THE LUMBOSACRAL PLEXUS AND DISEASES AFFECTING IT.

SUBMITTED IN FULFILMENT OF THE
DEGREE OF MASTER OF MEDICINE (NEUROLOGY)
AT THE UNIVERSITY OF CAPE TOWN

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CONTENTS.

DECLARATION.

I, AHMED IQBAL BHIGJEE, hereby declare that the work on which this dissertation is based is original (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other University.

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SUMMARY.

This study comprised a review of lumbo-sacral plexopathies seen at Groote Schuur Hospital from January 1979 to September 1984. The aims were to :

1) determine the frequency of the disorder.
2) examine the aetiological spectrum.
3) emphasise features in the bedside assessment that would lead to accurate localisation of the neurological deficit, and
4) highlight the value of relatively non-invasive investigations.

Material was obtained from :

1) computer records - January 1980 to August 1984.
2) cases seen in the EMG laboratory - January 1979 to September 1984, and
3) patients seen personally - January 1984 to September 1984.

There was a total of 29 cases. Sixteen had diabetic amyotrophy, three were related to trauma, and three were due to neoplastic infiltration. The rest constituted of single cases of systemic lupus erythematosus, radiation damage, retroperitoneal haemorrhage, infection related disease and idiopathic plexopathy.

From the cases studied the following clinical picture, which suggested plexus localisation, emerged :

1) unilateral symptoms and signs (except in cases of diabetic amyotrophy).
2) absence of spinal pain and negative straight leg raising test.
3) absence of sphincter disturbance.
4) weakness in muscle groups which could not all be explained on the basis of a single nerve or single root lesion, and
5) sensory loss, when present, was patchy, insignificant and did not correspond to a recognised dermatome.

The most useful special investigations were electromyography and CT scanning of the abdomen and pelvis. In the case of the former, denervation in several muscle groups and the demonstration of normal para-spinal muscles indicated a plexus lesion. CT scanning, which has made the rather inaccessibly located plexus more open to evaluation, was useful in not only confirming the site of the lesion in some cases, but also demonstrating the nature and extent of the pathology.

With careful clinical assessment and the use of the above investigations, invasive procedures, such as myelography, and inappropriate management might be avoided.

INTRODUCTION.

The lumbo-sacral plexus[1,2] is formed by the ventral rami of the lumbar roots 2, 3 and 4 (the lumbar portion) and of lumbar roots 4, 5 and sacral roots 1, 2, 3 and sometimes 4 (sacral portion) (Figure 1).[3] The lumbar plexus is situated in the psoas muscle in front of the lumbar vertebrae. The sacral portion lies in front of the piriformis muscle and behind internal iliac vessels, ureters and the terminal ileum.

The important branches of the lumbar plexus are the muscular twigs to the psoas major (L2, L3), the femoral nerve (L2, L3, L4) which supplies the quadriceps femoris and the obturator nerve (L2, L3, L4) which supplies the adductors of the thigh. The main branches of the sacral plexus are the sciatic, superior and inferior gluteal nerves and the posterior cutaneous nerve of the thigh. The sciatic nerve supplies the hamstrings (L4, L5, S1, S2, S3) and via its common peroneal and tibial branches supplies all the muscles below the knee.

As the sympathetic outflow from the cord stops at the first lumbar segment, the ventral roots below this level do not contain any sympathetic fibres.[4] The L2 to S5 segmental nerves (and hence the plexus) receive their sympathetic supply from the sympathetic chain via the grey rami communicantes which join the nerves distal to the roots.

This study, which refers to the Groote Schuur experience, was undertaken to :

1) examine the various aetiologies of lumbo-sacral plexopathy.
2) emphasise features in the bedside assessment that would point to the accurate localisation of the pathology, and
3) highlight the value of relatively non-invasive investigations in helping to make a correct diagnosis.

MATERIAL AND METHODS.

Cases of lumbo-sacral plexopathies were drawn from the computer records, EMG laboratory records and patients personally examined. A computer search for the diagnostic labels "lumbo-sacral plexus lesions" and "diabetic amyotrophy" was made for the period January 1980 to August 1984. The EMG records from January 1979 to August 1984 were examined. The personally examined patients were cases seen in the first eight months of 1984.

RESULTS.

There were sixteen cases of diabetic amyotrophy, three cases of trauma, three of neoplastic infiltration, two related to aneurysms and aneurysm surgery, and one each of haemophilia, radiation damage, systemic lupus erythematosus, plexopathy associated with pulmonary tuberculosis and one case for which no cause was found.

These cases are discussed in detail under the various aetiologies.

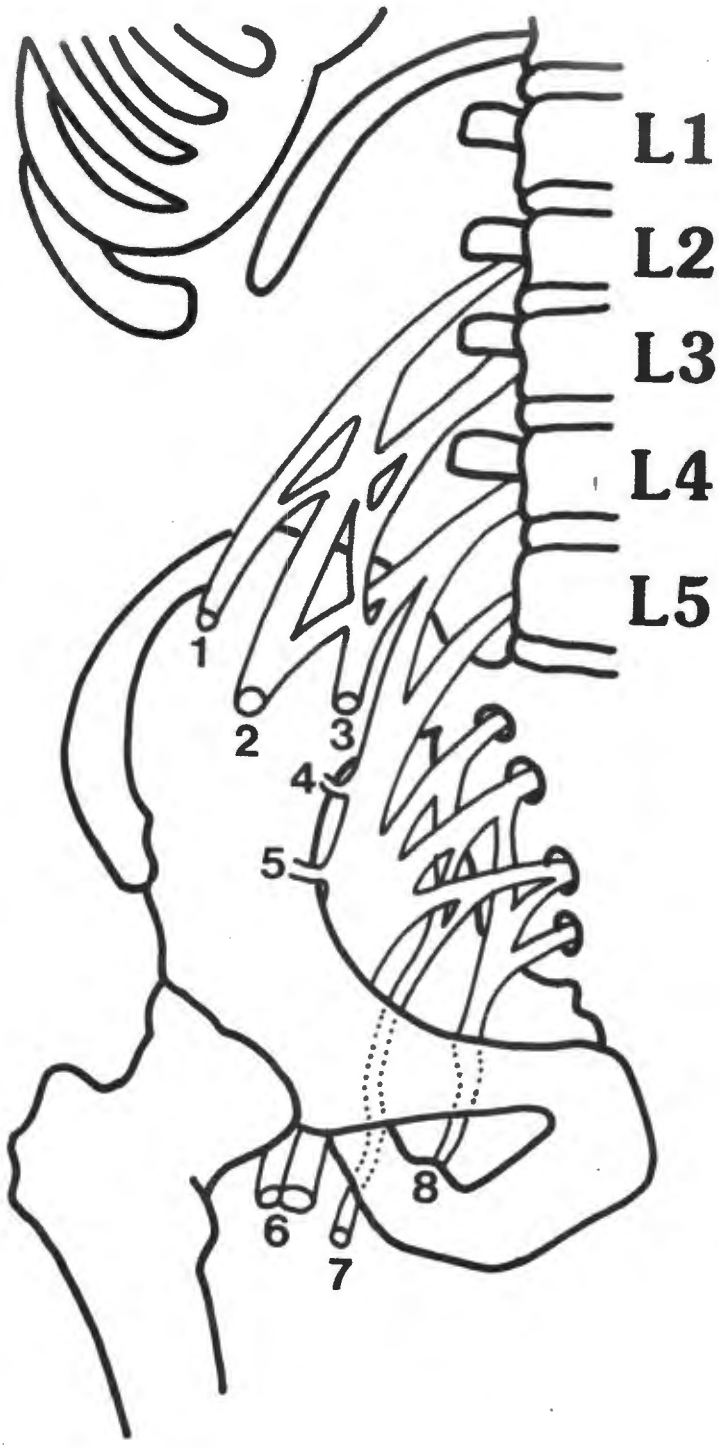

FIGURE 1 : Diagrammatic representation of lumbosacral plexus :

1) lateral cutaneous nerve of the thigh.
2) femoral nerve.
3) obturator nerve.
4) superior gluteal.
5) inferior gluteal
6) sciatic nerve.
7) posterior cutaneous nerve of thigh.
8) pudendal nerve.

(modified from Thomas and Dale[3]).

DIABETES MELLITUS
CASE HISTORIES.

Details of the cases of diabetic amyotrophy are summarised in Tables I and II. Patient number three, who was examined personally, is described fully later.

The ages of the sixteen patients ranged from 24 to 82 years, with a mean of 64,8 years. Only one patient was below 45 years of age. There were 11 whites (7 males and 4 females) and 5 coloureds (3 males and 2 females). The duration of manifest diabetes, which was recorded in eleven cases, varied from 1 to 24 years with a mean of 9,5 years. Random plasma glucose ranged from 5,3 to 18,5 with a mean of 11,9 m mol/ℓ. Eight patients were on oral anti-diabetic agents, 5 on insulin, 1 on both insulin and oral agents, 1 on no treatment and in one the therapy was not recorded. The duration of leg symptoms before the diagnosis of amyotrophy was made varied from six weeks to one year with a mean of 5,8 months. The duration of symptoms was not known in four patients. Pain was a prominent feature in nine patients, absent in three and in two there was no record of whether pain was absent or present. Muscle weakness and/or wasting was bilateral in 10 (asymmetrical in four) and unilateral in six.

Sensory disturbance was absent in six and not commented on in three patients. Of the rest, in only two cases could the sensory loss be attributed to a plexopathy. The others had symmetrical peripheral sensory disturbance in a stocking distribution. One or more tendon jerk was absent in every patient. The knee jerk was absent on one or both sides in 11 patients, depressed in two, normal in one and not commented on in one. The ankle jerks were bilaterally present and normal in three patients. Only six patients were followed-up. Four improved, one who was seen after three months remained unchanged and one died in renal failure. Only one patient (case 3) had an additional neurological deficit; she had a right lateral rectus palsy. Diabetic retinopathy was noted in three of the 13 patients in whom findings on fundoscopy were noted.

Patient number 3 (EA - 53564555), a 57 year old Coloured woman, was seen on 17 February 1984.

She was found to be diabetic in 1970 and treated initially with oral agents, but required insulin since 1977. She gave a six month history of pain, tenderness and weakness in both legs, left worse than right. The pain was over the anterior aspect of the thighs and medial sides of both shins. There was no backache nor were there any symptoms of bladder or bowel dysfunction. She also gave a three week history of diplopia on looking to the right. Neurological examination revealed a right sixth nerve palsy, a small right foot (legacy of childhood poliomyelitis), decreased power in the pelvic girdle muscles (right worse than left) and weakness of knee flexors and extensors, especially the left quadriceps femoris (1/5 power). There was a grade 4/5 weakness of left ankle and foot muscles. Movement of the right was also reduced, but most of the weakness here was thought by the patient to be old. All the tendon jerks in the legs were absent. Pin-prick, touch, and proprioception were intact. Vibration sense was impaired up to the knees bilaterally. She had hyper-aesthesia of the soles of her feet. A random plasma glucose was 15,9 m mol/ℓ. Myelography and CSF examination were normal. EMG of both quadriceps, tibialis anterior muscles and gastrocnemii showed no definite denervation, but markedly reduced recruitment. EMG of the para-spinal muscles was not done.

DISCUSSION.

Diabetes affecting the plexus was first described by Bruns[5] and "rediscovered" by Garland [6 7] who initially called it "diabetic myelopathy" and later "diabetic amyotrophy". The disease is bilateral in two thirds of the patients.[8] It may extend beyond the area of innervation by the plexus as indicated by the findings of denervation of the para-spinal muscle[8 9] or may be confined to a single nerve, eg. a femoral neuropathy.[10] Hence it is not suprising that pathological studies (which are sparse) have given conflicting results. Raff *et al* [11] showed lesions confined to the plexus and proximal nerve trunks and no lesion in the roots. Others have found changes only in the roots[12 13] or

no lesion in the spinal cord or nerves but neurogenic changes in the muscle.[14]

The patients studied by Raff *et al*[11] had numerous infarcts suggesting an ischaemic basis, whilst Skanse and Gydell[15] noted patchy demyelination in the sciatic and femoral nerves. Williams and Shayer[16] concluded from their study of three patients that diabetic amyotrophy was distinct from a mono-neuropathy multiplex and postulated that it was part of a generalised neuro-pathology and that "disordered neuron-axonal metabolism" was the basis of the pathological and clinical changes. In all likelihood the pathogenesis may be multifactorial with varying emphasis in different patients.

With respect to the clinical manifestations our patients had features similar to that described in the literature[17 18], viz :

1) common in males.
2) tended to occur in middle aged and elderly patients.
3) diabetes mellitus of several years duration although in some cases it might be the presenting manifestation of the disease.
4) diabetic state under satisfactory control.
5) not related to the type of antidiabetic therapy.
6) pain was prominent yet sensory loss was inconspicuous.
7) unilateral in one third of cases and, where bilateral, was often asymmetrical.
8) symmetrical peripheral neuropathy was often present.
9) retinopathy and nephropathy were uncommon.
10) patients showed improvement with time.

Since two thirds of the patients, like the one described earlier, will have bilateral dysfunction, the physician might be misled and undertake myelography in search of an intraspinal lesion. However, recognition of the clinical picture, taken in conjunction with :

1) the absence of significant backache and bowel and bladder dysfunction, and
2) EMG findings.

should obviate the need for myelography in most cases.

TABLE I.

No	Name	Age (yrs)	Race	Sex	Duration of diabetes	Random blood sugar	Treatment
1	TK	48	W	M	1 yr	7,5	Insulin
2	AD	82	W	M	10 yr	5,8	Oral
3	EA	57	C	F	14 yr	15,9	Insulin
4	GDuP	46	W	M	14 m	12,5	Oral
5	WM	68	C	M	8 yr	11,5	Oral
6	HS	59	C	F	1 yr	6,2	Insulin
7	PM	24	C	M	3 m	18,5	No treatment
8	CA	53	C	M	11 yr	5,1	Insulin
9	ON	62	W	M	24 yr	15,8	Insulin and oral
10	CM	72	W	M	NK	10,0	Oral
11	HR	70	W	M	2 yr	5,9	Oral
12	HM	58	W	F	12 yr	12,4	Oral
13	CR	73	W	F	NK	15,0	Oral
14	AF	78	W	F	14 yr	10,3	Insulin
15	LD	62	W	M	NK	7,9	NK
16	HR	73	W	M	1 yr	5,9	Oral

NK = not known

TABLE II.

No	Duration of symptoms	Pain	Sensory disturbance	Retinopathy	Outcome
1	NK	NK	distal shocking loss	-ve	died - CRF
2	6 w	yes	distal shocking loss	+ve	recovered
3	6 m	yes	VS impaired to knees	+ve	unchanged at 3m
4	5 m	NK	NK	-ve	NK
5	NK	no	distal	-ve	NK
6	NK	no	NK	-ve	NK
7	3 m	paraesthesia	nil	-ve	NK
8	7 m	no	distal	-ve	improved
9	1 yr	yes	nil	+ve	improved
10	3 m	yes	impaired touch dorsum of foot	-ve	NK
11	7 m	yes	anterior aspect rt thigh	-ve	NK
12	3 m	yes	nil	NK	NK
13	7 m	yes	nil	-ve	NK
14	9 m	yes	medial aspect rt knee and lateral lt calf	-ve	improved
15	sudden onset	yes	lt medial shin	NK	NK
16	NK	yes	nil	-ve	improved

NK = not known

NEOPLASTIC DISEASE OF THE PLEXUS
CASE HISTORIES.

Case JG 56858954 (case examined personally).

This seventy-five year old Coloured man presented with a three week history of progressive pain in the region of the right thigh and ankle and weakness in the right leg. At no stage did he have backache, pain in a clear root distribution, pain aggravated by coughing or sneezing, bladder or bowel dysfunction. He also gave a six month history of intermittent haemoptysis. The referring diagnosis was a L5 root lesion and the question of myelography was raised.

The abnormal neurological findings were confined to the right leg. There was wasting of the muscles below the knee and the right buttock. Hip flexion, abduction and extension, and knee flexion were mildly weak (4/5). Ankle dorsiflexion, plantar flexion and big toe extension showed moderate weakness. All his reflexes were present and normal. Pinprick and touch were impaired on the dorsum of the right foot.

Radiographs of the lumbo-sacral spine revealed degenerative changes only. There was increased uptake at the L5, S1 apophyseal joint on the bone scan. This was interpreted as indicating degenerative disease rather than tumour. EMG showed moderately reduced recruitment but no denervation in the right tibialis anterior, peronei and gastrocnemius muscles.

EMG of the para-spinal muscles was normal. A CSF examination was normal. A CT scan of the lower abdomen and pelvis was requested but unfortunately never done. A mass was noted in the right hilum on chest radiography. Bronchial brushings showed atypical cells consistent with squamous carcinoma.

The patient received 3000 rads to his lumbo-sacral spine and para-vertebral area. When reassessed one month later, his pain had subsided and his weakness considerably improved.

Case CT 61244273.

This 68 year old woman was referred to the Department of Neurology in November 1982 as a problem of right sided sciatica for investigation. She presented with a one year history of burning pain on the outer aspect of the right calf, right thigh and right loin and a more recent history of weakness in that leg. There were no bladder or bowel symptoms, and no backache. She had a right nephrectomy for a hypernephroma in 1976.

On examination there was wasting and moderate weakness of the right quadriceps, mild weakness of the tibialis anterior and hamstrings (4/5). The knee jerk was absent. There was no sensory deficit. EMG of the right quadriceps, tibialis anterior and hamstrings showed reduced recruitment but no denervation. There was partial collapse of L4 vertebra on radiography. CT scan of the abdomen revealed a 4,5 cm right para-spinal mass and bilateral adrenal deposits.

The patient underwent radiotherapy to her spine and para-vertebral tissue but died soon after from pulmonary embolism. Necropsy was not performed.

Case SW 63248686.

In 1979, this patient was found to have carcinoma of the prostrate. He had surgery followed by oestrogen therapy. He presented in May 1983 with a three week history of throbbing pain in the right buttock radiating down the back of the leg. There was some question as to whether he had difficulty in initiating micturition.

On examination his straight leg raising and femoral stretch tests were negative. He had mild weakness of the right quadriceps and right tibialis anterior. The right knee jerk was absent but the adductor and ankle jerks were normal. Sensation and anal tone were normal.

Radiographs of his lumbar spine showed areas of sclerosis but the bone scan was normal. Denervation was noted on EMG in the tibialis anterior muscle but the quadriceps and gastrocnemius were normal. CSF examination was normal. CT scan of the pelvis showed right internal iliac lymphadenopathy (Figure 2).

He was referred to radiotherapy for further management.

DISCUSSION.

These three patients demonstrated several important features. All presented with pain, showed variable weakness and insignificant sensory deficit. None had backache or other features of root disease. Yet patient one was thought to have a L5 disc lesion and myelography was contemplated. Besides the history and examination, the absence of paraspinal denervation suggested that the lesion was distal to the roots. CT scanning in case 2 and 3 demonstrated soft tissue masses in the appropriate areas, thus confirming the plexus localisation of the patients neurological disease.

The literature regarding the nature and frequency of malignant infiltration of the plexus is rather sparse. A "Medlars" search failed to find a single paper from 1966 to November 1984. Possible reasons for this include :

1) misdiagnosis, eg. case 1 of this series and case 6 of the series by Wetzel *et al*[19] were both thought to have disc disease. The latter patient, in fact, underwent laminectomy despite a normal myelogram. He was later found to have a retroperitoneal rhabdomyosarcoma.

2) lumbo-sacral disease as one manifestation of disseminated carcinoma or lymphoma, might be considered to be of insufficient importance to require adequate clinical assessment and investigation. I was able to trace six probable cases of lumbo-sacral plexus infiltration but only three had adequate information for inclusion into this study.

3) inability to clearly demonstrate soft tissue deposits and the difficulty sometimes in differentiating between radiogenic damage and tumour recurrence. This problem has been largely solved with the advent of CT scanning[20 21], which for the first time allows the physician to examine non-invasively soft tissue structures not seen by conventional imaging techniques.

The usual malignancies that infiltrate the plexus are lymphoma, carcinomata of the prostate, rectum, cervix, lung and kidney. Lymphomas tend to involve mainly the lumbar plexus[22] while prostatic, cervical and rectal carcinomata cause disease of the sacral plexus by direct invasion of the lymphatics and peri-neural spaces.

In a study[23] of 42 patients with biopsy proven retroperitoneal lesions, eight of eleven patients with neurological disease had malignant infiltration - three

patients had lymphoma, two carcinoma of the gastro-intestinal tract, one Wilm's tumour, one lung carcinoma and one leiomyosarcoma. Of importance, is that six of these eight patients had either abdominal or rectal masses on careful palpation, thus indicating the site of lesion and guiding the choice of investigation.

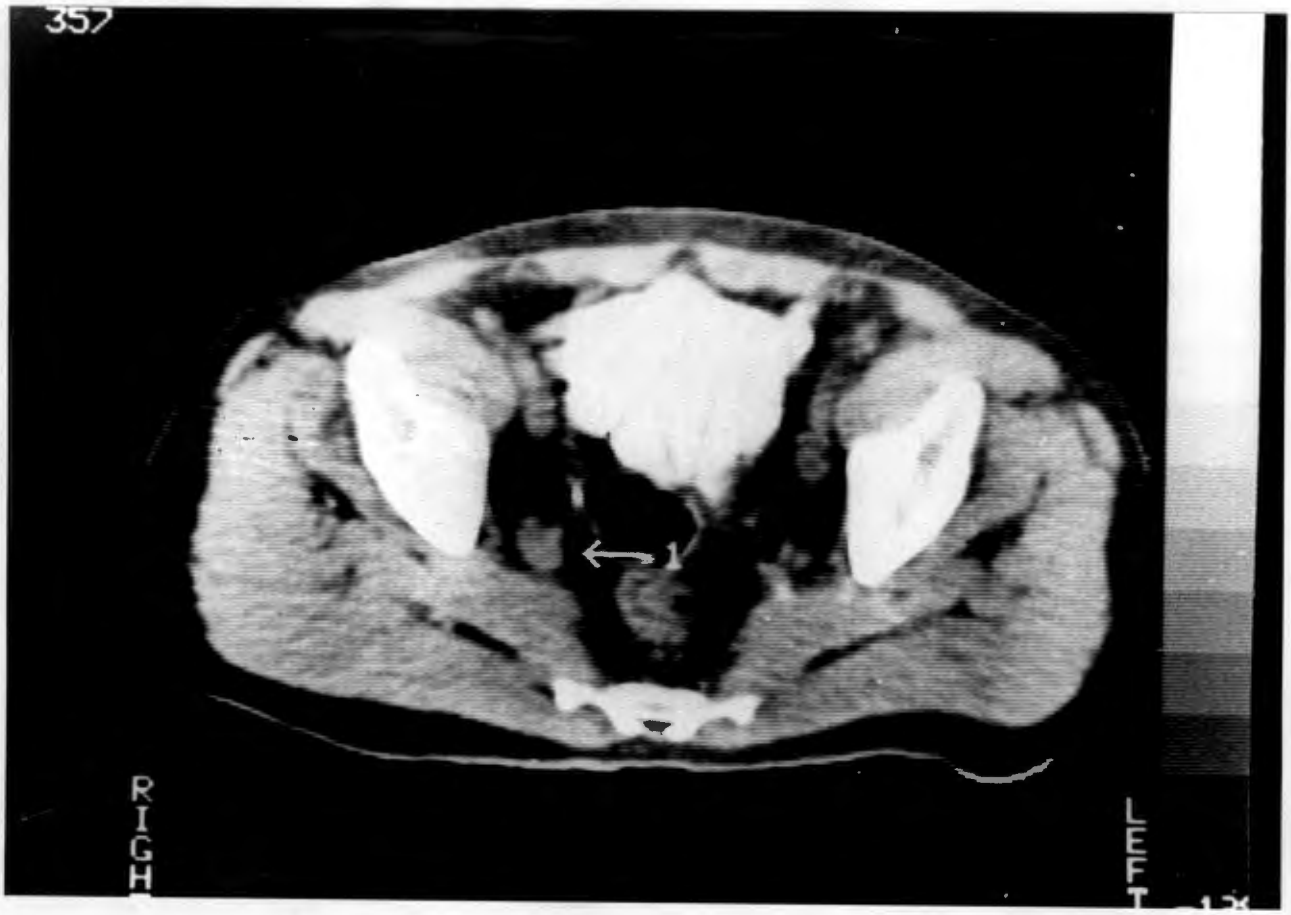

FIGURE 2 : CT scan showing internal iliac lymphadenopathy.

TRAUMA
CASE HISTORIES.

Case NCF (seen in EMG Laborary, 24 March 1983)

This patient was involved in a motor cycle accident in 1977, sustaining bilateral fracture dislocation of the acetabulae, vertical fractures of the pelvis, supra-condylar fracture of the right femur and fractures of the jaw. When mobilis-ation was started a limp was noted.

On examination abduction and internal rotation at the right hip was absent. There was mild weakness of the gluteus maximus and hamstrings and right foot drop. The left leg was normal and there was no bladder or bowel disturbance.

EMG of the gluteus medius showed profused fibrillation and positive sharp waves. No volitional activity was seen. The gluteus maximus, hamstrings and tibialis anterior all showed reduced recruitment but no spontaneous activity. The conclusion reached was that there was total necrosis of the superior gluteal nerve and partial damage of L4, L5 and S1 contributions of the lumbo-sacral plexus.

Case JLR 57202954.

This 23 year old man was knocked down by a car on 13 January 1979 and sustained fractures of his right scapula and clavicle, left humerus and upper ribs. No fracture of his pelvis was noted. At laparotomy he had a laceration of the liver and a large adjacent retroperitoneal haematoma. It was noticed he had a flail left leg. The right leg was normal and there was no sphincter disturbance. He was seen by a neurosurgeon who suggested myelography. This was normal.

The patient was seen in the EMG Laboratory on 26 July 1979. Apart from minimal hip adduction no other movement was possible in the left leg. The knee and ankle jerks were absent. Pin-prick and touch were impaired in the distal part of the thigh and lower leg.

Stimulation of the left common peroneal and tibial nerves did not produce any response. A normal sural action potential was obtained. EMG of the quadriceps and tibialis anterior showed fibrillations, positive sharp waves and no volitional activity.

Case LS 57267817.

This 23 year old woman shot herself in the abdomen with a .38 revolver in September 1980. The bullet entered the abdomen just above the umbilicus, then passed through the anterior and posterior stomach walls, across the medial aspect of the duodenum, past the left side of the inferior vena cava and lodged in the left para-vertebral gutter below and lateral to the transverse process of the third lumbar vertebra.

On admission she complained of pain and weakness in the right leg. The left leg was normal and there was no sphincter disturbance.

She was seen in the EMG Laboratory about one month later when she was noted to have mild weakness of the right quadriceps and severe weakness of the right tibialis anterior, gastrocnemius, hamstrings and glutei. The right knee and ankle jerks were absent. Sensation was impaired in the L5 to S2 distribution. EMG showed partial denervation of the right tibialis anterior, gastrocnemius, hamstring and glutei.

The patient was reassessed clinically and electrically in May 1982. No significant improvement was noted.

DISCUSSION.

Injury to the plexus is considered to be rare, eg. Patterson and Morton[24] observed only six cases over a period of ten years. However, the experience of Stöehr[25] would suggest that traumatic plexopathy is more frequent than is commonly believed. Ninety-one percent of Stöehr's[25] 53 patients were misdiagnosed as having lesions of the femoral and sciatic nerves. He suggested the following reasons for this state of affairs :

1) testing of the adductors and gluteal muscles were often neglected in routine neurological examination.
2) thorough examination might not be possible in the setting of multiple injuries and pain. In the latter instance careful EMG studies should lead to the correct diagnosis.

Still other cases might be misdiagnosed as root avulsions but this must be distinctly unusual. Goodell[26] explored the cauda equina of two patients in whom myelography demonstrated meningocoeles. In both these cases the roots were

intact. Christie and Jamieson[27] commented that the long course of the roots from their points of emergence from the cord to the spinal exit foramina may protect them from avulsion.

Both Stöehr[25] and Huittinen and Slatis[28] have noted that the mechanism of injury is traction to the plexus and was associated with double vertical fractures causing dislocation of the hemipelvis or with fractures through the acetabulum. This was probably the mechanism of injury in Case 1 (NCF). The disruption of the pelvic ring was not invariable as was found by Finney and Wolfman[29] and as demonstrated by Case 2 (JLR).

In patient 3 (LS) the bullet lodged on the left side. There was no evidence from the findings at laparotomy that the track of the missile included the right lumbo-sacral plexus. Therefore, damage to the plexus probably resulted from shock waves generated by the bullet. All degrees of damage might occur in such instances[30], ranging from transient paralysis to gross tears and loss of continuity.

The prognosis of traumatic damage is uniformly poor. All three cases described have shown little or no improvement.

Post-operative lesions have occurred most commonly with total hip replacement. The mechanism of injury here is also thought to be traction[31]. Direct injury at the site of operation is unlikely as the usually severe weakness of hip flexors suggest a more proximal site of damage. The prognosis in post-operative cases is good[32].

ANEURYSMS AND ANEURYSM SURGERY
CASE HISTORIES.

Case SP 65146326 (patient examined personally)

A 25 year old Coloured woman, hypertensive since 14 years of age, presented with intermittent claudication of her legs for two months. On examination, she was found to have an abdominal aortic aneurysm and absent pulses below the femoral arteries. Neurological examination was normal.

A few hours after an aorto-bifemoral bypass graft she became shocked, was resuscitated and taken back to theatre. A large retro-peritoneal haematoma was evacuated and a thrombectomy of the left profunda femoris artery performed. Three days after operation her left lower leg became swollen. Anterior and posterior fasciotomies were made to prevent muscle necrosis.

The time of onset of her neurological deficit is not certain, but weakness was noted for the first time on the fourth post-operative day. The surgeons attributed weakness in the left foot to a compartment syndrome but could not understand why she had quadriceps weakness. A neurological opinion was sought.

The tone in her left leg was reduced. There was marked weakness of hip flexion and adduction (grade 2/5) and moderate weakness of abduction and extension (4/5). Knee flexion was moderately weak but knee extension was not possible at all (0/5).

There was no movement at the ankle and toes. The left knee and ankle jerks were absent. Sensation was patchily impaired in the left leg. The right leg was normal apart from a depressed ankle jerk and impaired pin-prick in the L1 dermatome. There was no sphincter disturbance.

EMG of the left quadriceps and extensor digitorum brevis showed profuse positive sharp waves and no volitional activity. Spontaneous fibrillations, positive sharp waves and moderately decreased recruitment were noted in the left hamstring muscles. CT scan of the pelvis showed small collections of retro-peritoneal blood and areas of attenuation in both psoas muscles.

Considerable improvement was noted when the patient was reassessed four months later. Hip flexion and adduction, and knee extension were now grade 4/5. Ankle dorsiflexion and big toe extension were grade 2/5. The reflexes were still absent. No sensory deficit could be elicited. Repeat EMG of the left quadriceps still showed spontaneous activity but moderate recruitment was now present.

Case JN 65274797.

This 29 year old Coloured man, who was examined personally, was referred with a diagnosis of spinal injury following a stab wound.

About two weeks prior to admission he was stabbed in the lower back with a knife with a blade length of approximately 10 cm. Following the stab he was able to walk home and was well for about 24 hours. Thereafter he noticed pain in his right para-vertebral area radiating down to the lateral aspect of his thigh and weakness in the right leg. There was no sphincter disturbance nor pain or weakness in the left leg.

On examination a 1 cm scar was present about 3 cm to the right of the midline at the level of the third lumbar vertebra. The right hip was flexed to 45° indicating psoas spasm. The right gluteus maximus and quadriceps were wasted. There was mild to moderate weakness of hip flexion, extension and abduction. The quadriceps femoris showed no contraction at all. Knee flexion was mildly weak and distal power was normal. The right knee jerk was absent but the adductor and ankle jerks were normal. Pin-prick and touch were impaired on the anterior aspect of the thigh and the medial part of the lower leg. The left leg was normal. Ultrasound demonstrated a 25 cm pulsating mass on the anterior aspect of the psoas muscle (Figure 3). Angiography revealed a false aneurysm of a lumbar artery (Figure 4).

The feeding vessel was embolised and within 48 hours the pain decreased, there was improvement in hip movements and minimal function had returned to the quadriceps muscle.

DISCUSSION.

Aneurysms might give rise to neurological symptoms either by direct compression or compression following rupture with development of false

aneurysms. Razzuk *et al*[33] described two patients in whom femoral neuropathies developed following rupture of abdominal aortic aneurysms, whilst Chapman *et al*[34] reported sacral plexus compression by aneurysms of the iliac arteries. In other cases, plexus damage might follow spontaneous thrombosis of nutrient vessels.[35]

Most cases of aneurysms are related to atherosclerosis[35] but in some patients symptoms might be due to mycotic aneurysms. Aneurysms might also develop in the post-partum period as a result of trauma from forceps delivery or caesarean section.[36]

Aneurysm surgery might cause neurological dysfunction, either by resection or operative thrombosis of nutrient vessels, or by direct traction of the plexus during surgery. Usubiager *et al*[37] described a 75 year old man who underwent an aorto-bifemoral bypass graft. Post-operatively he had complete paralysis of all the muscles below the knee and weakness of the psoas, hamstrings and quadriceps muscles in his left leg. Necropsy revealed infarction of the psoas muscle and the lumbo-sacral plexus. A similar mechanism of ischaemic damage might be the most satisfactory explanation of Case SP's neurological damage as :

1) a retro-peritoneal haematoma would cause a transient dysfunction and little evidence of denervation on EMG, and
2) traction during surgery, especially if hand held, should primarily cause a conduction block. Further, the attenuation noted in both psoas muscles on CT scan might have indicated ischaemic damage.

In the second patient (JN), the site of the stab wound, the unilateral nature of his symptoms and the absence of sphincter disturbance should have alerted the referring physician to an extra-spinal site for his neurological deficit. The fact that he was able to walk home excluded significant nerve damage at the time of the assault, but suggested either the development of sepsis or subsequent haematoma formation. Ultrasonography proved extremely valuable as a non-invasive investigation since it not only demonstrated a mass, but also indicated its nature.

While aneurysms of the abdominal aorta and its branches following vascular surgery and trauma have been reported to be not uncommon,[38 39] lumbar

artery aneurysms must be exceptionally rare. In the extensive Vietnam experience[39] no cases of isolated lumbar artery aneurysm was found. A mannual search of the literature from 1960 to 1983 revealed just one case of lumbar artery aneurysm in a renal transplant patient[40] - the pathogenesis was unclear and there was no comment on his neurological status.

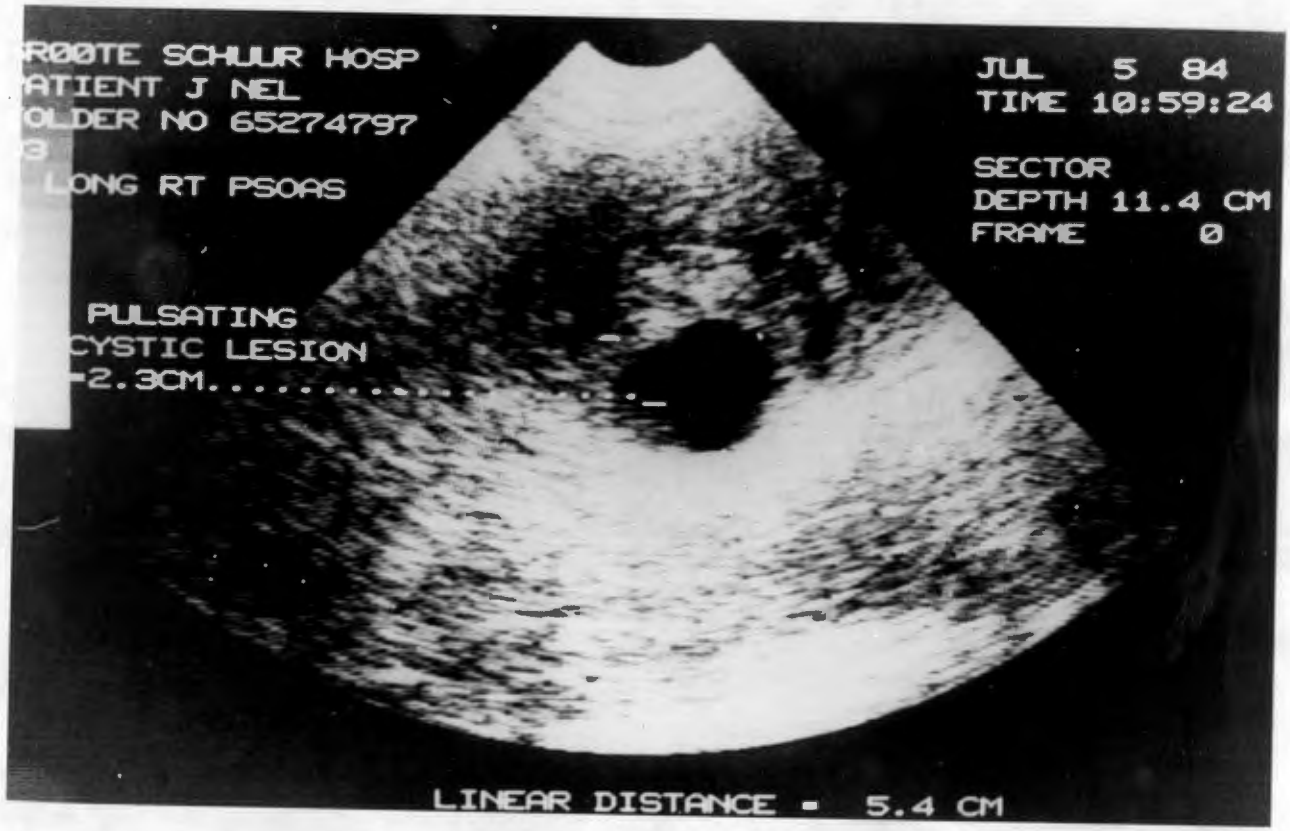

FIGURE 3 : Ultrasound study demonstrating the false aneurysm.

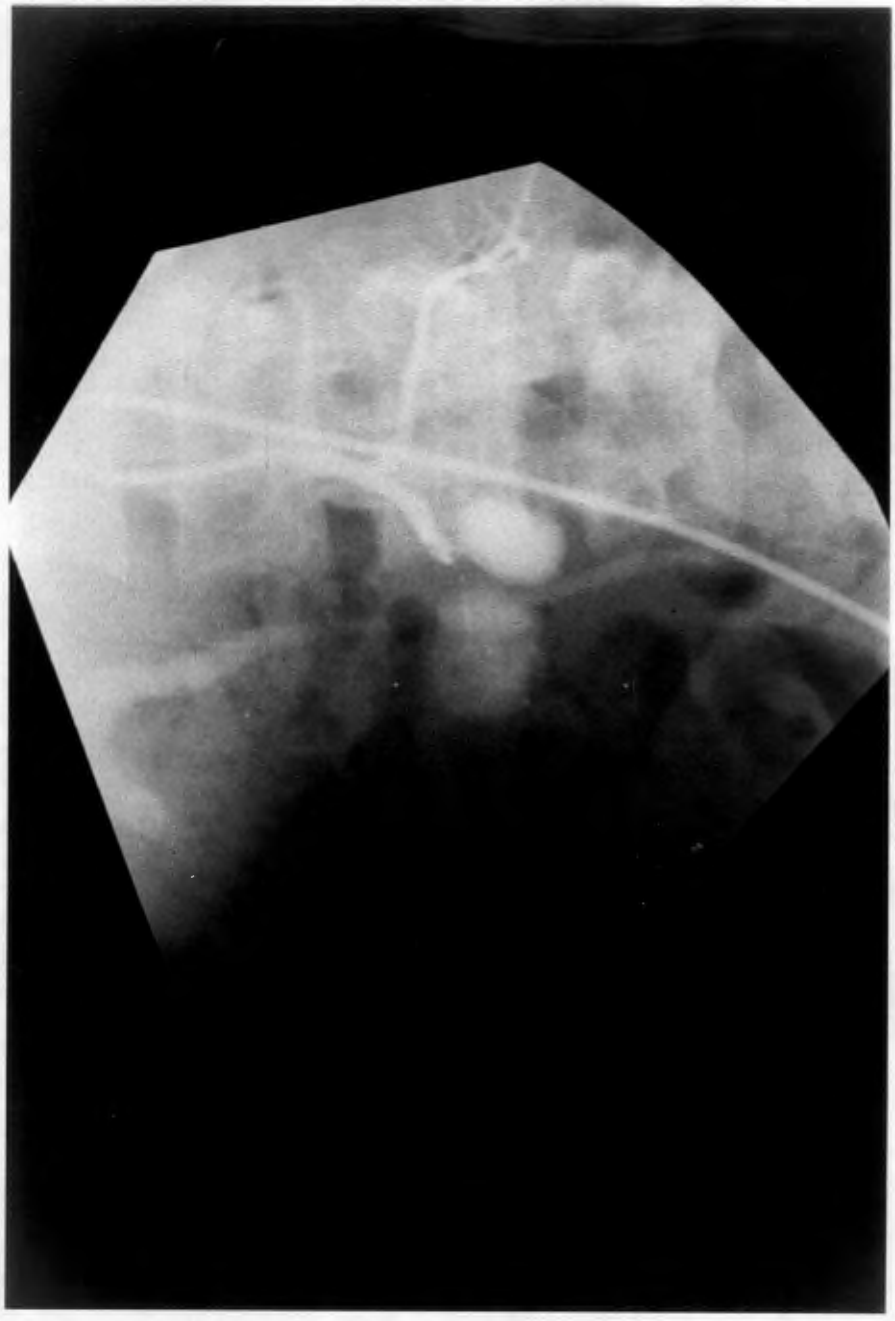

FIGURE 4 : Selective angiography demonstrating the false aneurysm.

VASCULITIDES
CASE HISTORY.

Case SB 65290777.

This 50 year old Coloured woman presented to Groote Schuur Hospital on 06 July 1984. The only history obtained was that she had been sick for two weeks. She appeared withdrawn, lay unconcerned in a pool of urine and nodded inappropriately to most questions and requests. The tendon reflexes were reported as being normal. A diagnosis of depression was made and she was transferred to the psychiatric unit. She became less responsive and when she became pyrexial was referred back to the medical emergency unit on 24 July 1984. She now complained of pain in her left leg. On examination hip movement was noted to be limited by pain. The reflexes in her leg were said to be absent, but formal power testing was not done. A radiograph of the left hip showed an increase in joint space and an isotope study demonstrated increased uptake in that area. A diagnosis of septic arthritis was made and the joint was explored. No pus was found but the biopsy of the synovium showed a vasculitis. Ten days later she developed central chest pain. Ausculation of the heart, which was previously normal, revealed the murmur of aortic regurgitation. Acute ischaemic changes were noted on ECG.

Subsequent to this she was noted to have weakness of her left quadriceps and by the next day movement at ankle was impaired. Neurological assessment undertaken personally on 20 August 1984 showed a normal mental state, normal cranial nerves, upper limbs and right leg. The left quadriceps and tibialis anterior muscles were wasted. Power grading was as follows: hip flexion 3, extension 2, abduction and adduction 3; knee extension 0, knee flexion 2, ankle dorsiflexion and plantar flexion 4, inversion and eversion 4, and big toe extension 3. The left knee jerk was absent but the ankle jerk was present. Pin-prick and touch was impaired on the anterior and lateral aspect of the thigh. CT scan of the pelvis and CSF examination were normal. EMG was done sometime later when the patient was already on prednisone. It showed moderately reduced recruitment in the left quadriceps tibialis anterior, gastrocnemius and hamstrings. There was no evidence of spontaneous activity. The ESR was raised (70-130 mm/hr) and the antinuclear factor was positive to a dilution of 1:2500. Prednisone was administered soon after the onset of the left leg weakness. When the patient was re-assessed three weeks later, power had improved considerably in all muscle groups and the knee jerk had returned.

DISCUSSION.

Vasculitides causing neurological dysfunction include the systemic necrotising angiopathies[41] (polyarteritis nodosa, the allergic granulomatosis of Churg and Strauss and the overlap syndrome), hypersensitivity vasculitis[42] (serum sickness, Henoch Schonlein purpura, cryoglobulinemic vasculitis and systemic lupus erythematosus), Wegeners' granulomatosis,[43] temporal arteritis,[44] Takayasu's disease[45] and Behcets disease.[46] Of the diseases mentioned, plexopathies have been recorded only with systemic necrotising vasculitis,[47] hypersensitivity vasculitis[48] and Wegeners' granulomatosis.[49] Bradley *et al*[50] describe a new syndrome of painful lumbo-sacral plexopathy with a raised ESR. In a five year period they collected six cases. Sural nerve biopsy showed axonal degeneration and perivascular cuffing by inflammatory cells, mainly lymphocytes. All their patients were over 60 years of age (3 men and 3 women). The onset of the illness was either acute or subacute and there were no systemic manifestations. The ESR ranged from 56-125 mm/hr. All demonstrated progressive disease and extensive investigations failed to reveal an underlying cause. Five patients were treated with prednisone (two were given cyclophosphamide as well) and in four there was improvement or stabilisation of the disease. The pathogenesis of the illness was unclear but the raised ESR and response to immunosuppressive suggested an immune mediated basis.

Fifty to seventy percent of patients with SLE develop neurological dysfunction.[51,52] This usually manifest as seizures, mental disorders and cranial nerve dysfunction. Peripheral nervous system involvement is less frequent. Dubois *et al*[53] noted peripheral neuropathy of varying severity in 11% of 520 patients. The patients either had a symmetrical sensori-motor neuropathy, a mononeuropathy, eg. radial palsy or a mononeuropathy miltiplex. An extensive search of the literature from 1960 to 1983 failed to reveal a single clear cut case of lumbo-sacral involvement. In this regard it was interesting to note that Dubois in his book[54] says "Quite often in addition to 'stocking' and 'glove' involvement ..... there is often spotty change elsewhere, suggesting the hit and miss involvement ....". It is possible that lumbo-sacral plexopathy in SLE is being overlooked because of inadequate clinical assessment.

The pathogenesis of neurological damage in SLE is not clear. In some cases antibodies directed against nervous tissue appear to be important.[52] In others, widespread deposition of immune complexes[52] are held responsible. Peri-vascular infiltrates have been noted in some pathological studies[55] suggesting an ischaemic basis for the neurological damage. The mechanism of injury

in the patient described above is not known, but the result was probably a neuropraxia as there was no denervation on EMG and the patient had improved rapidly.

RADIATION
CASE HISTORY.

Case EV 61449666.

In 1972, this woman received approximately 4500 rads to her lower abdomen and pelvis for an osteoclastoma of the left wing of the sacrum. In 1982 she presented with progressive weakness of the left leg. There was no pain but she noted mild numbness of the left toes and a hypersensitive area along the lateral border and sole of the foot. On examination she had wasting of her quadriceps and her toes were clawed due to weakness of the intrinsic muscles of the foot. There was marked weakness of hip flexion and moderate weakness of knee flexion and extension, ankle dorsiflexion and plantar flexion and toe flexion. The left knee jerk was depressed and ankle jerk was absent. The area of hyperaesthesia which the patient complained of was confirmed on examination. No sensory loss was found. The right leg was normal and there was no sphincter disturbance.

Radiographs of the pelvis showed a calcified mass involving the left sacro-iliac region. Old films of 1972 were available for comparison and no increase in the size of the mass was noted. A CT scan of the pelvis showed no tumour in the region of the plexus but interestingly showed atrophy of the psoas muscle (Figure 5). EMG showed reduced recruitment in the left quadriceps, tibialis anterior and gastrocnemius but no denervation. A myelogram was normal.

DISCUSSION.

In 1948 Greenfield and Stark[56] reported the development of leg weakness following abdomino-pelvic irradiation in three patients with testicular germ cell tumours. Since then there have been scattered reports of radiogenic damage to the lumbo-sacral plexus[57-59]. Klaga[60] described five cases of plexus damage in a series of 430 patients treated with abdomino-pelvic irradiation (1,3%). Thus radiation appears to be an uncommon cause of lumbo-sacral plexopathy. However, since the latent period can be of several years duration[58 59] one suspects that the incidence of this complication will rise as patients begin to survive longer with better care.

When a patient with malignant disease presents with neurological symptoms the

clinician is faced with the dilemma of differentiating radiogenic damage from tumour recurrence. Thomas and Holdorff[61] suggest four pre-requisites before considering the diagnosis of radiation induced damage :

1) the diagnosis must be made by exclusion. Therefore careful clinical assessment and investigation are necessary.
2) the nerve lesion must be within the radiation field.
3) there must be a latent period, and
4) the tolerance threshold of the nerve structures must have been excluded.

This threshold is difficult to evaluate as it depends on various factors (eg. area of treatment, thickness of radiated field and duration of treatment) but the patients described by Kristensen *et al*[62] developed paralysis after doses of 1597 and 1670 rets. The tolerance limit of 1400 rets given by Maier *et al*[57] corresponds to a focal dose of 4000 rads over four weeks with twenty fractions of Co. [55]

As the field of abdomino-pelvic radiation usually includes the lumbo-sacral cord, roots and plexuses, one is often uncertain of the exact site (ie. plexus) of the neurological damage. Two explanations for the frequently observed pure lower motor neuron signs are proposed :

1) selective injury to the anterior horn cell within the cord, and
2) injury to the lumbo-sacral plexus.

The experimental work of Bradley *et al*[63] suggests that the plexus is the major site of injury.

The pathogenesis of the nerve injury is a combination of reduced mitotic activity of the normal proliferating cells which are responsible for repair,[64] ischaemia due to the vascular damage and perineurial and endoneurial fibrosis causing entrapment.[65]

Symptoms may arise anytime from a few months[66] to years[59] following radiation. Paraesthesiae and numbness are often initial complaints, but pain, unlike malignant disease, is not a feature.[61] Wasting and weakness are prominent. Sensory deficit is insignificant and sphincter disturbance does not occur. The neurological dysfunction is slowly progressive or develops in a stepwise fashion. The condition may stabilise after a few months or years.

EMG usually shows features of denervation. Less common types of discharges may occur. Stöhr [67] found low frequency bizarre discharges, grouped discharges, myokymia and myoclonic discharges. Albers *et al* [68] also noted myokymia and felt that it was a helpful finding in distinguishing radiogenic damage from tumour. CT scans of the abdomen and pelvis help in excluding tumour recurrence.

The patient discussed above was an excellent example of radiogenic damage to the plexus because :

1) the question of malignant infiltration of the soft tissues as a cause of plexopathy did not arise.
2) the neurological damage occurred in the field of radiation.
3) there was a latent period of approximately ten years.
4) the total dose of radiation exceeded 4000 rads.
5) the field was unilateral and away from the midline, and hence the intra-spinal structures were not radiated.
6) she had no pain and no sphincter disturbance, and
7) CT scan not only showed that the original tumour was at a site distant from the plexus, but also the selective atrophy of the psoas muscle.

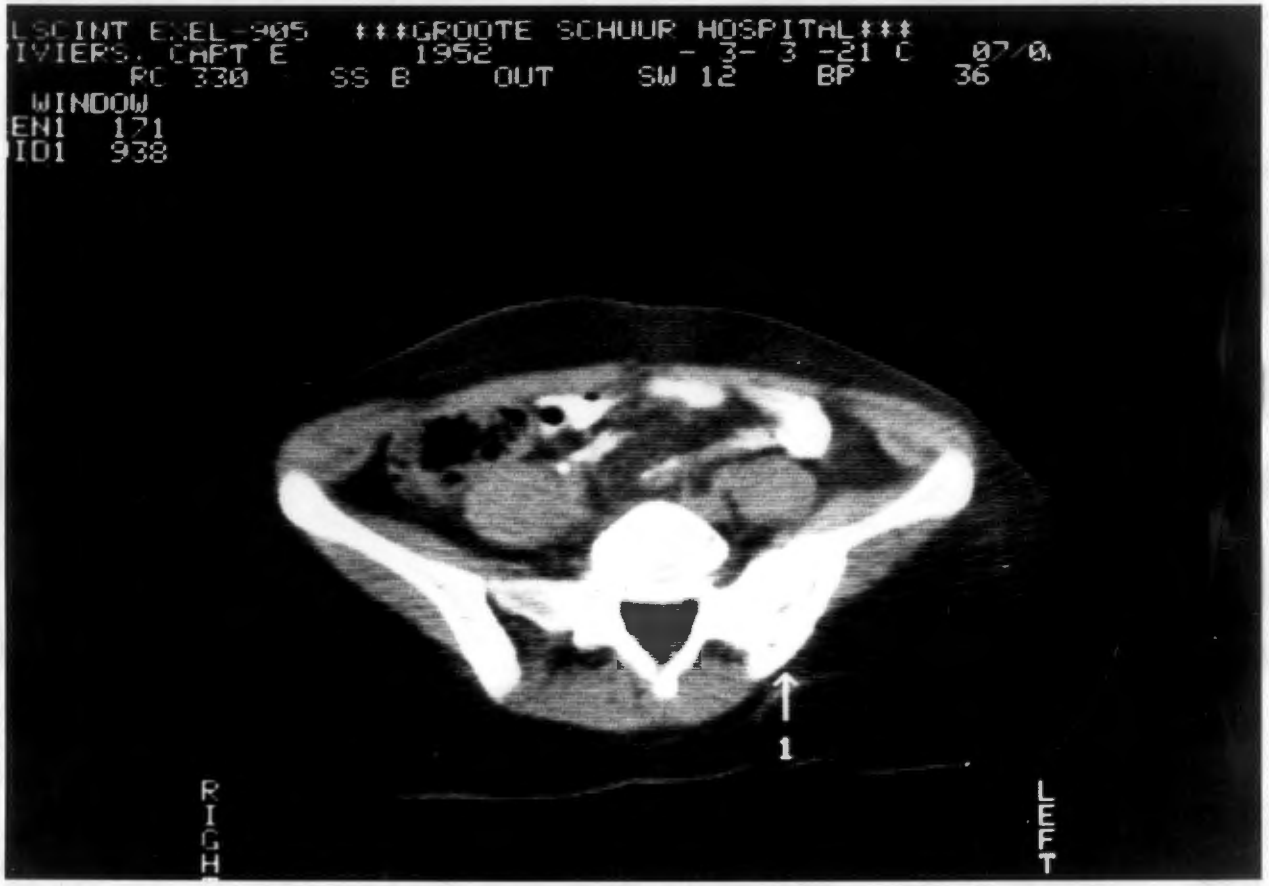

FIGURE 5 : CT scan showing the osteoclastoma of the left wing of the sacrum. Note the atrophy of the psoas muscle.

RETROPERITONEAL HAEMORRHAGE
CASE HISTORY.

Case FT 58352873.

This 52 year old man was a known haemophiliac. He presented on 27 September 1982 with a two day history of increasingly severe pain and swelling in the right groin. He complained of pain and weakness in his right leg. On examination he had a large mass extending from the right upper quadrant to the right iliac fossa. Power was difficult to assess because of pain, but hip flexion and knee extension were limited. The right knee jerk was absent and there was an area of hypoaesthesia over the anterior aspect of the thigh.

The CT scan (Figure 6) showed a large haematoma extending from the origin of iliopsoas to the inguinal ligament. There was another separate pre-sacral haematoma. EMG showed little volitional activity in the right quadriceps, but no features of denervation.

The patient was reassessed three months later. Power had improved considerably and repeat CT scan showed marked decrease in size of haematoma.

DISCUSSION.

After diabetic amyotrophy, retro-peritoneal haemorrhage was the most well documented cause of lumbo-sacral plexopathy. It might occur in haemophiliacs,[69] in patients on anticoagulants,[70] in disseminated intra-vascular coagulation and thrombocytopaenia due to leukaemia.[72]

The space between the fascia and the iliopsoas muscle is unyielding at its lower end. Thus, as the blood accumulates, the space acts as entrapping channel,[70] compressing the nerves. As it runs in the tight groove between the iliacus and psoas muscles, the femoral nerve is the most commonly affected branch of the plexus.

The haematoma may develop spontaneously or after minor trauma. The patient experiences pain in the groin or abdomen, which radiates into the thigh. Numbness and paraesthesia develop in the cutaneous area of supply of the nerves

that are compressed. The hip is held in flexion and weakness of the psoas, quadriceps and adductors may be demonstrated. The knee jerk is usually absent. A palpable mass may develop making the diagnosis fairly simple in the appropriate clinical setting. However, most cases do not develop a mass. Of the 26 cases that Chiu[70] collected from the literature, and from his own experience, only seven had palpable masses. Thus there may be a delay in diagnosis which could prove fatal. Misdiagnosis may lead to wrong therapy, eg. a case of Chiu's series[70] was thought to have lumbar disc herniation and was referred for physiotherapy. In this respect, CT scanning has been very valuable in enabling early diagnosis and prompt therapeutic intervention. [73] [74]

EMG shows decreased or absent volitional activity, but usually no evidence of denervation suggesting a conduction block. This is borne out by the fact that the prognosis for partial or complete recovery is good.[70]

In the patient described, the clinical picture and the results of the special investigations resembled that described in the literature.[69] [73]

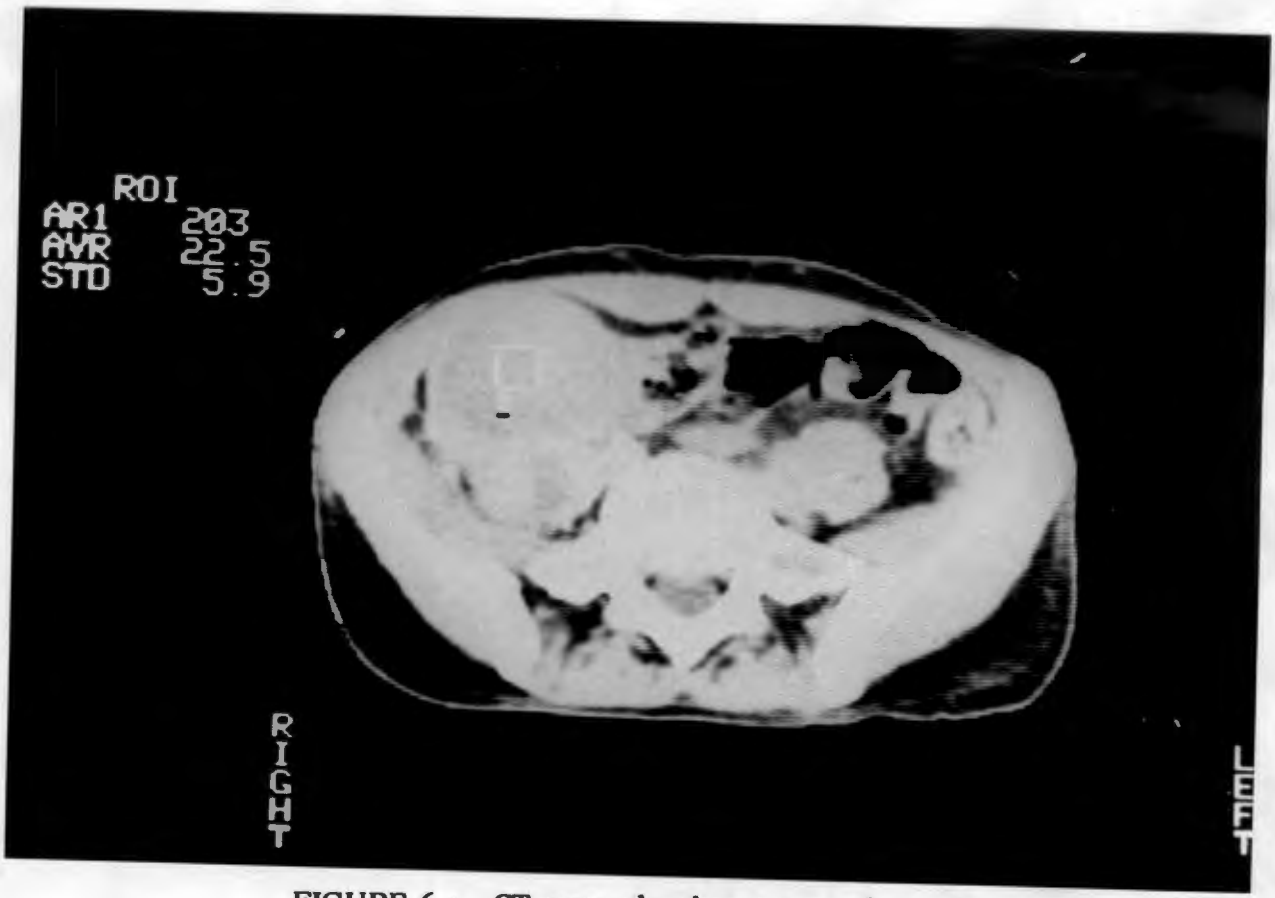

FIGURE 6 : CT scan showing retroperitoneal haemorrhage.

INFECTION-RELATED DISEASE
CASE HISTORIES.

Case AW 65983731 (case examined personally).

In December 1983 this 54 year old Coloured man developed a productive cough, pyrexia and sweating. Pulmonary tuberculosis was diagnosed on the basis of radiological and sputum examinations and he was admitted to the Brooklyn Chest Hospital on 20 January 1984. A few days after admission he became aware of pain and of spontaneous and evoked para-aesthesiae of the lateral aspect of the dorsum of his left foot. Three days later he noticed some weakness of dorsiflexion of the left ankle. This gradually progressed over a few days to a total foot-drop and paralysis of the toes. There was no pain or weakness in the right leg but he noticed some numbness of his toes. He also complained of frequency and urgency of micturition. At no stage did he have backache or pain in a radicular distribution.

On examination, he was sick, pyrexial and pale. The tone, power and sensation in the right leg was normal. In the left leg hip adduction and flexion were normal but extension and abduction were moderately weak. Knee extension was normal but flexion was grade 4. Ankle dorsiflexion was grade 1, plantar flexion grade 3. Toe movements were absent. Both ankle reflexes were absent. Pin-prick and touch was impaired on the dorsum of the left foot and toes. Vibration sense was absent at both ankles but joint position sense was normal.

EMG showed denervation in the left tibialis anterior, gluteus medius and maximus. The left gastrocnemius and hamstrings had reduced recruitment but no spontaneous activity. CT scan of the posterior abdominal wall and pelvis was normal. However, some irregularity of the bladder wall was noted. Myelography and CSF examination were normal. At cystometry the intravesical pressure began to rise when the bladder capacity reached 350 ml. Urine microscopy showed a few red cells and white cells but no organisms (including mycobacteria).

The patient was reassessed four months later. His general condition had improved. The power in his previously weak proximal muscles was now

normal. Ankle dorsiflexion had not improved, but plantar flexion was of near full power. Minimal extension of the big toe was noted. Pin-prick and touch had improved. The ankle jerks were still absent.

DISCUSSION.

Infections might give rise to plexopathies, either by direct invasion or as para-infectious immune phenomenon. Direct involvement might be seen with tuberculosis, lumbar osteomyelitis, pyelonephritis or appendicitis.[75] Stevens[76] commented that cold abscess due to tuberculosis was rare. This does not apply to South Africa, where the disease is common. Patient JN (63483572), who is not discussed in detail as the notes were inadequate, was a good example of a tuberculous psoas abscess. Her CT scan demonstrated the extent of the lesion clearly.

It is being increasingly recognised that a lumbo-sacral plexopathy, similar to the brachial variety, may follow a presumed viral infection of the upper respiratory tract.[77-79] The neurological damage is thought to be immunologically mediated. This problem is discussed in detail in a later section on idiopathic plexopathies.

Marra[80] described an interesting problem of recurrent lumbo-sacral and branchial plexopathy associated with Schistosoma japonicum in a 42 year old man. This patient presented with a three week history of lancinating pain in his buttocks radiating down the posterior aspects of both legs. He had no spinal pain and his straight leg raising test was negative. There was bilateral weakness of the hamstrings, gluteus medius, tibialis anterior and gastrocnemii. EMG showed evidence of denervation in these muscles but none in the paraspinal muscles. Lumbo-sacral myelography and CT scan of the abdomen and pelvis were normal. He was treated with prednisone and praziquantel. The patient presented again six months later with a similar problem in his upper limbs. When seen at a later follow-up, he was considerably improved and could walk independently. As there was no direct parasitic infestation, the author suggested a para-infectious dysimmune basis for the plexopathies.

It is postulated that Case AW developed a plexopathy or a similar para-infectious basis as the CT scan, CSF and myelogram were normal. A literature search from 1960 to 1983 failed to uncover a similar case report for tuberculosis.

IDIOPATHIC PLEXOPATHY
CASE HISTORY

Case AG 59869354.

This nine year old lad presented with a three month history of weakness of the right leg. There was a vague story of preceding pain in the right buttock, but no history of upper respiratory tract infection or recent immunisations.

On examination, he had grade 4 power at the hip, knee and ankle. The right knee jerk was absent and ankle jerk was depressed. Sensation was normal. No abnormality was detected in the left leg and there was no sphincter disturbance.

The following investigations were normal: ESR, x-rays of spine and pelvis, myelogram and CSF examination. The collagen screen was negative. CT scan of the pelvis did not demonstrate any masses but showed wasting of ilio-psoas muscle.

After discharge he has shown gradual improvement.

DISCUSSION.

Lumbo-sacral plexopathy similar to brachial neuritis has been seen occasionally.[81] Although not as common as brachial neuritis (Evans *et al*[77] collected ten cases in 15 years) it is probably underdiagnosed or misdiagnosed, eg. some patients have been subjected to laminectomy for presumed disc disease.[81] Further support for the existence of this entity was provided by :

1) the concurrent involvement of the brachial and lumbo-sacral plexuses, eg. this was seen in an influenza epidemic.[78]
2) preceding respiratory infections;[77] [76] and
3) administration of antisera.[82]

The onset of the illness was acute with pain followed by wasting and weakness. Sensory deficit and paraesthesiæ were not prominent. EMG showed patchy denervation and the CSF was usually normal or showed a mild increase in protein.[77] Recovery was gradual and the functional prognosis was good.[77] [76]

The patient presented in the case history followed a course similar to that described by others.[77] [79] The normal myelogram and CSF excluded any intra-spinal lesion, while the CT scan of the pelvis ruled out any pelvic mass.

MISCELLANEOUS DISORDERS.

Other diseases that might affect the plexus include :

1) familial plexopathy.[82-84]
2) heroin addiction.[85] [86]
3) peripheral plexopathy,[87] [88] and
4) neuropathy following gluteal artery injection.[89] [90]

None of the above type of cases was seen at Groote Schuur Hospital.

TABLE III. AETIOLOGICAL CLASSIFICATION OF LUMBO-SACRAL PLEXOPATHIES.

1) **Diabetes mellitus.** [7 8 9]

2) **Trauma.** [24 25 26 31]

   a) traffic accidents
   b) gunshot wounds
   c) knife wounds
   d) surgery

3) **Neoplasia.** [19 22 23]

   a) primary disease, eg. neurofibromatosis
   b) secondary deposits, eg. prostate, cervix

4) **Vascular disease.**

   a) aneurysm and aneurysm surgery [33 34 37]
   b) vasculitides, eg. [41 42 56]

      - polyarteritis nodosa
      - rheumatoid arthritis
      - systemic lupus erythematosus
      - with high ESR

5) **Radiation.** [55 58 61 91]

   a) primary damage
   b) radiation induced neurofibroma and malignant nerve sheath tumours

6) **Disorders of coagulation.** [69–73]

   a) anticoagulant therapy
   b) haemophilia
   c) disseminated intravascular coagulation
   d) leukaemia

7) **Infection.** [76 80]

   a) tuberculous cold abscess
   b) infections such as osteomyelitis, pyelonephritis and appendicitis
   c) para-infections

      - viral
      - schistosomal
      - ?tuberculous

8) **Idiopathic.** [77 79]

9) **Miscellaneous.**

a) hereditary[82-85]
b) heroin addiction[85]
c) pregnancy related[87 88]
d) intra-gluteal artery injection[89 90]

CONCLUSION.

1) Lumbo-sacral plexopathy was seldom considered in the differential diagnosis of lower limb weakness. Of the six cases that were examined personally, three were referred as root lesions and two as peripheral nerve problems. The sixth patient was correctly diagnosed as a case of diabetic amyotrophy.

The main reason for misdiagnosis was inadequate clinical assessment, especially failure to take note of the history and to test all the important muscle groups. From the cases studied, the following clinical picture emerged which suggested a plexus lesion :

a) unilateral symptoms and signs (except in cases of diabetic amyotrophy).
b) absence of spinal pain and negative straight leg raising.
c) absence of sphincter disturbance.
d) weakness in muscle groups which could not all be explained on the basis of a single root or single peripheral nerve.
e) sensory loss, when present, was patchy, insignificant and did not correspond to a recognised dermatome.

2) The most common cause was diabetic amyotrophy followed by trauma, neopalstic infiltration and aneurysm related disease. There were single cases of retroperitoneal haematoma, SLE, radiation damage, infection related disease and idiopathic plexopathy. A literature search from 1960 to September 1984 failed to reveal clearcut examples of SLE related disease or plexopathy occurring in tuberculosis on a para-infectious basis.

The diseases that might affect the plexus are summarised in Table III.

3) The useful investigations were EMG, CT scanning and ultrasonography. The EMG often demonstrated abnormalities in several muscle groups, making single root or single nerve disease unlikely. In cases of neoplastic disease and other compressive plexopathies the CT scan demonstrated the mass and in some instances the nature and extent of the lesion. Ultrasonography effectively demonstrated a post-traumatic aneurysm in one patient.

4) A high index of suspicion should lead to more cases being diagnosed correctly and thereby enable the patient to escape myelography, and inappropriate management.

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