a description of the medical background of insulin-dependent diabetes mellitus (i.d.d.m.). This will include age of onset, diagnosis, signs and symptoms, aetiology, blood glucose monitoring, and long-term complications. This outline of the available literature will also attempt to provide evidence which both supports and refutes the relationship between spinal joint dysfunction and i.d.d.m.

2.2 MEDICAL BACKGROUND

2.2.1 Introduction

I.d.d.m. is a syndrome resulting from a variable interaction of hereditary and environmental factors. It is characterised by abnormal insulin secretion, inappropriately elevated glucose levels, and a variety of end-organ complications, including nephropathy, retinopathy, neuropathy and accelerated atherosclerosis (Berkow, 1987: 1069).

2.2.2. Aetiology of i.d.d.m.

As far as the aetiology of i.d.d.m. is concerned, numerous possible causes have been discussed in the literature. The first is the presence of certain types of genes on a specific chromosome which may increase the person's risk of developing i.d.d.m. (Jones, 1989: 7; Kozak, 1982: 22). Secondly, certain viruses, such as Coxsackie B4, have been shown to cause diabetes in some humans (Jones, 1989: 9; Kozak, 1982: 23). Thirdly, most newly diagnosed insulin-dependent diabetics contain in their blood an antibody to their pancreatic islet cells. This may reduce the islet cells' ability to produce insulin. (Kobberling and Tattersall, 1982: 125; Kozak, 1982: 22.)

Jones (1989: 9) believes that people with i.d.d.m. are born with an inherited increased risk of developing it. This inherited factor is conferred by genetic material located on at least three different chromosomes, or parts of the cell nuclei. However, the most important genetic material is located on the sixth chromosome and is very close to the part of the chromosome known as the HLA DR3 and DR4. This chromosomal material is responsible for helping the body to recognise foreign proteins (antigens) and allows the white blood cells (WBCs) to attack damaged or diseased cells. Ninety per cent of insulin-dependent diabetics have this genetic characteristic.

The HLA DR3 DR4 antigen is then found on the surface of the cells in the pancreas which produce insulin (beta cells of the islets of Langerhans) in people who later develop i.d.d.m.

This appears to result in the body's immune system attacking the insulin-producing cells with WBCs as well as with antibodies. Eventually, the beta cells die, and insulin production ceases. This lack of insulin then allows the blood sugar to rise and the symptoms of i.d.d.m. to develop.

Jones (1989: 9) is of the opinion that while inherited factors do play a role in the development of diabetes, a triggering mechanism is required which causes the diabetes to appear. At present, the most likely candidate is a virus which triggers off the body's immune defence system, resulting in attack against the islet cells of the pancreas. Antibodies against these cells have been seen in many insulin-dependent diabetics at the time of diagnosis but have also been seen in other people who have had virus infections. However, when the blood of newly diagnosed diabetics is tested for recent virus infections, it is only rarely positive. The theory put forward to explain this is that the virus infection must have taken place many months, or even years, previously. It then resulted in the gradual destruction of the beta cells.

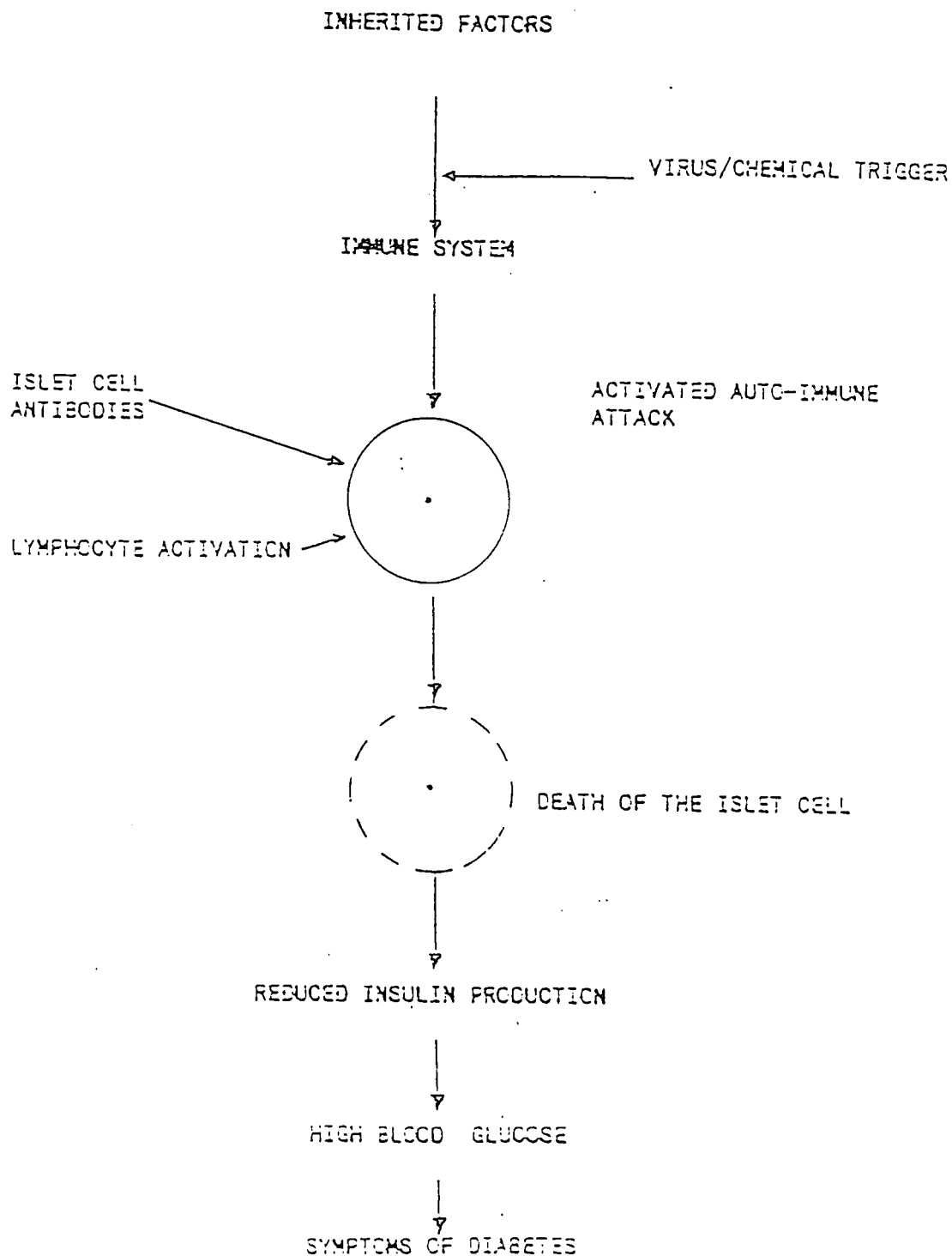
Other possible factors described by Jones (1989: 10) which have been implicated in the cause of diabetes of this type include chemicals which may damage the pancreatic beta cells. (See fig.1.)

Stress, too, has been implicated but this is very difficult to assess. Stress is such a part of normal life that it would be nearly impossible to find anyone who has not had some form of stress in their lives.

Before dismissing the idea altogether, however, it is known that severe stress can cause disturbances to the body's immune system and could theoretically make some people more likely to succumb to viral infection (Jones, 1989: 10).

No literature was found which discussed the possible role of physical stress or trauma in the aetiology of i.d.d.m. This trauma may have included direct trauma to the head or injury to the spinal cord.

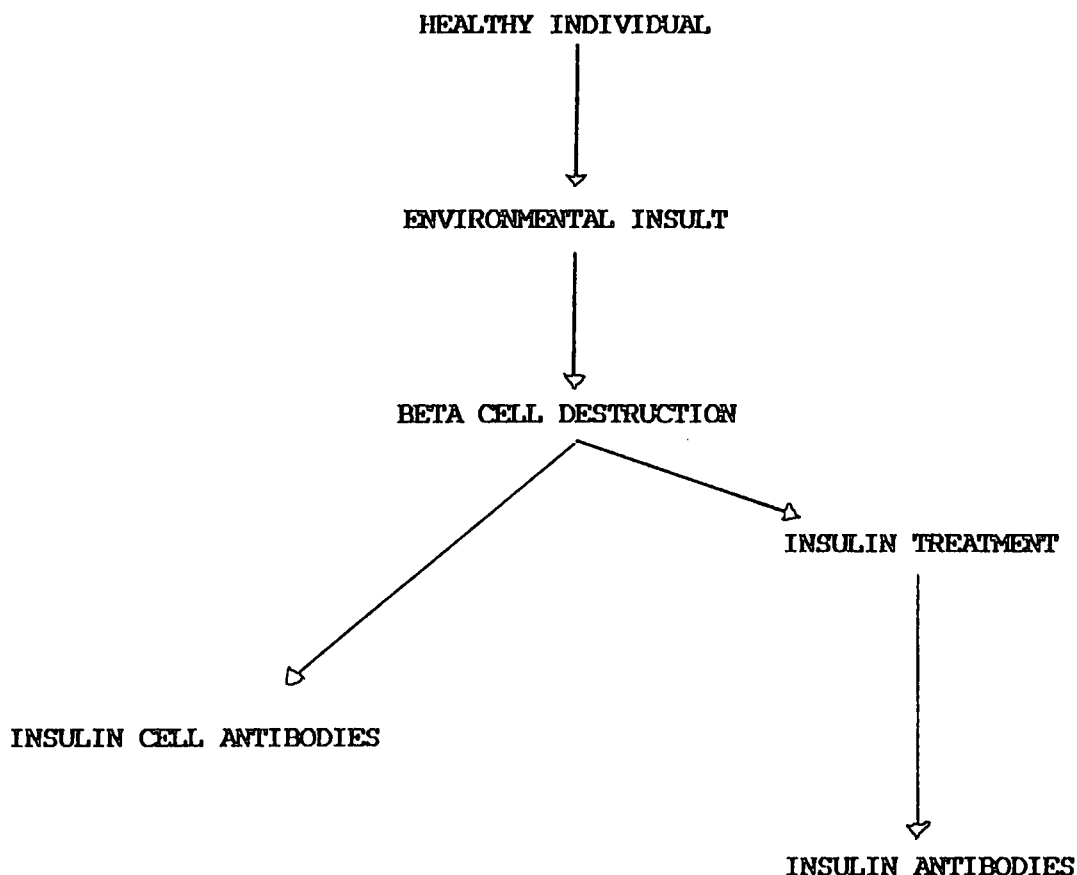
FIGURE 1 : FACTORS IMPLICATED IN THE CAUSE OF I.D.D.M.



Kobberling and Tattersall (1982: 127) indicate that the cases of i.d.d.m. with co-existent auto-immune diseases comprise less than 5% of total i.d.d.m. population. They suggest that a healthy individual may receive an environmental insult which triggers destruction of the beta cells of the pancreas. It is not quite understood what is meant by an "environmental insult", but may suggest a viral infection, hypersensitivity reaction or even physical trauma. There may be genetic differences in susceptibility to the environmental insult but different genetic factors then transform it into the beta cells destruction which causes i.d.d.m.

Over 70% of juvenile diabetics have insulin cell antibodies (ICA) within six months of the onset, but in less than 20% do the ICA persist for five years. Insulin cell antibodies may be a result rather than a cause of islet cell destruction. (See fig. 2.)

FIGURE 2 : PROPOSED AETIOLOGICAL MECHANISM OF IDDM



It has also been suggested by Kobberling and Tattersall (1982: 129) that inheritance of i.d.d.m. is dominant. One very reasonable hypothesis as to how the HLA antigens might confer a susceptibility to the development of i.d.d.m. is as cell-surface receptors, perhaps to pancreatotropic virus.

Kozak (1982: 1) explains that the normal pancreas produces 20-40 units of insulin per day. That of an i.d.d.m. patient is far less.

He identifies certain aetiological factors of i.d.d.m.

- 1) Auto-immunity or autoregression: Within the first week of diagnosis, 85-90% of these patients are positive for islet cell antibodies (ICA); that is IgM and IgG.
- 2) Viruses: There is a suggested relationship between seasonal epidemics of certain viruses and a subsequent higher frequency of individuals developing diabetes. Examples include mumps, infectious mononucleosis, rubella, varicella, cytomegalo-virus and infectious hepatitis.
- 3) Histocompatibility antigens (HLA): These are more prevalent in diabetics than the general population; for example, HLA - B8 antigen in the normal population is 31% positive, while in diabetics it is 49% positive; HLA B15 antigen - found in 10% of the normal population and 21% of people with i.d.d.m. The general consensus, therefore, is that patients who possess the B8 or B15 antigen have a significantly greater risk of having, or developing, i.d.d.m.

Kozak (1982: 22) suggests that it is possible that the component of i.d.d.m. that is hereditary is, in fact, a defect in the short arm of chromosome 6 and close to the location of these HLA antigens. He speculates that these antigens lead to the formation of certain cell surface proteins that are molecularly different from the cell surface proteins whose synthesis is determined by other HLA antigens.

He suggests that these specific cell surface proteins produced by these "insulin dependent diabetes-related antigens" render the cell surface of the beta cell particularly attractive to and/or penetrable by certain viruses.

2.2.3 Age of Onset

The age of onset of i.d.d.m. is often less than 30 years, and there is usually a history of diabetes in the family (10% of diabetics have parents or siblings with the disease) (Berkow, 1987: 1071). Although it most commonly presents between the ages of four and 30 years, with a peak in the 10-15 year age group, it may appear at any age (Tunbridge and Home, 1991: 20).

2.2.4 Diagnosis of Insulin-dependent Diabetes Mellitus.

Although no precise marker for i.d.d.m. has yet been established, the diagnosis of insulin-dependent diabetes mellitus is confirmed by one of the following three positive tests (Berkow, 1987: 1072):

- a) The unequivocal elevation of plasma glucose concentration, together with the typical symptoms of polyuria, polydipsia, ketonuria and rapid weight loss.

- b) A fasting plasma glucose concentration greater than or equal to 140 mg / dL(7.8 mmol/l) on more than one occasion.
- c) An elevated plasma glucose concentration after an oral glucose challenge on more than one occasion.

The diagnosis of i.d.d.m. is also confirmed by using reagent strips. Any such estimation should, however, be confirmed in the laboratory. Glycosylated haemoglobin concentrations two percent above the upper end of the normal range may also be regarded as diagnostic. Doubt thus occurs only where random blood glucose concentrations are below 10 mmol/l (whole venous blood), and glycosylated haemoglobin is not markedly elevated. In these circumstances an oral glucose tolerance test may be useful. A diagnosis must never be made on urinary glucose estimations alone, as a low renal threshold for glucose can give misleading results.(Tunbridge and Home 1991: 22.)

Criteria for the diagnosis of i.d.d.m. (Tunbridge and Home 1991: 20):

- i) Symptoms of diabetes and venous plasma glucose of greater than or equal to 11.1 mmol/l (or capillary glucose of greater than or equal to 11.1 mmol/l or venous blood glucose of greater than or equal to 10.0 mmol/l).
- ii) Venous plasma glucose of greater than or equal to 11.1 mmol/l on two separate occasions (or equivalent blood glucose).

iii) Symptoms of diabetes and abnormal glucose tolerance test.

iv) Two abnormalities: fasting and two-hour (or repeated two-hour) glucose tolerance test.

2.2.5 Signs and symptoms of i.d.d.m.

The signs and symptoms in the younger age group (that is < 20 years old) are generally those of polyuria, followed by polydipsia and weight loss. This often leads to a high consumption of soft drinks with high glucose content, thus exacerbating the symptoms. The history may, however, be too short for weight loss to have been evident. A history of infection of the skin or genito-urinary tract is also uncommon in the younger age group. (Tunbridge and Home 1992 : 21.)

Accelerated fat catabolism in the untreated insulin-dependent patient produces ketoacidosis leading to anorexia, nausea, vomiting, air hunger and, if untreated, coma and death (Berkow, 1987 : 1070). The progression into ketoacidosis will be announced by a complaint of feeling unwell, followed by loss of appetite, and then vomiting. Abdominal pain may accompany ketoacidosis and mimic an acute abdomen. Acidotic breathing, severe dehydration and loss of consciousness are severe events of grave prognostic significance if medical help is not sought immediately. (Tunbridge and Home 1992 : 21.)

Over the age of twenty years symptoms may develop quite slowly, with weight loss despite increased appetite being a major marker.

These patients may report problems with furunculosis, balanitis, vulval or vaginal candidiasis, and may have a stigma of other auto-immune diseases. Below the age of thirty years patients presenting with diabetes are likely to require early insulin therapy. (Tunbridge and Home 1991: 21.)

A bilateral, comparatively symmetric, distal polyneuropathy (predominantly sensory)is the most frequent form of diabetic neuropathy. Symptoms generally occur earlier and more severely in the feet, occasionally with sensory loss in a "glove and stocking" distribution or with the appearance of painless, penetrating plantar ulcers. Nerve involvement may be characterised by lancinating pain in the distribution of a single dermatome, or the posterior columns of the spinal cord may be affected, producing loss of position sense and deep tendon reflexes with a positive Romberg sign. Major nerve trunks may be involved, with pain, sensory loss, motor weakness, and deprivation of sympathetic innervation in the distribution of a major spinal or cranial nerve. The third and sixth cranial nerves are most often involved. (Berkow, 1987: 1071.)

Diabetic amyotrophy is found characteristically in elderly men, producing severe pain and muscle weakness around the hip and upper leg. The autonomic nervous system may be involved diffusely, and autonomic insufficiency often occurs early as sweating disturbances or postural hypotension with significant symptoms.

Sexual impotence in the male may be the most common symptom (50-60%) of neuropathy in diabetes; over a period of six months to one year, there is gradual onset of decreasing firmness of erection. While constipation is perhaps the most common intestinal manifestation of diabetic autonomic neuropathy, it tends to be overshadowed by diarrhoea, which is usually intermittent, watery, and frequently worse at night. (Berkow, 1987: 1071.)

2.2.6 Major metabolic effects of i.d.d.m.

The three major metabolic effects of i.d.d.m. according to Solomon et al. (1990: 626) are:

i) Decreased utilisation of glucose

In diabetics, cells dependent on insulin can take in only about 25% of the glucose they require for fuel. Glucose remains in the blood and the blood glucose levels rise (hyperglycaemia). The blood-sugar load is further increased by the liver, which cannot effectively trap glucose or store glycogen without insulin. Whereas glucose does not appear in the urine of nondiabetics, the blood-glucose concentration is so high in the diabetic that sugar spills out into the urine (glucosuria).

ii) Increased fat mobilisation

The absence of insulin promotes the mobilisation of fat stores, so that the blood-fatty acid level may reach five times the normal level, leading to development of atherosclerosis. Unfortunately, the increased fat metabolism by the cells also increases formation of ketone bodies (acetone and other breakdown products of fat metabolism). Ketone bodies build up in the blood, a condition known as ketosis. Ketone bodies can interfere with normal pH balance by releasing hydrogen ions. The pH can become too low, causing acidosis.

When ketone levels rise in the blood, ketones appear in the urine. When ketones are excreted in the urine, they take sodium with them, and the resulting sodium depletion contributes further to acidosis and its fatal consequences.

iii) Increased protein utilisation

Lack of insulin also causes protein wasting. Normally, proteins are constantly being broken down and built up. Without insulin to stimulate protein synthesis, the balance is disturbed and there is a shift in the direction of protein breakdown. Amino acids are taken to the liver and converted to glucose, further compounding the excess glucose problem. The untreated diabetic becomes thin and emaciated, despite (usually) a voracious appetite.

Solomon et al. (1990: 936) further explains that when glucose and ketones are excreted by the kidneys, they take water with them because of increased urinary osmotic pressure. Polyuria results causing dehydration which results in polydipsia.

2.2.7 Factors affecting blood glucose levels

A relative or absolute lack of insulin secretion associated with an excess of circulating stress hormones (including glucagon, catecholamines and cortisol) is responsible for inappropriate elevation of blood glucose and associated alterations in lipid metabolism characterising the metabolic syndrome (Berkow, 1987: 1070).

According to Guyton (1987: 588) cortisol causes a moderate decrease in the rate of glucose utilisation by the cells. Though the cause of this decrease is unknown, most physiologists believe that somewhere between the point of entry of glucose into the cells and its final degradation, cortisol directly delays the rate of glucose utilisation.

Guyton (1987: 601) also mentions a number of other factors that may contribute to an alteration of blood glucose levels. He explains that epinephrine (and to a slight extent norepinephrine as well) is also a potent promoter of liver glycogenolysis. Epinephrine has a similar effect to glucagon, although not quite as strong. Glucagon causes an increase in the blood glucose concentration. Guyton (1987: 660) also describes other hormones such as gastrin, secretin, cholecystokinin and gastric inhibitory peptide which are secreted in the gastrointestinal

tract after a meal. These hormones seem to cause an anticipatory increase in blood insulin in preparation for the glucose and amino acids to be absorbed from the meal.

Apart from glucose which increases insulin secretion, some of the amino acids have a similar effect. This, however, does not occur in patients with i.d.d.m. because insulin promotes transport of amino acids into the tissue cells and also promotes intracellular formation of protein. Insulin is therefore important for the proper utilisation of the excess amino acids as well the excess glucose. (Guyton, 1987: 599)

Other hormones mentioned by Guyton (1987: 599) include growth hormone and adrenocorticotropin. Both these hormones can increase blood glucose levels which would promote the secretion of insulin in non-diabetic patients.

Guyton (1987: 444) explains, too, how sympathetic stimulation also has metabolic effects, causing release of glucose from the liver, and increase in blood glucose concentration and in glycogenolysis in muscle. Stimulation of the sympathetic nerves to the adrenal medullae causes large quantities of epinephrine and norepinephrine to be released into the circulating blood, and these two hormones are in turn carried in the blood to all the tissues in the body. The circulating hormones have almost the same effects on the different organs as those caused by direct sympathetic stimulation. These effects last, however, about ten times as long, because norepinephrine and epinephrine are only slowly removed from the blood.

2.2.8 Blood glucose monitoring

Monitoring of the diabetics plasma glucose levels is achieved by means of self-blood-monitoring techniques involving reagent strips and a glucometer, as well as through the use of stable glycosylated haemoglobin determinations (Berkow, 1987: 1075). Since the half-life of the erythrocyte and its haemoglobin is 60 days, the percentage of stable glycosylated haemoglobin reflects the mean blood glucose concentration over the preceding two months (Berkow, 1987: 1074).

According to Jamison (1984: 17), levels of 6% glycosylated haemoglobin are considered normal, 8% acceptable and over 10% indicative of poor glycaemic control. The process of glycosylation of the red blood cell occurs when the glucose molecule attaches to the haemoglobin. This process is relatively irreversible and remains in that form for the life of the red blood cell which is 120 days. The build-up of glycosylated haemoglobin within the red blood cells (RBC) reflects the average level of glucose that the cell has been exposed to during its life cycle. Berkow (1987: 1075) explains that determinations of glycosylated haemoglobin are helpful in judging the degree of chronic glucose control in both i.d.d.m. and n.i.d.d.m. patients and in judging efficacy of changes in therapy.

Stathopoulos (1984: 54) indicates that for every 10mg/dL (0.5mmol/l) increase in glucose level, there is a corresponding increase in HbA1c of about 0,35%. He explains that the rate of formation of HbA1c depends on blood glucose concentration and is a slow, continuous, non-enzymatic

process occurring during the normal 120-day life span of the red blood cell. Stathopoulos states that glycosylated haemoglobin is a useful test that measures a patient's long-term blood glucose levels free of short-term fluctuations.

2.2.9 Long-term complications

Certain long-term complications may arise in diabetics whose plasma glucose levels are not adequately controlled. The complete clinical syndrome of diabetes mellitus involves hyperglycaemia, microvascular disease (retina and kidney), macrovascular disease (large vessel atherosclerosis) and neuropathy (Berkow, 1987: 1069).

In studying diabetic pathology, it is of interest to note that tissues with the greatest incidence of diabetic complications, e.g. lens, retina, nerves, kidneys, blood vessels, islet cells and red blood cells are freely permeable to glucose and do not require insulin for glucose entry, as do muscle and adipose tissue (Tunbridge and Home, 1991: 24).

Classically late tissue damage of diabetes is divided into microvascular disease and macrovascular disease.

(i) Microvascular disease:

A feature of many tissues in people with diabetes is thickening of the basement membrane which occupies the space between the endothelial cells lining, the capillaries and the cells of the tissues.

It is an early feature in the retina and the renal glomerulus. It has been suggested that such thickening is universal in diabetes, and is an important factor in the development of microvascular disease (Tunbridge and Home, 1991: 44).

Tunbridge and Home (1991: 44) explain that these changes result in renal failure (if glomerular capillaries are involved) or visual loss (if the retinal capillaries are affected) as well as nephropathy of which the first indication is proteinuria. The greater the proteinuria, the more rapid is the development of renal failure. Renal failure is seen in 50% of i.d.d.m. patients after 20-30 years of diabetes.

Diabetic retinopathy is usually first detected five years or more after the diagnosis of diabetes is made and is present to some degree in 50% of patients after 10 years. (Berkow 1987 : 1071.)

(ii) Macrovascular disease:

Diabetics have an increased incidence, earlier onset, and increased severity of atherosclerosis in the intima and increased calcification in the media of the arterial wall. This atherosclerosis predisposes the diabetic to such conditions as ischaemic heart disease, hypertension and peripheral vascular disease. A diabetic has a risk of cardiovascular death 3,5 times that of a nondiabetic of the same age. About 30% of all diabetics eventually develop peripheral vascular disease.

Leg and foot amputations are five times more frequent than in nondiabetic persons, and a significant majority of these amputees have a history of smoking. (Berkow, 1987: 1070.)

Other complications include neuropathy which involves segmental injury to nerves, associated with demyelination and Schwann cell degeneration, as well as involvement of the sensory and motor peripheral nerves, nerve roots, the spinal cord, and the autonomic nervous system. Affected nerves show basal lamina thickening similar to the capillary abnormalities. Distal polyneuropathies result in pain, loss of position sense and deep tendon reflexes, sensory loss and motor weakness.

(Berkow, 1987: 1070.)

Autonomic nervous system insufficiency often occurs early as sweating disturbances or postural hypotension as well as constipation. Other conditions include bladder dysfunction, erectile impotence, diabetic diarrhoea and cardiorespiratory arrest (Tunbridge and Home, 1991: 49).

2.3 RELATED ANATOMY OF THE PANCREAS

2.3.1 Innervation of the pancreas

Solomon et al. (1990: 624) describe how the sympathetic nervous system inhibits enzyme and insulin secretion from the pancreas as well as promoting glucagon secretion. These effects cause an increase in the blood glucose concentrations.

The parasympathetic system, on the other hand, stimulates the secretion of insulin, thus reducing the blood glucose concentrations.

Moore (1985: 223) also describes the nervous supply of the pancreas which is derived from both vagus (parasympathetic) and splanchnic nerves (sympathetic). The sympathetic and parasympathetic fibres reach the gland by passing along arteries from the coeliac and superior mesenteric plexuses. The greater splanchnic nerves receives branches from the fifth to the ninth thoracic sympathetic ganglia, the lesser splanchnic from the ninth and tenth thoracic sympathetic ganglia and the lowest splanchnic from the eleventh and twelfth thoracic sympathetic ganglia. The superior nerve plexus receives the parasympathetic fibres from the coeliac division of the posterior vagal trunk and its sympathetic fibres from the superior mesenteric ganglion.

The vagus nerve is attached by eight or ten rootlets to the medulla oblongata, below the glossopharyngeal nerve, in the groove between the olive and the inferior cerebellar peduncle. The fibres of the vagus nerve are connected to four nuclei in the medulla oblongata, viz. dorsal nucleus, nucleus ambiguus, nucleus of the tractus solitarius, and spinal nucleus of the trigeminal nerve. The rootlets of the nerve unite, and form a flat cord which passes below the flocculus of the cerebellum to the jugular foramen, through which it leaves the cranium. In emerging through this opening, the vagus nerve is accompanied by and contained in the same sheath of dura and arachnoid mater as the accessory nerve. In this situation the vagus nerve presents a well-marked enlargement, named the superior ganglion.

After its exit from the jugular foramen the vagus nerve enlarges into a second swelling, named the inferior ganglion which lies anterior to the anterior arch of the first cervical vertebra. The vagus nerve then passes vertically down the neck within the carotid sheath, lying between the the internal jugular vein and internal carotid artery as far as the upper border of the thyroid cartilage. The vagus nerve then passes down into the thorax and abdomen. (Gray, 1980: 1076-1081.) Whether a subluxation of the upper cervical vertebrae can result in direct stimulation of the vagus nerve has still to be established.

2.4 EVIDENCE OF CHIROPRACTIC TREATMENT OF I.D.D.M.

The question of whether chiropractors or other practitioners can alleviate or cure certain diseases of internal organs by somatic manipulation is fraught with controversy. Even though it is a central belief of chiropractic philosophy that treatment is not given per se for any disorder directly, many, if not most chiropractors, report anecdotally that patients with many types of visceral disorders often improve under their care. (Dhami and DeBoer, 1980: 115.) Whether this is due to the manipulation or the natural history of the disease, is not clear.

The rationale for the spinal intervention in the management of visceral conditions is based upon empiricism; that is, it is based on practical experience, rather than on scientific evidence provided by randomised controlled studies (Jamison et al. 1992: 172).

Mannino (1979: 225) may have been the first to demonstrate a direct or indirect effect of somatic stimulation on endocrine function. He measured serum aldosterone levels in 10 normotensive and 35 hypertensive patients. He reported a significant drop in aldosterone following osteopathic spinal manipulative therapy, but not after sham manipulations. Further details regarding this study were not furnished.

Chronic hyperactivity of the innervating sympathetic pathways seems to be a prevailing theme in many clinical conditions, involving many organs and tissues (Korr, 1978: 229).

Long-term exposure to osteopathic theory and practice as well as research experience in related fields has led Korr (1978: 229) to the following hypotheses:

- (i) Long term hyperactivity of particular sympathetic pathways is deleterious to the target tissues and may indeed have a rather general clinical significance.
- (ii) Clinical manifestations are determined by the organs or tissues which are innervated by the hyperactive sympathetic neurons, each responding in its own way, even to the sympathetically induced vasoconstriction that may be a common factor.

- (iii) The high impulse traffic in selected sympathetic pathways may be related to musculoskeletal dysfunction, especially in the spinal and paraspinal area.

With respect to the above hypotheses proposed by Korr (1978), he does not go into any detail to explain what is meant by "a rather general clinical significance".

Although conservative medicine accepts the importance of the autonomic nervous system in achieving body homeostasis, it has yet to come to terms with the notion that the spinal joint fixation can deleteriously influence normal body reflexes with predictable results (Korr, 1978).

In his book, "The neurodynamics of the vertebral subluxation", Homewood (1963: 185) states that the subluxation of an upper cervical vertebral segment disturbs the arterial supply to and the venous drainage from the corresponding nerve roots and neuromeres. He adds that such disturbance of vascularity and nutritional status involves the upper neuromeres and the more distal portion of the medulla oblongata, thereby altering the irritability of the components. Homewood emphatically states that there can be little reason to doubt the likelihood that the controlling neuronal pools for visceral structures, located in the grey matter of the medulla, are caused to react in a manner productive of functional aberrations in the visceral structures supplied. He goes on to explain that the intimate relation of the cells of the Nucleus of the Spinal tract of the Trigeminal nerve with the fibres conducting noxious impulses into the cord by way of the

posterior nerve roots of upper cervical nerves and the intimate connection that exists between this nucleus and the Dorsal Nucleus of the Vagus facilitates the disturbance of Vagal function. He believes that through such neural connections and the alterations of irritability created by the fixations, the possibilities of visceral symptoms become very real and probable. He concludes that coupled with the clinical evidence there can be little doubt of the pernicious influence of the cervical subluxation upon visceral control by the vagus nerve. In this particular chapter of his book, Homewood (1963: 185) frequently makes mention of clinical evidence available to substantiate the contention that cervical subluxations result in inimical functions of the organs and structures supplied. However he never actually quotes any specific clinical evidence, let alone any scientific evidence.

The other important factor discussed by Homewood (1963: 187) is the presence, or absence, of fixations lower in the spine that may be influencing the sympathetic supply to the same viscus. He is of the opinion that a subluxation in the thoracic region, fifth to the ninth, may over-activate the release of glucose from the liver with a hyperglycaemia developing. Conversely, the fixation may inhibit the sympathetic supply and change the sympathetic-parasympathetic balance with the parasympathetics being in the ascendancy, resulting in hypoglycaemia with its disturbing influences upon the irritability of the nervous system generally. Such conditions may result from upper cervical subluxations disturbing vagal function, or combinations of thoracic and cervical subluxations, which is perhaps the the more common circumstance. (Homewood 1963: 184-213.)

The affected tissues may in turn become secondary sources of abnormal afferent bombardment that helps sustain, intensify and spread the sympathetic hyperactivity (Lewis and Kellgren, 1939; Kellgren, 1940) (cited in Korr, 1978: 255).

In his study on the "Correlation of somatic dysfunction with visceral disease", Nicholas (1975: 426) concludes that the data demonstrated a more than inconsequential correlation between somatic dysfunction and visceral disease. A grand total of 286 patients were examined, with 73 separate disease entities being seen and classified into the following systemic groupings: respiratory disease, cardiovascular disease, gastrointestinal disease and genitourinary disease. Somatic dysfunction at the different spinal levels was noted for each system. I.d.d.m. was not covered in this study. With regard to Nicholas' conclusions, the question of cause and effect has to be considered. Nicholas (1975: 426) tries to establish the fact that there is a correlation between somatic dysfunction and visceral disease, but he does not suggest which was a consequence of the other. Did the somatic dysfunction cause the visceral disease or was the somatic dysfunction as a result of a chronic visceral disease?

Gitelman and Fitz-Ritson (1984: 63-64) categorized subluxation-related visceral disorders into four groups:

- a) Vertebrogenic disorders with reflex changes which mimic visceral pathology.

- b) Acute visceral disorders which facilitate the development of associated spinal subluxations. Correction of subluxations constitutes symptomatic treatment; curative therapy needs to be directed at the visceral condition.

- c) Chronic visceral disorders in which viscerosomatic reflexes are subject to prolonged and repeated stimulation. Established pain patterns persist even after the primary disorder has been corrected.

- d) Chronic subluxations which, via their reflex pathways, are postulated to create an environment in which organ susceptibility to disease is enhanced. Persistent vertebrogenic lesions, via reflex pathways, are deemed to reduce the resistance of viscera to environmental insults.

Gitelman and Fitz-Ritson's (1984: 63-64) theories seem feasible, but that is exactly what their postulations are: theories. They fail to provide any scientific evidence to substantiate their claims.

The hypothesis underlying chiropractic intervention in visceral disorders is based upon the proposition that spinal adjustment can modify autonomic nervous system balance and/or activity. Bony subluxations are believed to affect somato-somatic, somato-visceral, viscerosomatic and viscerovisceral pathways (Faucet *et al.* 1980: 171-172).

The existence of a somatoautonomic reflex has been repeatedly demonstrated in animal experiments, and the notion that somatosympathetic pathways may become routes for aberrant neural transmission when adversely affected by spinal subluxation is familiar in chiropractic and osteopathic literature (Leach, 1980: 141-142; Haldeman, 1972: 28; Sato, 1980: 93).

Leach (1986: 141-142) quotes Tweed as having documented the effects of vertebral lesions on various organs. Changes in the pancreas (especially the islet of Langerhans) were induced by lesions at the levels of T8-10. Dozens of guinea pigs and rabbits were fed high, normal or low-carbohydrate diets. Some were subjected to various lesions, but many had lesions of unknown origin prior to the experiment (these lesions were, nevertheless, identified by x-ray and palpation). Others guinea pigs were utilized as controls. Tweed does not explain what is meant by a "lesion", nor does he describe how these lesions were induced. One may also question the reliability of using x-ray in the detection of these lesions, as well as the use of palpation. Chiropractors nor osteopaths are probably as proficient at palpating guinea pigs as they are at palpating humans.

All the animals were fasted for one day, prior to being sacrificed. Following a sharp blow to the head, a syringe previously rinsed with potassium oxalate solution was thrust through the chest wall into the heart. Generally, 6-9cc of blood were withdrawn and placed in a bottle containing some potassium oxalate crystals. Tissue of the pancreas was prepared for slides.

Tweed (1931) (cited in Leach, 1986: 141-142) found that blood sugar in normal control high-carbohydrate-diet guinea pigs varied from 100-125 mg/100 cc of blood. Blood sugar in the lesioned high-carbohydrate-diet guinea pigs, however, varied greatly from 82-166 mg/100 cc of blood. It is not made clear what the significance of such readings are.

In the progeny of the lesioned high-carbohydrate-diet guinea pigs, the blood sugar varied even more from 20-200 mg/100 cc of blood. Similar findings in the low-carbohydrate-diet control and lesioned guinea pigs and in the high-carbohydrate-diet control and lesioned rabbits appeared to verify that various vertebral lesions may exert a marked influence on blood sugar levels.

Microscopic examination of the islets of Langerhans and other pancreatic tissues in animals with corresponding vertebral lesions showed signs of atrophy, connective tissue proliferation, granulator degeneration and haemorrhagic areas.

No mention was made of whether or not similar pathology was seen in in the low- or high-carbohydrate-diet control groups.

Tweed suggested that the only probable explanation for these findings was that circulation to these glands was affected by vertebral lesions at the corresponding spinal levels.

According to Tweed (1931) (cited in Leach, 1986), the mechanism to explain the effect of vertebral lesions is that they affect the circulation of the various organs by way of the sympathetic nerves that control constriction (tone) of blood vessels, especially those of the viscera.

These somatosympathetic pathways apparently become routes for aberrant neural transmission when they are affected adversely by way of spinal fixation.

Wiles (1990: 380) also suggests a possible relationship between spinal dysfunction and visceral disorders. The classic argument has always been that spinal joint dysfunctions can reduce the efferent sympathetic discharge activity, although most evidence suggests segmental facilitation to be the most likely effect of a spinal joint dysfunction.

He suggests that the most plausible theory related to the "reduced neural output" concept is Korr's idea that long-term sympatheticotonia can be deleterious not only to target tissues but also to the afferent nerves themselves.

Wiles (1990: 380) states that an important distinction must be made between treating visceral disease and treating patients with visceral disease. Chiropractors have the therapeutic goal of spinal joint dysfunction correction which, in turn, normalizes neurological function and promotes the healing process. Subluxations are found in patients with, and without, visceral disease. For example, a non-diabetic patient may present for a check-up and spinal joint dysfunctions of T7-9 may be found and treated. Another patient may present with diabetes mellitus and, on examination, T7-9 spinal joint dysfunction may be found. These will also be treated, albeit more aggressively, because of the potential segmental relationship with the pancreas. (Wiles 1990: 380.)

Penn (1969) (cited in Hoag, Cole and Bradford, 1969: 357) discusses various methods of managing the diabetic patient in order to develop a normal biomechanical balance. He believes that it is extremely important to ensure normal functioning of the musculoskeletal system and that care should be taken to detect and correct any postural or gross structural abnormalities. Localized areas of restriction should be mobilized by manipulation to restore normal function. He suggests that in the early stages of diabetic therapy manipulative treatment to normalize motion and maintain the correction may be required daily, or two or three times a week. He does not, however, provide any evidence to support this suggestion, nor does he explain for what length of time the patient should be treated.

Perrin (1969) (cited in Hoag, Cole and Bradford, 1969:364) believes that disturbances in insulin secretion are not only due to pancreatic dysfunction, but also to influences exerted by the pituitary, midbrain, thyroid, adrenal and gonads. These influences can be widely disseminated by way of the neuro-endocrine control and regulatory mechanisms. He recommends that osteopathic lesions in areas segmentally related to any of these areas should be searched for and corrected. The changes so induced may not be dramatic, but normal function will be found to be important in the body responses of these patients to both the disease and its management. No evidence is provided to support his beliefs, and they may therefore be seen as speculative.

Hoag, Cole and Bradford (1969: 357) state that the islet of Langerhans, unlike other endocrine glands, receive various direct terminals from the vagi as well as the typical postganglionic fibres from both the parasympathetic and sympathetic divisions. Stimulation of the right vagus nerve appears to increase the output of insulin, but denervation of the pancreas results in little or no detrimental effect on islet activity. This suggests that nervous control of these cells is not essential for their function. This is a fairly strong claim which again is not adequately supported by evidence from any study.

However, in a vagotonic individual, it is theorised that that mechanism might well be hyperactive to the extent of producing hyperinsulinism, either a chronic state or as an acute symptomatic reaction to stress. This vago-insulin system is independent of the sympathetico-adrenal mechanism which is activated in emergencies.

Hoag, Cole and Bradford (1969: 364) further theorise that viscerosomatic and visceromotor reflexes from the pancreas may result in vague, generalized gastrointestinal activity as a secondary response. Paravertebral muscle rigidity is found in the muscles in the midthoracic segments, especially on the left side. An explanation for a "vague, generalised gastrointestinal activity" is not provided by the authors.

Sprovieri (1990: 77) explores the question of whether osteopathic manipulative therapy (OMT) can help treat the symptoms and complications of diabetes.

He believes that while OMT alone cannot control diabetes, many osteopaths believe that manipulation of the thoracic spine can influence the amount of insulin secreted by the pancreas. He quotes Cifala who explains that although using OMT to improve insulin production is theoretical, the treatment may work by normalizing circulation in the affected region and by stimulating somato-visceral reflexes via the sympathetic nervous system.

Korr (1978: 255) ventures to offer an explanation for the hypotheses which purport to link the clinical and experimental material and their apparent mechanisms to those at work in manipulative therapy. In view of the rich access of somatic afferents, via spinal and supraspinal pathways, to sympathetic neurons, it would be truly amazing if even relatively minor disturbances in motion of intervertebral or other joints, which are amenable to manipulative therapy, did not have autonomic and, therefore, circulatory, metabolic and visceral repercussions of some degree. He suggests that effective manipulation is that which results in the re-establishment of coherent patterns of afferent input such that local adjustive reflexes are once more appropriate and harmoniously integrated in the total, supraspinally-directed patterns of activity and adaptive response.

As one can see from the evidence supplied by a review of the literature, it is not clear whether the specific spinal joint dysfunctions found in insulin-dependent diabetics are the primary cause or merely secondary to pancreatic dysfunction.

It is also difficult to establish whether spinal manipulation will have a beneficial effect on insulin-dependent diabetics, as no randomised, controlled studies were found involving patients with i.d.d.m.

CHAPTER 3

3. MATERIALS AND METHODS

3.1 The data

3.1.1 The primary data

The primary data used in this study were gathered from the following sources :

- Case history form (Appendix 6).
- Physical examination form (Appendix 7).
- Cervical spine regional examination form (Appendix 8).
- Screening questionnaire (Appendix 1).
- Pathology laboratory blood test results for venous glucose and glycosylated haemoglobin which were recorded on a laboratory log form (Appendix 2).
- Patient daily logs (Appendix 3) of capillary glucose levels and units of insulin administered.
- Subjective Health Status Questionnaires (Appendix 4).
- S.O.A.P. note form (Appendix 9).

3.1.2 The secondary data

The secondary data needed to substantiate and explain the problems at hand were obtained using the following:

- books
- journal articles
- periodicals
- pamphlets.

Other secondary data needed in order to fulfil the requirements of sub-problem 3 will be obtained from the results and conclusions from sub-problems 1 and 2.

3.2 The Research Methodology - Methods, Techniques and Measurements

3.2.1 The Sample

The sample of 21 subjects entered into the study consisted of insulin-dependent diabetics whose diabetes had stabilized; that is those patients who had not been admitted to hospital in the past three months for their diabetes. The sample group was obtained by advertising in local newspapers in Durban, as well as from interviewing patients at the King Edward Diabetic Clinic and Beatrice Street Diabetic Clinic.

Twelve of the 21 subjects who completed the study were admitted after responding to the advertisements published in the Daily News and Natal Mercury newspapers (convenience sampling). Advertisements were also put up at various shopping centres and chemists (convenience sampling).

The remainder of the subjects were recruited by the researcher from the King Edward Diabetic Clinic and Beatrice Street Diabetic Clinic in Durban in May and June 1994. Admittedly, subjects obtained from these clinics were not convenience-sampled and represented a biased portion of the population of people in the Durban area with i.d.d.m. These subjects were generally from a lower socioeconomic background than those subjects who were already participating in the study. Very few of the patients at these clinics met with the requirements of the study as many were noninsulin-dependent and did not have glucometers of their own.

During the period of the study, a total of 57 subjects were interviewed. Of this total number, 33 subjects were admitted into the study, with 12 not completing the treatment. Four of these non-compliant subjects had already completed five to nine treatments when they dropped out of the study. The remaining eight subjects either received one treatment or their treatment program was not started. Six subjects had to discontinue their treatment owing to excessive work or study commitments. One subject developed upper thoracic pain after five treatments which was diagnosed as a posterior facet syndrome and treated accordingly. Another of the subjects was admitted to hospital after apparently suffering a mild heart attack.

3.2.2 The process of randomisation

Six possible groups of four were formed viz. 1) ECEC, 2) EECC, 3) CCEE, 4) CECE, 5) ECCE, 6) CEEC and were then numbered from 1 - 6. A dice was then thrown to establish the order in which the subjects would be allocated to either the experimental or control group. The letter "E" was representative of the experimental group, while the letter "C" represented the control group.

The dice was thrown six times, establishing the following order: EECC, CCEE, CCEE, CEEC, ECEC, ECCE. The sample group was randomly divided into either the experimental or control group as they were accepted into the study. Non-compliant patients were systematically replaced as and when new subjects entered into the study.

3.2.3 Method

The following steps were followed in the execution of the study:

- At the initial consultation, each subject was required to complete a screening questionnaire (Appendix 1) in order to establish whether they were eligible or not.
- It was then explained to each subject that they would be randomly allocated to either the treatment or placebo groups.
- Once the subjects had had the procedure explained to them to their satisfaction, each subject then read and signed the patient informed consent form (Appendix 5).

- The subjects were then given a subjective health status questionnaire (Appendix 4) to complete.
- The subjects were instructed to try to maintain their present diet and exercise programs as closely as possible so as ensure that these variables remained as constant as possible.
- At this stage a full case history and physical examination were performed on each subject and recorded on the Technikon Natal case history form (Appendix 6) and physical examination form (Appendix 7).
- Those subjects in the experimental group had a cervical spine regional examination performed on them, which was recorded on a cervical spine regional examination form (Appendix 8).
- X-ray requisition forms (Appendix 10) were then completed for those subjects in the experimental group, and cervical and thoracic spine x-rays were performed by the Radiographic Department at Technikon Natal. Reports of these x-rays were obtained from the resident radiologist before the subjects were treated. It was not ethically desirable nor necessary to x-ray those subjects in the control group. The x-rays were taken to exclude any contraindication to manipulation and not in order to make a diagnosis.
- Any subluxations located in the upper cervical (C0 - C3) or mid to lower thoracic (T7 - T12) region were recorded on the S.O.A.P. notes (Appendix 9) along with the techniques used in adjusting the subluxations.

- Subluxations located in these regions in the experimental group were then appropriately adjusted, depending on the type of subluxation detected.
- The subjects in the control group received the vacotron suction pads from the interferential current (IFC) unit over the level of the transpyloric plane; that is, level of L1 vertebra. Two pads were placed posteriorly approximately 15 centimetres either side of the spine and two placed just anterior to the coronal plane. The milliamperage was set on zero; the subjects were therefore receiving a placebo.
- Each subject received treatment once a week over the four month period. Penn (1969) (cited in Hoag, Cole and Bradford, 1969) suggested that in the early stages of diabetic therapy, manipulative treatment to normalise motion and maintain the correction might be required daily, or two or three times a week. He did not, however, provide any evidence to substantiate his statement and did not state for how long the patient should be treated. No other evidence describing frequency or period of treatment was found. It was therefore decided that, owing to the required blood tests, once a week would be adequate. A four-month treatment period was decided on owing to the fact that a minimum period of 60 days is necessary between glycosylated haemoglobin tests in order for the results to be reliable and valid.
- The subjects were given blood test request forms from Pillay, McIntosh and Partners laboratory every two weeks. Blood test request forms for glycosylated haemoglobin (HbA1c) and venous glucose were given to the subjects at the initial consultation,

eighth treatment and final treatment. Blood test request forms for venous glucose were given to the subjects at every second treatment. Results of these tests were sent to Technikon Natal Chiropractic Day Clinic.

- At the eighth and final treatments the patients were again required to fill in the subjective health status questionnaire. The subjective health status questionnaire was designed to assess the subjects' own perception of their health. The subjects' second and third responses to this questionnaire were to enable the researcher to ascertain to what degree this perception had changed over the period of the treatment.
- For ethical and medical reasons the subject's present treatment, viz. insulin therapy, was not altered in any way.

3.2.4 Measurements

The data were extracted from the following sources:-

- i) Case history and physical examination records.
- ii) Cervical spine regional examination records.
- iii) Venous blood glucose levels.
- iv) Glycosylated haemoglobin levels.
- v) Glucometer readings of capillary blood glucose levels (mmol/l)
- daily log.
- vi) Units of insulin per injection - daily log.
- vi) Subjective health status questionnaire.

3.3 The criteria governing the admissibility of the data

- Only data collected from compliant subjects were statistically analysed.
- These data were collected from the above-mentioned questionnaires and forms.
- Data collected from non-compliant subjects were not statistically analysed but were discussed separately.

3.4 Apparatus

The main piece of apparatus utilised was the interferential current (IFC) unit: Endomed-M 433 and Vacotron 436. This apparatus was used in the "treatment" of the control group. These units were manufactured by Enraf Nonius, P.O. Box 483, 2600, Al Delft, The Netherlands. They were supplied by Mediotronics Natal Pty (Ltd), 677 Umgeni Rd. Unit 1, Croydon Court, Stamford Hill, Durban.

3.5 The treatment of the data

3.5.1 Sub-problem 1

The first sub-problem is to evaluate the effectiveness of upper cervical and lower thoracic manipulation on the experimental group's glycosylated haemoglobin levels, venous and capillary glucose levels and units of insulin administered, as well as their subjective perception of their health in order to establish the extent of the relationship between these variables and spinal manipulation.

i) Data needed

The data took the form of numerical readings obtained from venous blood glucose and glycosylated haemoglobin laboratory reports from Drs Pillay, McIntosh and Partners. Venous blood glucose levels were tested every second week, while the blood glycosylated haemoglobin levels were tested every two months. The reason for testing the glycosylated haemoglobin levels only every two months is that the half-life of an erythrocyte containing the haemoglobin is 60 days. Any tests performed within this time period may not reflect any possible alteration in the glycosylated haemoglobin levels. Levels of 6% glycosylated haemoglobin are considered normal, 8% acceptable, and over 10% indicative of poor glycaemic control (Jamison, 1984: 17).

The rest of the data were presented in one of two forms. Firstly, it took the form of numerical readings of capillary blood glucose in mmol/l and units of insulin administered per injection. These data were located on log sheets completed daily by the subjects. The capillary glucose readings were recorded by the subjects before breakfast, lunch and dinner as well as just before going to bed. These readings were recorded at different times each day, with the units of insulin being recorded at every injection. The capillary glucose levels were grouped into groups of four and an average obtained.

Secondly, the data consisted of numerical responses given by the patients to the subjective health status questionnaire (Appendix 4) in order to describe their subjective perception of their health during the period of the treatment. This subjective health status questionnaire was completed by the patient at the initial consultation, eighth treatment and last treatment. This questionnaire was designed to establish to what degree the subjects' diabetes interfered with their lives and what possible long-term complications they experienced. The second and third readings provided an indication of any alterations that may have occurred over the period of treatment. The subjects' responses were according to a scale which indicated the frequency with which they experienced the signs and symptoms listed, if at all. These numerical readings were then totalled for each of the three questionnaires.

ii) Capturing and securing the data

The readings for the venous blood glucose and glycosylated haemoglobin (HbA1c) were received from the pathology laboratory. These readings were then recorded on a laboratory log sheet (Appendix 2). These data were then analysed using the Wilcoxon Signed Rank test in order to calculate the amount of fluctuation, if any, of blood glucose and glycosylated haemoglobin levels within the treatment group.

Each subject recorded daily readings of their capillary blood glucose levels and units of insulin injected. The capillary blood glucose levels were obtained from digital glucometer readings. The units of insulin administered were read from the novopen or syringe at the time of injection. The readings from the subjects' daily logs were analysed by means of time series. Means of the capillary blood glucose as well as the units of insulin injected were calculated per four-day period and then statistically analysed.

The totals from the subjective health status questionnaires (Appendix 4) were also analysed by means of the Wilcoxon Signed Rank test.

3.5.2 Sub-problem 2

The second sub-problem is to evaluate the effectiveness of de-tuned interferential current over the transpyloric plane on the control group's glycosylated haemoglobin levels, venous and capillary glucose levels and units of insulin administered, as well as their subjective perception of their health in order to establish the extent of the relationship between these variables and the placebo "treatment".

i) Data needed

The same data were used for the control group as were used for the experimental group.

ii) Capturing and securing the data

The same statistical methods were employed for the control group as were used for the experimental group.

3.5.3 Sub-problem 3

The third sub-problem is to integrate the results obtained from the experimental and control groups in order to determine what contribution chiropractic manipulation of the upper cervical and lower thoracic vertebrae makes in the management of i.d.d.m.

i) Data needed

These data were of a numerical form as well as the statistical interpretations and conclusions drawn from sub-problems 1 and 2. The data were located in the results of sub-problems 1 and 2 in Chapter 4. The data were obtained by simply referring back to the interpretation of the data gathered as well as the conclusions drawn from sub-problems 1 and 2.

ii) Capturing and securing the data

Comparisons were drawn between the experimental and control groups by using the Mann Whitney U-test. The two groups' glycoylated haemoglobin, venous glucose and subjective health status questionnaire responses were analysed in this manner. The two groups' capillary glucose and units of insulin injected could not be fitted to any statistical model, and so visual comparisons had to be made by using a line graph. A mean of every four days was calculated for each group for the four-month period. These means were subsequently plotted on a graph in order to observe any alterations in the readings during the treatment period.

3.6 General remarks

All of the data required were transferred from each subject's file to spreadsheets on Lotus 123. The information on these spreadsheets was then statistically analysed using the Statgraphics Plus Version 6 as supplied by Manugistics Inc.

All of the statistical analysis was performed at Technikon Natal with the assistance of Mr. Kevin Reich. The entire dissertation was typed using Wordperfect Version 5.1.

CHAPTER 4

4. THE RESULTS

4.1 SUB-PROBLEM 1

4.1.1 Glycosylated haemoglobin readings

TABLE 1A : Glycosylated haemoglobin readings for the experimental group

	Blood Test 1	Blood Test 2	Blood Test 3
HbA1c	10.77+/-3.072	10.31+/-2.326	10.65+/-2.512

The Wilcoxon Signed Rank test was used to analyse the three glycosylated haemoglobin readings.

Reading 1 and 2 : Large sample test statistic $z = 0.933564$

Two-tailed probability of equalling or exceeding $z = 0.350527$

Reading 2 and 3 : Large sample test statistics $z = 0.8664$

Two-tailed probability of equalling or exceeding $z = 0.386269$

Reading 1 and 3 : Large sample test statistic $z = 0.829288$

Two-tailed probability of equalling or exceeding $z = 0.406939$

No significant difference was noted between reading 1 and 2, reading 2 and 3, or reading 1 and 3.

4.1.2 Venous glucose readings.

The data were analysed using the Wilcoxon Signed Rank test.

TABLE 2A : Venous glucose readings for the experimental group

GLUCOSE 1	GLUCOSE 2	GLUCOSE 3	GLUCOSE 4
11.22+/-4.502	9.87+/-4.645	11.04+/-4.788	12.11+/-3.990

GLUCOSE 5	GLUCOSE 6	GLUCOSE 7	GLUCOSE 8
10.99+/-5.842	12.2+/-6.920	10.81+/-4.564	11.34+/-7.460

On analysis of the data, no significant change was noted between subsequent readings, nor over the entire treatment period of four months.

4.1.3 Capillary glucose readings.

No statistical model could be fitted to the data owing to the inconsistent nature of the readings obtained.

4.1.4 Units of insulin injected.

The data were analysed using time series.

TABLE 3A : Summary of fitted model for units of insulin in the experimental group.

PARAMETER	ESTIMATE	STD.ERROR	T-VALUE	P-VALUE
AR (1)	0.98662	0.05132	19.22617	0.00000
MEAN	55.50038	0.45334	122.42497	0.00000
CONSTANT	0.74277			

The first order Autoregressive model for the treatment group is:

$$Y_t = 0.987 Y_{t-1} + 0.743$$

No significant difference was noted over the four-month period.

4.1.5 Subjective health status questionnaire responses.

The three readings obtained were analysed by means of the Wilcoxon Signed Rank test.

Reading 1 and 2: Large sample test statistic $z = 1.6004$

Two-tailed probability of equalling or exceeding $z = 0.109511$

Reading 2 and 3: large sample test statistic $z = 0.355409$

two-tailed probability of equalling or exceeding $z = 0.722279$

Reading 1 and 3: Large sample test statistic $z = 1.15584$

Two-tailed probability of equalling or exceeding $z = 0.247745$

There was no significant difference between the 1st and 2nd, 2nd and 3rd, and 1st and 3rd readings.

4.2 SUB-PROBLEM 2

4.2.1 Glycosylated haemoglobin readings

The Wilcoxon Signed Rank test was used to analyse the three glycosylated haemoglobin readings.

TABLE 1B : Glycosylated haemoglobin readings for the control group

	Blood Test 1	Blood Test 2	Blood Test 3
HbA1c	10.92+/-3.099	11.02+/-2.802	10.04+/-2.749

Reading 1 and 2: Large sample test statistic $z = 0.829288$

Two-tailed probability of equalling or exceeding $z = 0.406939$

Reading 2 and 3: Large sample test statistic $z = 2.08955$

Two-tailed probability of equalling or exceeding $z = 0.0366577$

Reading 1 and 3: Large sample test statistic $z = 2.29341$

Two-tailed probability of equalling or exceeding $z = 0.0218242$

There was a significant difference between HbA1c readings 2 and 3 ($p = 0.037$) and between HbA1c readings 1 and 3 ($p = 0.022$). There was no significant difference between HbA1c readings 1 and 2.

4.2.2 Venous glucose readings

The data was analysed using the Wilcoxon Signed Rank test.

TABLE 2B : Venous glucose readings for the control group

GLUCOSE 1	GLUCOSE 2	GLUCOSE 3	GLUCOSE 4
11.74+/-6.717	12.08+/-5.342	13.42+/-7.067	12.85+/-7.523

GLUCOSE 5	GLUCOSE 6	GLUCOSE 7	GLUCOSE 8
13.78+/-9.053	13+/-6.771	11.04+/-3.766	9.56+/-4.885

No significant difference was noted between subsequent readings, nor over the four-month period.

4.2.3 Capillary glucose readings

No statistical model could be fitted to the data owing to the inconsistent nature of the readings obtained.

4.2.4 Units of insulin injected

The data were analysed using time series.

TABLE 3B : Summary of fitted model for units of insulin in the control group.

PARAMETER	ESTIMATE	STD.ERROR	T-VALUE	P-VALUE
AR (1)	1.01161	0.03505	28.86210	0.00000
MEAN	57.78517	0.36611	157.83462	0.00000
CONSTANT	-0.67098			

The first order Autoregressive model for the control group is:

$$Y_t = 1.011 Y_{t-1} - 0.671$$

No significant difference was noted over the four-month period.

4.2.5 Subjective health status questionnaire responses

The three readings obtained were analysed by means of the Wilcoxon Signed Rank test.

Reading 1 and 2: Large sample test statistic $z = 1.22315$

Two-tailed probability of equalling or exceeding $z = 0.221271$

Reading 2 and 3: Large sample test statistic $z = 0.509647$

Two-tailed probability of equalling or exceeding $z = 0.610296$

Reading 1 and 3: Large sample test statistic $z = 1.01929$

Two-tailed probability of equalling or exceeding $z = 0.308062$

There was no significant difference between the 1st and 2nd, 2nd and 3rd, and 1st and 3rd readings.

4.3 SUB-PROBLEM 3

4.3.1 Glycosylated haemoglobin readings

In comparing the glycosylated haemoglobin readings for the experimental and control groups, the Mann Whitney U- test was used.

Reading 1 (for experimental and control group): Large sample test statistic $z = 0.422645$

Two-tailed probability of equalling or exceeding $z = 0.672551$

Reading 2 (for experimental and control group): Large sample test
statistic $z = 0.457865$

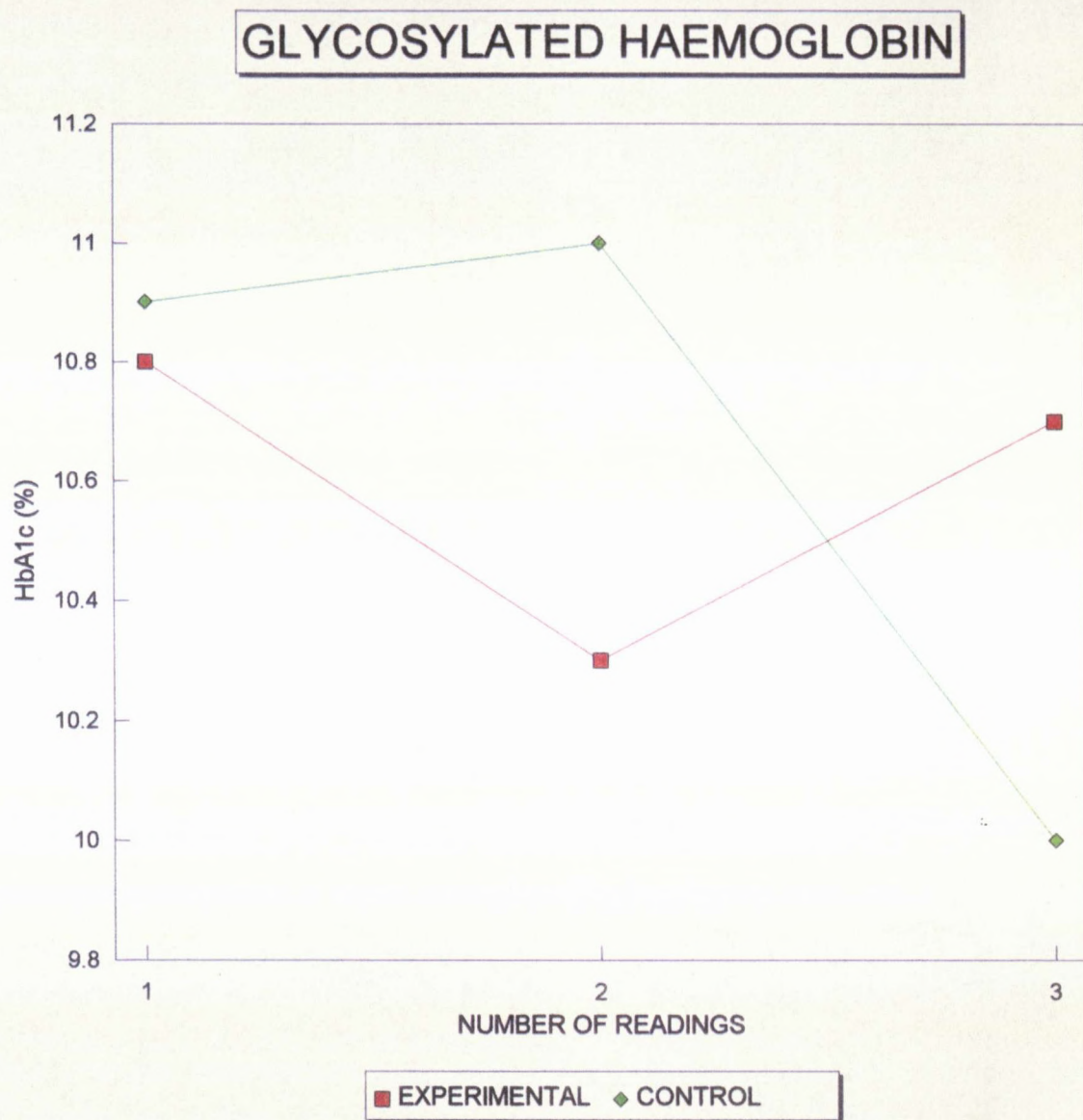
Two-tailed probability of equalling or exceeding $z = 0.647046$

Reading 3 (for experimental and control group): Large sample test
statistic $z = 0.529349$

Two-tailed probability of equalling or exceeding $z = 0.59656$

No significant difference was noted between the readings obtained for
the experimental and control groups.

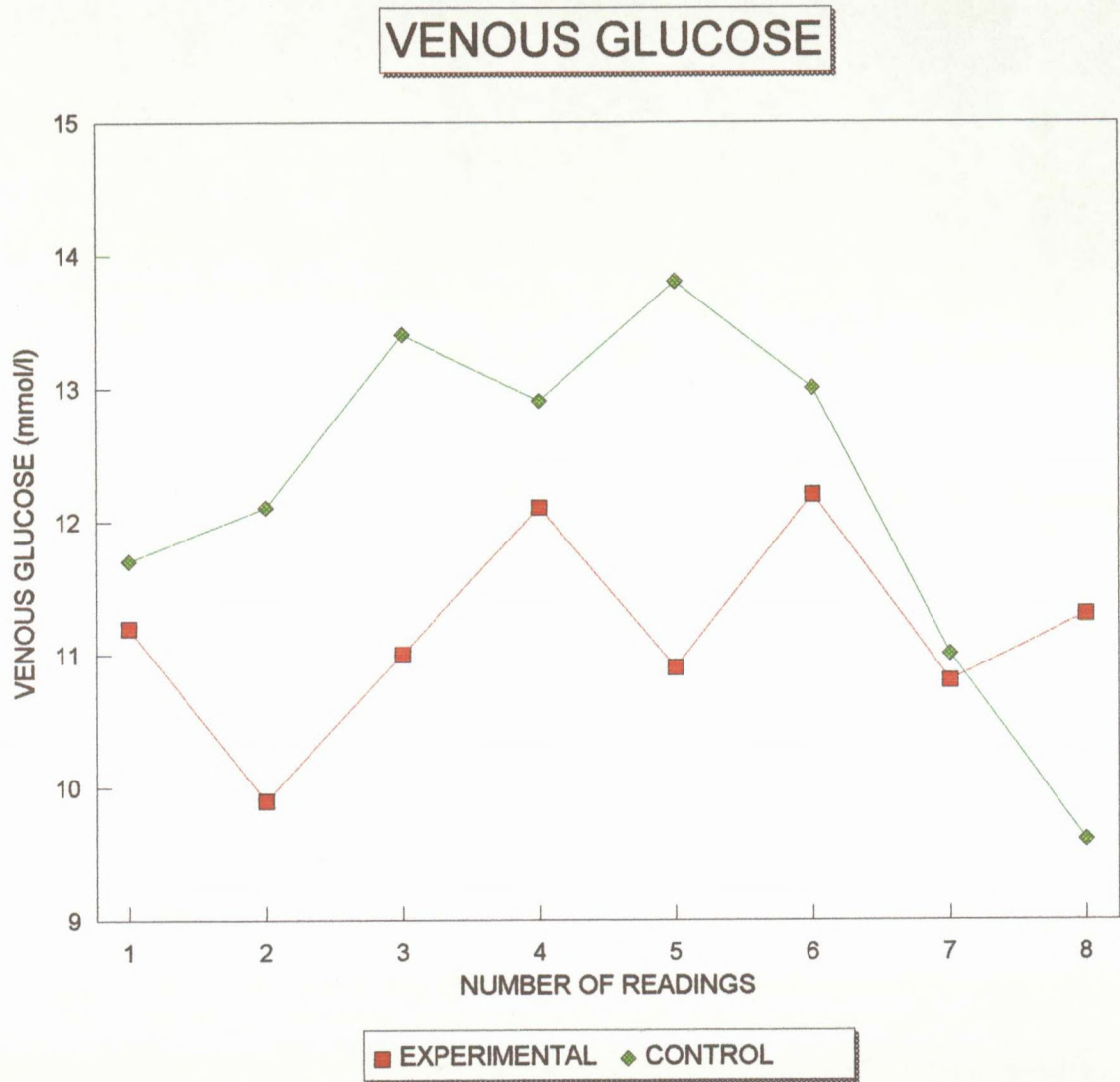
FIGURE 3 : COMPARISON OF GLYCOSYLATED HAEMOGLOBIN READINGS FOR THE EXPERIMENTAL AND CONTROL GROUPS.



4.3.2 Venous glucose readings

Owing to the fact that no statistical model could be fitted to compare the data obtained, a graph was used to depict any alterations that may have occurred during the four-month period of the study.

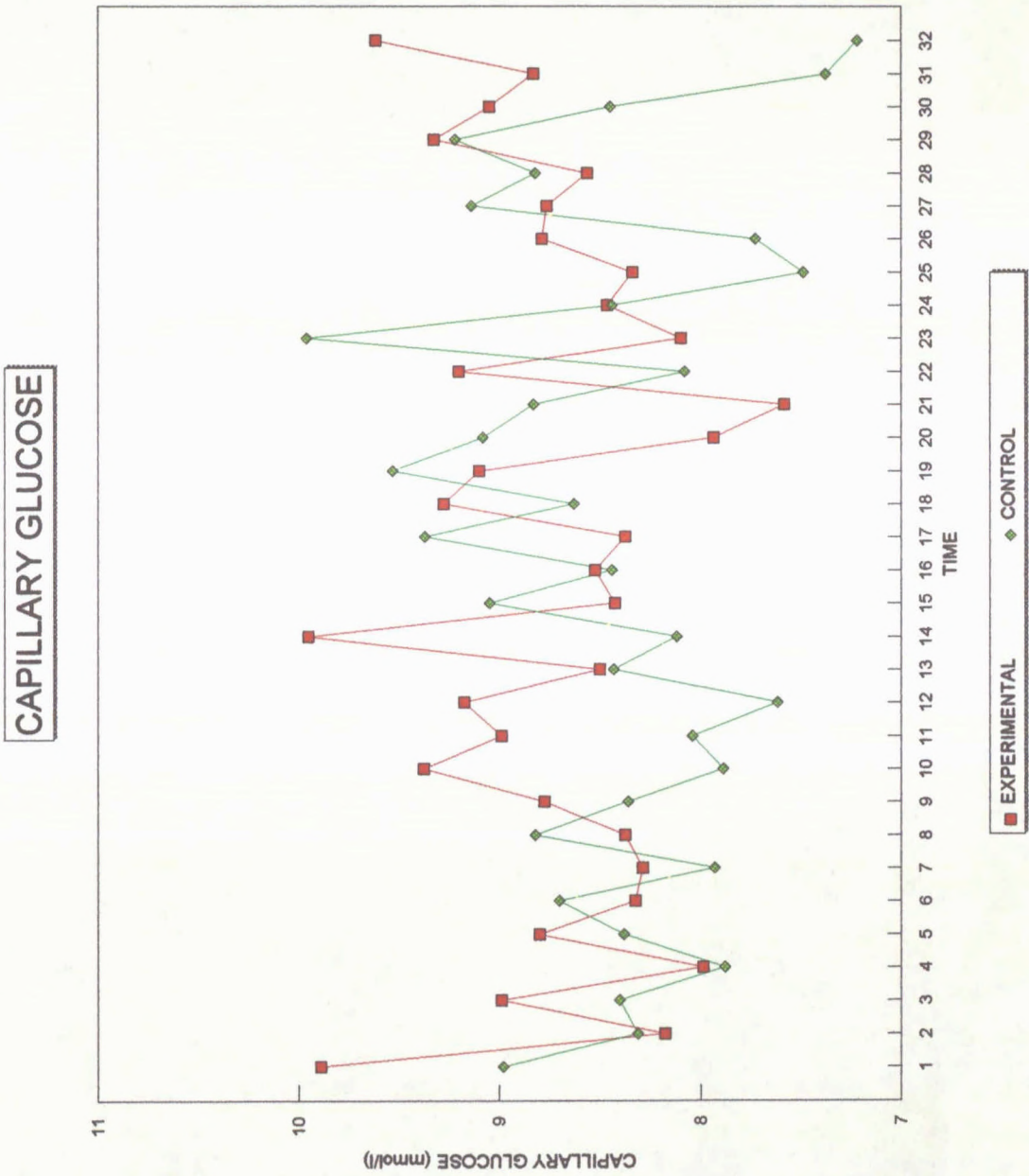
FIGURE 4: COMPARISON OF VENOUS GLUCOSE READINGS FOR THE EXPERIMENTAL AND CONTROL GROUPS



4.3.3 Capillary glucose readings

A graph was also used to illustrate any possible differences between the experimental and control group as no statistical model could be fitted to compare the two sets of data.

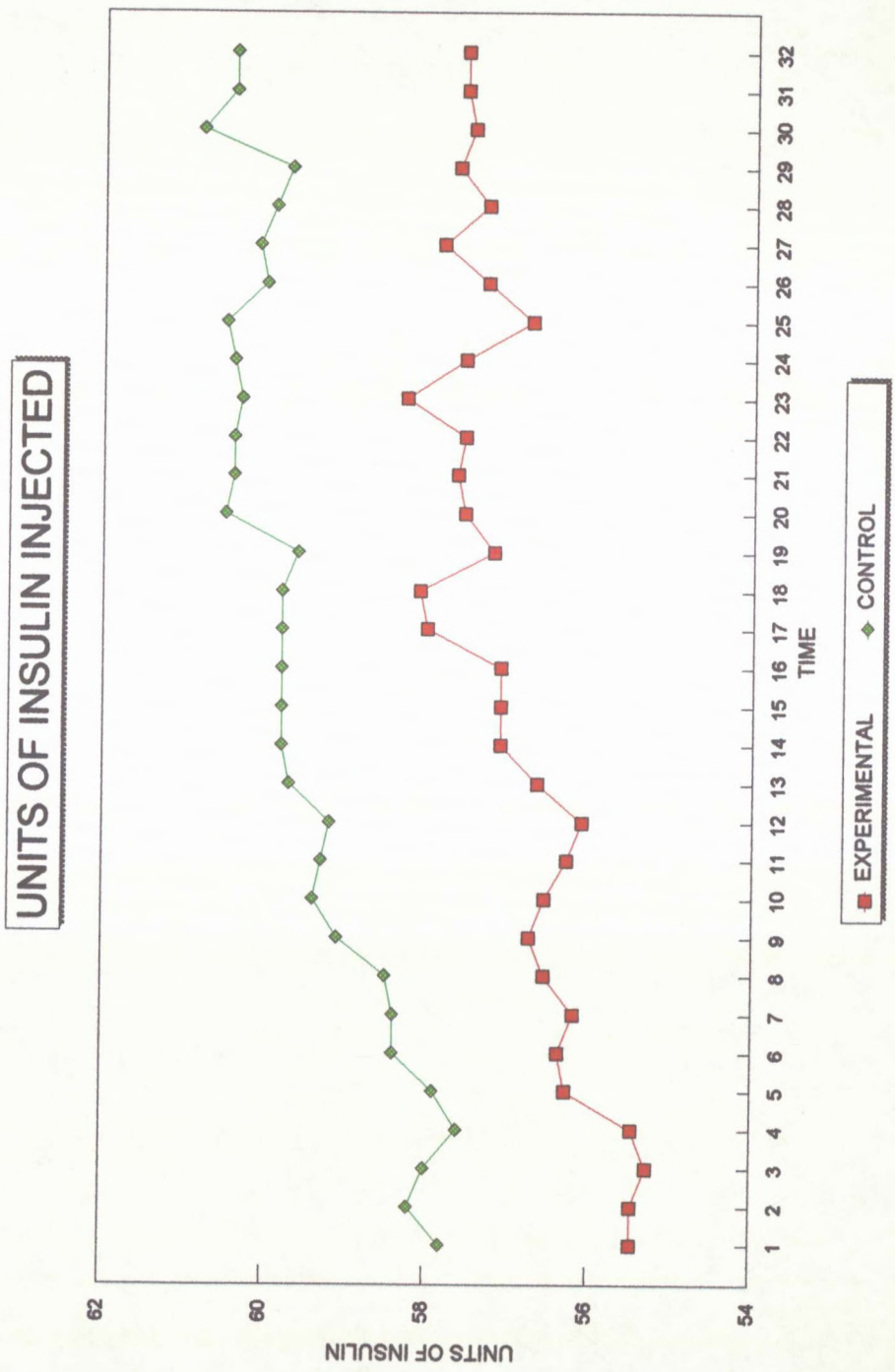
FIGURE 5:
COMPARISON OF CAPILLARY GLUCOSE READINGS FOR THE EXPERIMENTAL
AND CONTROL GROUPS



4.3.4 Units of insulin injected

A comparison of the units of insulin injected was best explained by means of a graph owing to the fact that a statistical model could not be used to compare the sets of data of the two groups.

FIGURE 6:
COMPARISON OF UNITS OF INSULIN INJECTED FOR THE
EXPERIMENTAL AND CONTROL GROUPS



4.3.5 Subjective health status questionnaire responses

The Mann Whitney U-test was again used to make a comparison between the responses of the experimental group and control group.

Reading 1 (for experimental and control group): Large sample test statistic $z = 1.40973$

Two-tailed probability of equalling or exceeding $z = 0.158619$

Reading 2 (for experimental and control group): Large sample test statistic $z = 1.23512$

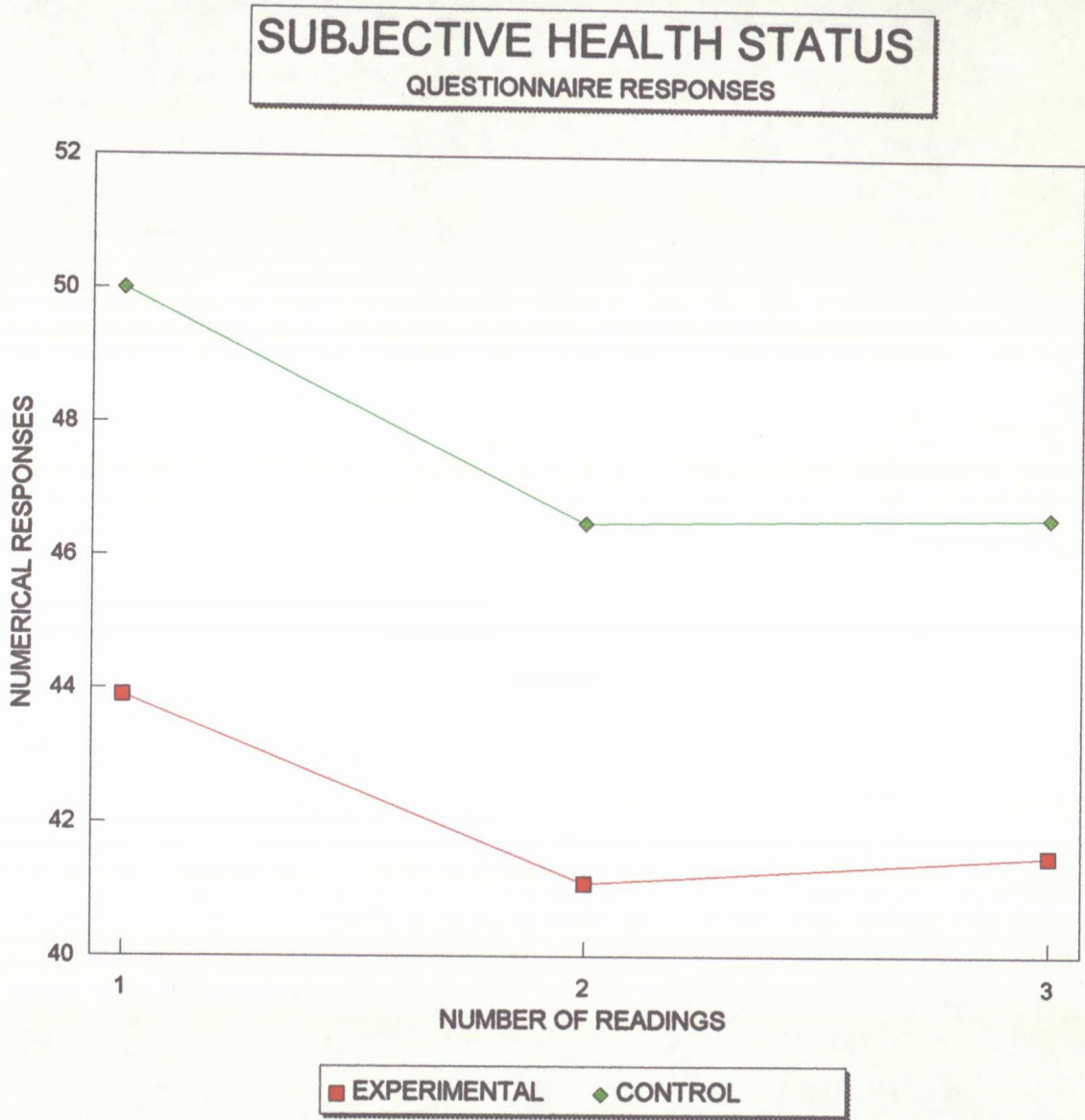
Two-tailed probability of equalling or exceeding $z = 0.216784$

Reading 3 (for experimental and control group): Large sample test statistic $z = 1.16607$

Two-tailed probability of equalling or exceeding $z = 0.243586$

Again, no statistically significant difference was noted between the responses of the two groups.

FIGURE 7: COMPARISON OF THE S.H.S.Q. RESPONSES FOR THE EXPERIMENTAL AND CONTROL GROUPS



On account of the above results, none of the hypotheses could be accepted and therefore had to be rejected.

4.4 The Screening Questionnaire

TABLE 4: Demographic data obtained from the screening questionnaire

QUESTION	EXPERIMENTAL	CONTROL
AGE	17 - 63	20 - 64
SEX	M = 5 F = 6	M = 4 F = 6
RACE	White 5 Indian 4 Black 2	White 3 Indian 7
OCCUPATION	Pensioner 1 Student 1 Housewife 1 Unemployed 2 Scholar 1 Comput. operat. 1 Secretary 1 Valuator 1 Foreman 1 Medical rep. 1	Pensioner 2 Student 1 Clerk 1 Housewife 5 Lecturer 1
AGE AT DIAGNOSIS	15 - 53	5 - 44

QUESTION	EXPERIMENTAL	CONTROL
FAMILY HISTORY OF I.D.D.M.	Grandmother 2 Son 1 Father 1 Brother 1	Father 1 Uncle 1 Brother 1 Sister 1 Son 1
NO. OF INJECTIONS PER DAY	4 per day = 8 3 per day = 1 2 per day = 2	4 per day = 6 2 per day = 2 1 per day = 2
INSULIN INJECTED	Actrapid and Protophane = 5 Actrapid and Humulin = 3 Monotard and Activad = 1 Lyspro and Humulin = 1 Actraphane = 1	Actrapid and Protophane = 4 Actrapid and Humulin = 1 Actrapid and Ultratard = 1 Actraphane = 2 Protophane = 2
PRESUMED CAUSE	Genetic = 1 Stress = 2 Tonsillitis = 1 Unknown = 5 Excess eating = 1	Genetic = 5 Unknown = 3 Depression = 1 Myocardial infarction = 1

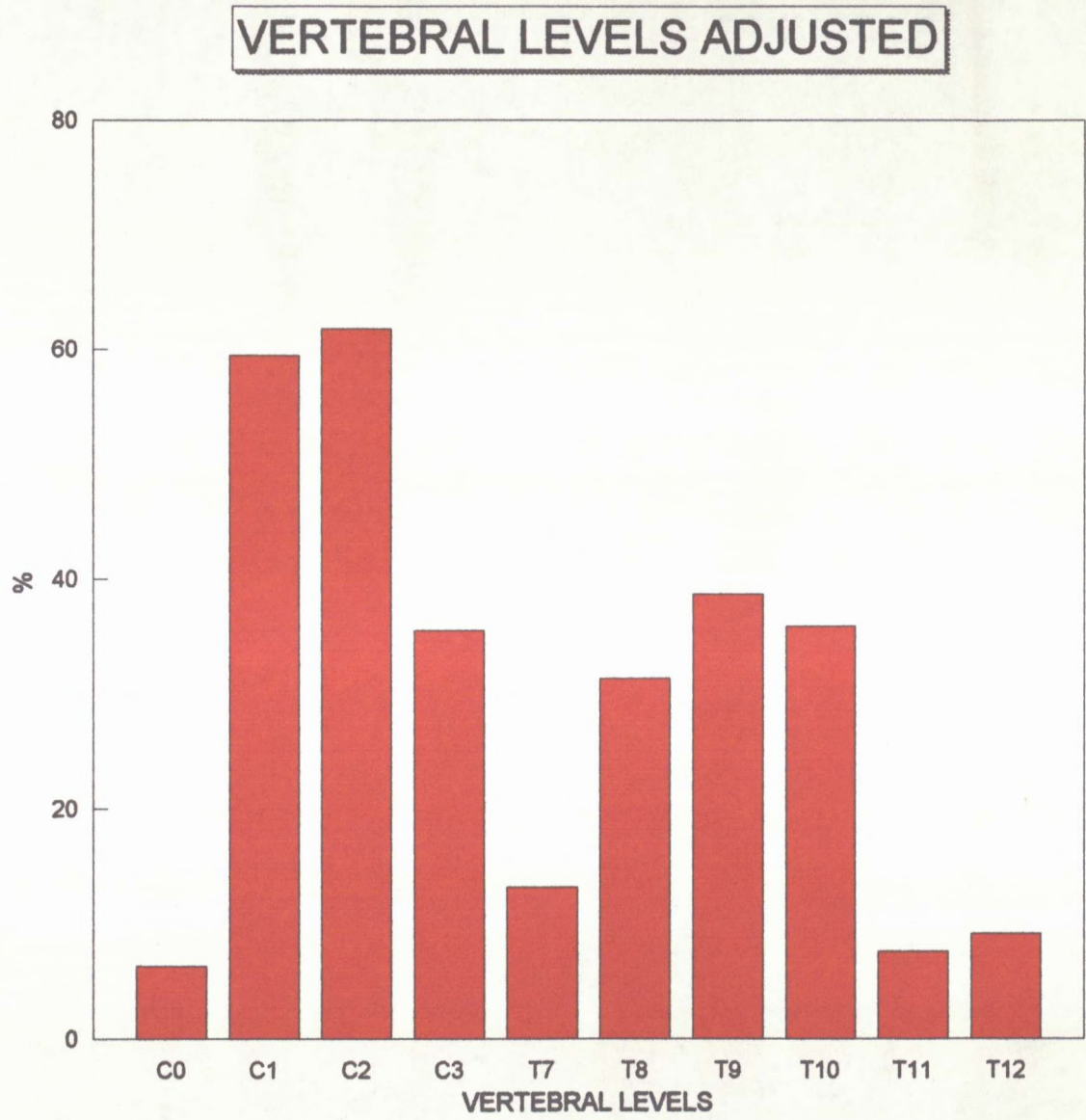
QUESTION	EXPERIMENTAL	CONTROL
STRESS AT TIME OF DIAGNOSIS	Divorce = 2 Moved school = 1 Family death = 1 Back injury = 1 None = 6	Family death = 2 Divorce = 2 Back injury = 1 None = 5
NO. OF YEARS SINCE DIAGNOSIS	2 - 40	7 - 32

4.5. Vertebral levels adjusted in the experimental group

Although the adjustment of vertebral levels was not mentioned in the sub-problems, it was felt that recording these vertebral levels might be of some benefit in determining whether or not the correct vertebral regions, viz. C0 - C3 and T7 - T12, were adjusted. No previous study was found to suggest which vertebral levels should be adjusted.

In his experiment on the effects of vertebral lesions on various organs, Tweed (1931) (cited in Leach 1986) explains how changes in the pancreas were induced by lesions at the levels of T8 - T10. Wiles (1990) suggested that T7 - T9 should be adjusted in patients with i.d.d.m.

FIGURE 8: VERTEBRAL LEVELS ADJUSTED IN THE EXPERIMENTAL GROUP



CHAPTER 5

5. DISCUSSION OF THE RESULTS

5.1 Introduction

The results of this study will be discussed under each variable, viz. glycosylated haemoglobin readings, venous glucose readings, capillary glucose readings, units of insulin injected, and subjective health status questionnaire responses. Sub-problems 1 and 2 will be discussed together, while sub-problem 3 will be discussed separately.

As discussed earlier, 21 subjects completed the four-month treatment period. There were 10 subjects in the control group and 11 subjects in the experimental group. This study may therefore be regarded as a pilot study, owing to the small sample size.

5.2 DISCUSSION OF THE RESULTS FROM SUB-PROBLEM 1, 2 AND 3

5.2.1 Glycosylated haemoglobin readings

The three glycosylated haemoglobin readings obtained from the two groups were analysed using the Wilcoxon Signed Rank test. In the experimental group, no statistically significant difference was seen between the 1st and 2nd, 2nd and 3rd, and 1st and 3rd readings. However, in the control group a significant difference was noted between the 2nd and 3rd ($p = 0.037$) and the 1st and 3rd ($p = 0.022$)

readings. The reason for this difference is not understood as the variable being measured was of an objective nature and was not subjective. The objective nature of these readings should have nullified the expected 30% improvement found in placebo groups.

Several other variables may have been responsible for this unexpected alteration in readings. Although the subjects were instructed to maintain their present diet and exercise program, it would have been virtually impossible for them to have done so over the four-month period. Other reasons may have included illnesses from which the subjects suffered, or stress induced by pressures at work, personal relationships, financial difficulties or concerns regarding their health. There are still more factors, which are discussed in section 2.2.7, which could have affected the subjects' readings. It is, however, difficult to relate these changes in the glycosylated haemoglobin readings to the above-mentioned variables as they would have had an influence on both the experimental and control groups.

Another possibility is that certain acupuncture points may have been stimulated by the suction pads from the Vacotron. These pads were placed posteriorly approximately 15 centimetres either side of the spine and 2 placed just anterior to the coronal plane. This area corresponds with Liver 13 which is located at the free end of the eleventh rib. This acupuncture point is one of the ten points suggested by Jayasuriya (1982) in the treatment of diabetes mellitus.

The Mann Whitney U-test was used to compare the glycosylated haemoglobin readings of the two groups. The graph (Fig. 3) which compares the experimental and control groups' readings clearly illustrates the difference between the two over the four-month period. It appears, however, that over the first two months there is a greater decrease in the HbA1c readings of the experimental group than in the control group. This trend, however, reverses in the last two months with the control group's readings markedly decreasing and the experimental group's readings increasing.

5.2.2 Venous glucose readings

The readings for both the experimental and control groups were analysed using the Wilcoxon Signed Rank test. No significant decrease in the subjects' venous glucose readings were noted over the four-month period for either of the groups.

As one can see from the graph (Fig. 4), there is no distinct trend in either of the groups. Initially one can see a distinct decrease in the experimental group between the first and second readings but the following six readings are somewhat irregular. In the control group, on the other hand, there is an initial steady increase until the fifth reading, after which there is a steady decline from 13,78 to a level of 9,56. Despite these visual alterations, once a statistical method was fitted to the data, it revealed no significant differences within either group.

This irregularity in readings is similar to the findings of Tweed's study (1931)(cited in Leach, 1986) which found that blood sugar in normal control high-carbohydrate-diet guinea pigs varied from 100-125 mg/100 cc of blood. Blood sugar in the lesioned high-carbohydrate-diet guinea pigs, however, varied greatly from 82-166 mg/100 cc of blood.

5.2.3 Capillary glucose readings

Owing to the irregular nature of the capillary glucose readings it was necessary to plot them on a graph as the data could not be analysed by means of the time series analysis. Both of the groups' readings were so irregular that not even a visual trend could be seen on the graph (Fig.5).

5.2.4 Units of insulin injected

Time series analysis was used when analysing the units of insulin injected for each group. No statistically significant difference was noted.

As can be seen on the graph (Fig. 6), there was, however, a general upward trend, with both the experimental and control groups' units of insulin injected increasing over the four-month period. The overall increase in both groups appears to be relative to one another, despite the fact that the control group's units of insulin were initially higher. Both groups showed an increase in the amount of insulin injected, which further illustrates the poor control by both groups.

5.2.5 Subjective health status questionnaire responses

The Wilcoxon Signed Rank test was again used to analyse the S.H.S.Q. responses. Again, no significant difference was noted in either of the two groups.

The graph (Fig. 7) which compares the two groups' S.H.S.Q. responses does, however, reveal that initially both groups' responses decrease and then reach a plateau over the last two months.

It is quite noticeable that in the figures 4, 6 and 7 that the control group's readings are consistently higher throughout the treatment period. Perhaps this was as a result of the randomisation process or owing to the small sample size.

5.3 SCREENING QUESTIONNAIRE

The screening questionnaire was initially used to obtain demographic data as well as to try to ascertain what aetiologic factors might be involved with each subject.

According to Berkow (1987: 1071), the age of onset of i.d.d.m. is often less than 30 years old. Tunbridge and Home (1991: 20) suggest that i.d.d.m. most commonly presents between the ages of four and 30 years, with a peak in the 10-15 year age group, although it may appear at any age. In this study the age of onset the subjects ranged from five - 53, with the average for the 21 subjects being 26.4.

This average falls well within the ranges described by Berkow(1987: 1071), as well as Tunbridge and Home (1991: 20).

According to Berkow (1987: 1071), there is usually a history of diabetes in the family (10% of diabetics have parents or siblings with the disease). Of the 21 subjects in this trial 47,6% revealed that they had grandparents, parents or siblings who were also diagnosed as having i.d.d.m.

Jones (1989: 9) believes that 90% of people with i.d.d.m. are born with an inherited increased risk of developing it. Although it was not the objective of this study to establish the aetiology of each subject's i.d.d.m., 28,6% believed that their i.d.d.m. had a genetic origin. As far as stress was concerned, 14% felt that their i.d.d.m. was due to emotional stress.

Jones (1989: 10) also discussed that severe stress can cause disturbances to the body's immune system and could theoretically make some people more likely to succumb to viral infection. When questioned on stress, 19% of the subjects revealed that they had been divorced or had had marital problems prior to diagnosis, 19% had had a death in the family, while 5% had moved home or school.

Kobberling and Tattersall (1982) indicate that the cases of i.d.d.m. with co-existent auto-immune diseases comprise less than 5% of the total i.d.d.m. population.

They suggest that a healthy individual may receive an environmental insult which triggers destruction of the beta cells of the pancreas. It is not quite understood what is meant by an "environmental insult", but may suggest a viral infection, hypersensitivity reaction or even physical trauma. As far as physical stress was concerned, 10% of the subjects experienced some form of spinal injury.

5.4 VERTEBRAL LEVELS ADJUSTED

As can be seen on the graph (Fig. 8), C1 and C2 were the most frequently adjusted levels with T9, T10, C3 and T8 being the next most frequent. The remaining levels were infrequently adjusted. The vertebral level C2 was adjusted most frequently at 61,8% of the treatments, C1 59,5%, T9 38,7%, T10 35,9%, C3 35,5% and T8 at 31,4% of the treatments.

CHAPTER 6

6.1 CONCLUSIONS AND RECOMMENDATIONS

6.1.1 Conclusions

As one can see from the results, it can be safely concluded that chiropractic does not significantly influence the signs and symptoms or the course of insulin-dependent diabetes mellitus. Chiropractic does not appear to contribute much to the management of i.d.d.m., either objectively or subjectively. The mere fact that the control group showed a significant improvement in their glycosylated haemoglobin readings in comparison to the experimental group is an indication of the irrelevance of chiropractic treatment of patients with i.d.d.m.

6.1.2 Recommendations

It is recommended that in order for this subject to be effectively studied, a substantially larger sample group will be required. A sample group of only 21 subjects is quite obviously not large enough from which to draw any reliable conclusions.

When referring back to the results, it is noticeable that positive changes did occur in the experimental group within the first two months of treatment. In particular, there was an initial decrease in the venous glucose readings and a decrease in the glycosylated haemoglobin readings during the first two months, as well as an improvement in the

S.H.S.Q. responses. However, the last two months saw a reversal of these findings and a general improvement in the control group, especially with the glycosylated haemoglobin. Perhaps it would be of benefit to have a two-month pre-test period during which the subjects could have the necessary blood tests and record their own capillary glucose and units of insulin. It might also be advisable to lengthen the treatment period and treat the patients more frequently, although this may create problems as far as compliance is concerned. This might better illustrate what effect the chiropractic treatment has on i.d.d.m.

Despite the danger of biasing the sample group, all of the subjects should be recruited from diabetic clinics. This is possibly the only way that a large enough sample group will be obtained. Trying to obtain subjects by means of newspaper advertisements is both ineffective and costly, and does not always seem to reach the required group. Interviewing patients at diabetic clinics is labour-intensive, but has a far greater success rate.

Perhaps one of the reasons for the poor response to the advertisements was because of the public's image of the role of chiropractic in the management of i.d.d.m. Another reason might have been that they feared being taken off their current insulin therapy.

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A P P E N D I X 1

SCREENING QUESTIONNAIRE

- 1. NAME : _____
- 2. AGE : _____
- 3. SEX : _____
- 4. D.O.B.: _____
- 5. ADDRESS: _____
- 6. TELEPHONE: _____

7. Which type of diabetes do you suffer from?
- i) Type I (Insulin-dependent)
 - ii) Type II (Non-insulin dependent)
 - iii) Diabetes insipidus

8. At what age were you diagnosed as being diabetic? _____

9. How long have you been Diabetic? _____ years _____ months

10. Do any other members of your family have diabetes?
If so, who? _____

11. How many times do you inject yourself per day? _____ per day.

12. How many units of insulin do you inject per injection? _____

13. What type/s of insulin do you inject? _____

14. How often do you visit your G.P. or Specialist for a check up?

15. What do you believe caused your diabetes? _____

16. Do you recall experiencing any physical or emotional stress just prior to being diagnosed with Diabetes Mellitus?

- i) Final examinations?
- ii) Death in the family?
- iii) Divorce of parents?
- iv) Accident, e.g. motor vehicle accident?
- v) Injury to the neck or back?
- vi) Head injury?
- vii) Other?

If so, please give a brief account.

17. Have you had any previous chiropractic or osteopathic treatment?
If so, when?

18. Have you ever suffered from mumps, infectious mononucleosis, rubella varicella, cytomegalo-virus or infectious hepatitis?
If so, when?

19. Is your diabetes well-controlled?
Describe briefly.

20. Are you a haemophiliac?

LABORATORY LOG

PATIENT'S NAME:				
M O N T H	VENOUS	BLOOD	GLUCOSE	GLYCOSYLATED HAEMOGLOBIN
	READING (1)		READING (2)	
JANUARY				
FEBRUARY				
MARCH				
APRIL				
MAY				
JUNE				
JULY				
AUGUST				
SEPTEMBER				
OCTOBER				
NOVEMBER				
DECEMBER				

A P P E N D I X 4

SUBJECTIVE HEALTH STATUS QUESTIONNAIRE

Kindly answer the questions below using the scale provided:

Scale : 1 = Never
 2 = Almost never
 3 = Occasionally
 4 = Almost always
 5 = Always

1. Do you ever lack energy? ()
2. Do you often find that you get depressed? ()
3. Does your diabetes interfere a great deal with your life? ()
4. Do you have any personal worries which are not caused by physical illness? ()
5. Do you have any particular fears regarding your diabetes? ()
6. Are you bothered by your aches and pains? ()
7. Are you prone to any infections? ()
8. Do you ever have any severe hypoglycaemic attacks? ()
9. Are you ever excessively thirsty? ()
10. Are you ever excessively hungry? ()
11. Do you pass excessive amounts of water? ()
12. Do you ever have any urinary disturbances? ()
13. Do you ever suffer from numbness or pins and needles? ()
14. Are you ever constipated? ()
15. Do you ever suffer from diarrhoea? ()
16. Do you ever have any dizzy spells? ()
17. Do you suffer from intermittent temperature changes in the arms or legs? ()
- 18) Do you ever have any visual disturbances? ()

A P P E N D I X 5

PATIENT CONSENT FORM

I have been asked to participate in a study to determine the effect of chiropractic care on insulin dependent diabetes mellitus. The information required of me will be my capillary glucose levels, urinary ketone levels and the number of units of insulin injected, all on a daily basis, during the period of the trial (six months) and for three months thereafter.

I undertake to report to the Technikon Day Clinic weekly for treatment and every two weeks for the drawing of blood samples. During the period of the trial, I undertake not to change my current diet and exercise programme.

Neither my name nor my house or business address or telephone number(s) will appear on any of the questionnaires or logs kept for this study and that any information recorded during this study will be kept strictly confidential.

I understand that I will be allocated either to the experimental or the control group. I have had this explained to me to my satisfaction. I understand and accept all the above conditions and agree to participate in this study.

Signature: _____

Date:

Signature: _____

Date:

Appendix 6

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC

CASE HISTORY

Patient: _____ Date # _____

File #: _____

X-ray #: _____

Age: _____ Sex: _____ Occupation: _____

Intern: _____ Signature: _____

FOR CLINICIAN'S USE ONLY

Initial visit clinician: _____

Signature: _____

Case History:

Examination:

Previous: TN
Other

Current: TN
Other

X-ray Studies:

Previous: TN
Other

Current: TN
Other

Clinical path. lab.:

Previous: TN
Other

Current: TN
Other

Case status:

PTT: Conditional: Signed off: Final sign out:

Recommendations:

Intern's case history

1. Source of history:
2. Chief complaint: (patient's own words)

3. Present illness:

Location

Onset

Duration

Frequency

Pain (character)

Progression

Aggravating factors

Relieving factors

Associated S & S

Previous occurrences

Past treatment and outcome

4. Other complaints:

5. Past history:

General health status

Childhood illnesses

Adult illnesses

Psychiatric illnesses

Accidents/injuries

Surgery

Hospitalizations

6. Current health status and life-style:

Allergies

Immunizations

Screening tests

Environmental hazards
(home, school, work)

Safety measures
(seat belts, condoms)

Exercise and leisure

Sleep patterns

Diet

Current medication

Tobacco

Alcohol

Social drugs

7. Family history:

Immediate family:

Age

Health

Cause of death

DM

Heart disease

TB

HBP

Stroke

Kidney disease

CA

Arthritis

Anaemia

Headaches

Thyroid disease

Epilepsy

Mental illness

Alcoholism

Drug addiction

Other

8. Psychosocial history:

Home situation

Daily life

Important experiences

Religious beliefs

9. Review of systems:

General

Skin

Head

Eyes

Ears

Nose/sinuses

Mouth/throat

Neck

Breasts

Respiratory

Cardiac

Gastro-intestinal

Urinary

STANDING EXAMINATION.

Minor's sign

Skin changes

Posture

erect

Adam's

''Ranges of motion:

T/L spine: Flexion: 90 Fingers to floor
Extension: 50
R.lat.flex.: 30 Fingers down leg
L.lat.flex.: 30 Fingers down leg
Rot.to R.: 35
Rot.to L.: 35

Flex.

L.Rot.

R.Rot.

L.lat
flex.

R.lat.
flex.

Ext.

/ = pain-free limitation; // = painful limitation.

Romberg's sign.

Pronator drift.

Trendelenburg's sign.

Gait.

rhythm

balance

pendulousness

on toes

on heels

tandem

Half squat.

Scapular winging.

Muscle tone.

Spasticity/Rigidity.

Shoulder:

skin
symmetry
ROM - glenohumeral
scapulo-thoracic
acromioclavicular
elbow
wrist

Chest measurement

inspiration
expiration

Visual acuity

Breast examination:

Inspection:

skin
size
contour
nipples
arms overhead
hands against hips
leaning forward.

Palpation:

axillary lymph nodes.

SEATED EXAMINATION.

Spinal posture

Head

scalp
skull
face
skin

Eyes

conjunctiva
sclera
eyebrows
eyelids
lacrimal gland
nasolacrimal duct
alignment
corneal reflex
ocular movement

L
III IV VI

R
III IV VI

visual fields
accomodation
iris
pupils
red reflex
optic disc

vessels
general background
macula
vitreous
lens

Ears:

auricle
ear canal
drum
auditory acuity
Weber test
Rinne test

Nose:

external
internal
septum
turbinates
olfaction
Sinuses (frontal & maxillary):
tenderness
transillumination

Mouth and pharynx:

lips
buccal mucosa
gums and teeth
roof
tongue
inspection
movement
taste
palpation
pharynx
inspection
CN X

Neck:

posture
size
swelling
scars
discoloration
hair line

ROM:

Flexion: 45 chin to larynx
chin to sternum
Extension: 55 forehead parallel
to floor
L.lat.flex: 40
R.lat.flex: 40
L.rot.: 70
R.rot.: 70

Flex.

L.Rot.

R.Rot.

L.Lat.
flex.

R.lat.
flex.

Ext.

lymph nodes
trachea
thyroid
carotid arteries (thrills, bruit)
CN V
CN VII
CN VIII (nystagmus)
CN IX
CN XI
TMJ
Inspection
ROM
deviation
Palpation
crepitus
tenderness

Neurological:

Dermatomes

C5

C6

C7

C8

T1

Tendon reflexes

biceps

triceps

brachioradialis

Muscle strength

C5

C6

C7

C8

T1

Coordination:

point-to-point

dysdiadochokinesia

Thorax:

Chest:

Inspection:

skin

shape

respiratory distress

rhythm (respiratory)

depth "

effort "

intercostal/supraclavicular retraction

Palpation:

tenderness

masses

respiratory expansion

tactile fremitus

Percussion:

lungs (posterior)

diaphragmatic excursion

kidney punch

Auscultation:

breath sounds

vesicular

bronchial

adventitious sounds

crackles (rales)

wheezes (rhonchi)

voice sounds

broncophony

whispered pectoriloquy

egophony

Cardiovascular:

auscultation (aortic murmurs)

Allen's test

SUPINE EXAMINATION

JVP

PMI

auscultation heart (L.lat.recumbent)

respiratory excursion

percussion chest (anterior)

breast palpation

The abdomen:

Inspection:

skin

umbilicus

contour

peristalsis

pulsations

hernias (umbilical/incisional)

Auscultation:

bowel sounds

bruit

Percussion:

general

liver

spleen

Palpation:

superficial reflexes

cough

light

rebound tenderness

deep

liver

spleen

kidneys

aorta

intra-/retro-abdominal wall mass

shifting dullness

fluid wave

Acute abdomen:

where pain began and now

cough

tenderness

guarding/rigidity

rebound tenderness

Rovsing's sign

psoas sign

obturator sign

cutaneous hyperaesthesia

rectal exam

Murphy's sign.

Male genitals and hernias.

Inspection:

skin
prepuce
glans
meatus
nits/lice
scrotum
inguinal/femoral bulges

Palpation:

penis (tenderness/induration)
testes
epididymis
inguinal canal
femoral canal
cremasteric reflex

Auscultation:

scrotal mass.

Peripheral vasculature:

Inspection:

skin
nail beds
pigmentation
hair loss

Palpation:

pulses - radial, brachial, femoral, popliteal, post.tibial,
dorsalis pedis

lymph nodes - epitrochlear, femoral (horizontal & vertical)
temperature (feet & legs)

Manual compression test

Retrograde filling (Trendelenburg) test

Arterial insufficiency test

Musculoskeletal:

ROM

hip

flex. 90/120

ext. 15

abd. 45

add. 30

int rot 40

ext rot 45

knee

flex. 130

ext. 0/15

ankle

plantar flex 45

dorsiflex 20

inversion 30

eversion 20

leg length

Neurological:

dermatomes

L1

L2

L3

L4

L5

S1

muscle strength

hip flexion

knee extension

ankle dorsiflexion

plantar flexion

tendon reflexes

patellar

Achilles

plantar reflex

Rectal examination:

Inspection

sacrococcygeal & perianal areas

Palpation

sphincter tone

tenderness

induration

nodules

prostate

seminal vesicles

Mental status

Appearance and behaviour:

level of consciousness

posture and motor behaviour

dress, grooming, personal hygiene

facial expression

affect

Speech and language:

quantity

rate

volume

fluency

aphasia (prn)

Mood

Thought processes (logical, relevant, organized)

Memory and attention:

orientation (time, place, person)

remote memory

recent memory

new learning ability

Higher cognitive functions:

information and vocabulary (general & specialised knowledge)

abstract thinking.

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC.

REGIONAL EXAMINATION -- CERVICAL SPINE.

PATIENT: _____

FILE # : _____ DATE: _____

INTERN/RESIDENT: _____

SUPERVISING CLINICIAN : _____

OBSERVATION :

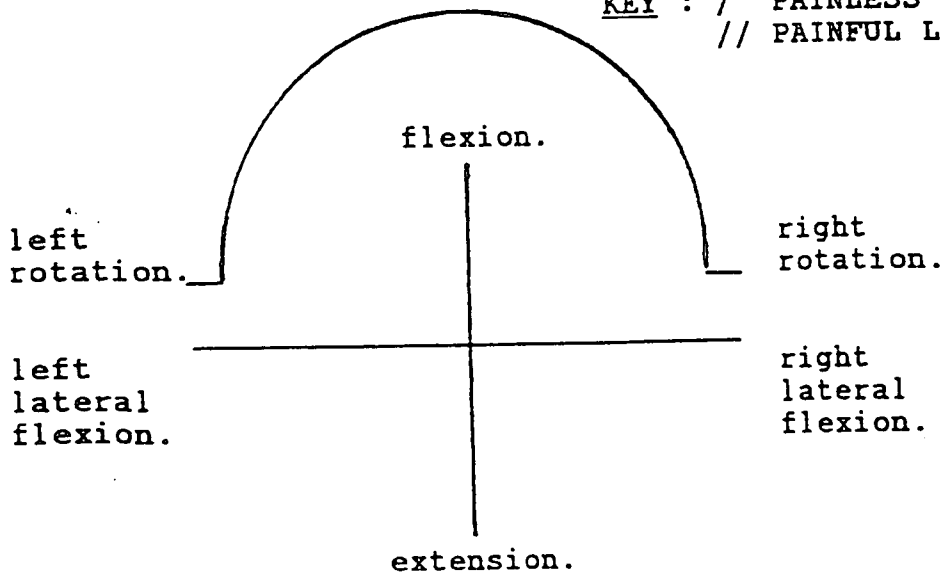
Posture
Swellings
Scars
Discoloration
Hair Line
Bony and soft tissue contours

Shoulder position:
Left =
Right =
Muscle spasm
Facial expression

RANGE OF MOTION:

Flexion = 45 degrees.
Extension = 70 degrees.
L/R Rotation = 70 degrees.
L/R Lateral flexion = 45 degrees.

KEY : / PAINLESS LIMITATION.
// PAINFUL LIMITATION.



PALPATION : lymph nodes.
trachea.
thyroid gland.

ORTHOPAEDIC EXAMINATION :

Tenderness

Active MF Trigger Points :

- SCM.
- Trapezius.
- Scaleni.
- Levator Scapulae.
- Posterior Cervical musculature.

Doorbell Sign

Kemp's Test

Cervical Distraction

Halstead's Test

Hyperabduction Test (Wright's)

Shoulder abduction Test

Dizziness rotation Test

Brachial Plexus Tension

Cervical Compression

Lateral Compression

Adson's Test

Costoclavicular Test

Eden's (traction) Test

Shoulder depression Test.

Lhermitte's Sign

O'Donoghue Manoeuvre

Remarks : _____

NEUROLOGICAL EXAMINATION :

DERMATOMES: Left; Right. MYOTOMES: Left; Right. REFLEXES: Left; Right.

C2		C1		C5	
C3		C2		C6	
C4		C3		C7	
C5		C4			
C6		C5			
C7		C6			
C8		C7			
T1		C8			
		T1			

VASCULAR :

LEFT.

RIGHT.

BLOOD PRESSURE.

CAROTIDS.

SUBCLAVIAN ARTERIES.

WALLENBERG'S TEST.

_____	_____
_____	_____
_____	_____
_____	_____

COMMENTS: _____

MOTION PALPATION :

Jt. play		Left						Right						Jt. play	
P/A	Lat	Fle	Ext	LF	AR	PR		Fle	Ext	LF	AR	PR	P/A	Lat	
							C0								
							C1								
							C2								
							C3								
							C4								
							C5								
							C6								
							C7								
							T1								
							T2								
							T3								
							T4								

Appendix 9

SUBJECTIVE OBJECTIVE ASSESSMENT PLAN
(S.O.A.P.) FORM

PATIENT NAME: _____ FILE#: _____ PAGE# _____

DATE: _____ VISIT# _____ INTERN: _____ CLINICIAN: _____

S:

A:

O:

P:

SPECIAL ATTENTION TO:

NEXT APPOINTMENT:

DATE: _____ VISIT# _____ INTERN: _____ CLINICIAN: _____

S:

A:

O:

P:

SPECIAL ATTENTION TO:

NEXT APPOINTMENT:

Appendix 10

REQUISITION FOR RADIOGRAPHIC EXAMINATION

Radiographic Laboratory 1
 Technikon Natal
 Ritson Rd,
 Durban.

DATE: _____

NAME (block letters) _____ ID number _____ x-ray number _____

Age: _____ Race: _____ Sex: _____ Date LMP _____

TRANSPORT TO RADIOGRAPHIC LABORATORY _____ Previous x-rays.....
 walking.....chair.....stretcher.....

Patient's history and clinical findings: _____

Radiographic examination required: _____

Information required: _____

Referring Doctor (name & signature) _____

FOR OFFICIAL USE OF RADIOGRAPHIC LABORATORY ONLY

Radiographic Factors

Part	kV	mA	time	bucky	room

Time of arrival:.....

Time of x-ray:.....

Time of departure:.....

FILMS

No.	size	code	Passed by