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VOL. II PART II AUGUST 1966.

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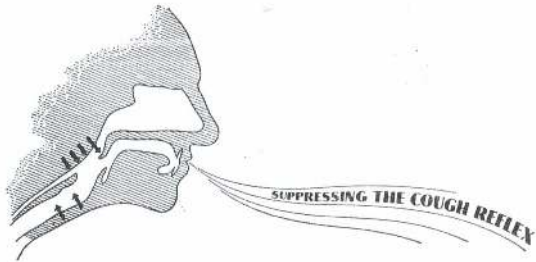


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# The JOURNAL OF THE JAFFNA CLINICAL SOCIETY

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## EDITORIAL NOTICE

Original articles and papers of interest to the Medical Profession are invited.

Scripts submitted for publication in the journal should be sent to *Dr. A. Gabriel F.R.C.S., Hony. Editor, General Hospital, Jaffna.* Papers should be prepared in typescript, double-spaced with 1½" margins, and submitted in final form so as to reduce the necessity for later corrections. The author (s) should retain a carbon copy for reference.

Authors should give their names, professional qualifications and their present post or place in which the work was carried out optionally.

**The title** should be precise and, if possible, brief. If the title given is unsuitably long to head subsequent pages, a short title should be indicated for this purpose.

**Tables and Illustrations** should fit within the page size of the journal 9×7½" and be kept separate from the text. Glossy photographs should be supplied unmounted and drawings should be finished suitably for reproduction. Descriptive legends should be typewritten and annexed to the appropriate figures which should be serially labelled.

**References in the text** should bear the author's surname and the year of publication, both within parentheses, *e.g.* (Smith 1960).

When the author's name is part of a text sentence, the form to be used is exemplified thus: ..... "as Fernando (1945) observed,..... "

If the reference is to joint authorship, all their names should be indicated on first appearance in the text thus:

Fernando, Perera and Pieris, (1962) .....

In subsequent references in the text, the form should be Singham *et al*, (1960).

**References should all be listed** at the end of the text in alphabetical order and not numbered. The Hansard system should be followed here too, with modification as follows:

**Galley Proofs** will be submitted to the first-named author unless otherwise requested for final revision and return within 14 days.

# Editorial....

IT is now eleven years since the scheme of pre-registration internship was introduced.

The scheme requires that every doctor applying for registration with the Ceylon Medical Council, should have done six months of Surgery and six months of Medicine while six months of Midwifery could be counted in lieu of one or the other.

During this period, every doctor should have had a carefully supervised training in these disciplines and at the end of which the Public will expect to have treating them a man or woman of a reasonable degree of competence and a high sense of responsibility.

The Intern, moving into Residence in a hospital after the relative comforts (though not always) of home life, meets the general public for the first time in a new relationship—quite different from the situation of student meeting patient in a ward class or ward round.

He is the first to see an emergency case brought in and the Ward Staff look to him to make the first responsible decision as to what treatment should be given and what further line of action should be followed. The emergency case could be quite unnerving as happened to one of the editors when, hardly had he deposited his luggage and looked around his new hospital when a staff nurse rushed up to him and said "Doctor, a patient has had a reaction to blood transfusion, what shall I do?" The doctor in question was only able to stare at the agitated nurse in stupefaction; whereupon the nurse said "Doctor, we usually give Adrenaline". At this, the doctor said "Ah, yes, of course, Adrenaline. Do give Adrenaline" and retired to the nearest convenience to wipe his fevered brow and regain his composure.

The Intern is responsible for the smooth running of the ward. In the first place, he outlines the first line of treatment which is based on his clinical examination, without the help of any investigations. He orders these later according to what diagnosis he wishes to confirm or what conditions he wants to exclude. He will later be helped by his Physician or Surgeon or Obstetrician but the first moves are his alone and this being so he should set about this in a most careful way. This is something which those who are supervising the work of Interns should emphasise.

The Intern is the administrator of the ward. He is expected to see that the results of laboratory and Radiological investigations are quickly obtained and filed, that all treatment ordered is meticulously carried out, that he maintains a close and harmonious liaison between clinical and para-clinical staff. Failure to do this may result in confusion, dissatisfied patients and even to unpleasant situations.

It is in this first year that the young doctor learns or should learn that his patient is a sick human being and not a "case", and that anxious relatives must be consoled, and sometimes restrained firmly but with much gentleness and kindness and that even frank rudeness must be met with patience and courtesy, irrespective of his own personal feelings.

Dr. V. A. BENJAMIN

*We are the first to admit that all these things may not be easy to carry out, but there is nothing like trying and if the Intern even partially succeeds in this, what a fine specimen of a doctor, the man in the street will find receiving him in hospital when he or his loved ones fall sick, or who he will meet in the Private Dispensary, should our Intern choose the Private sector to earn his living.*

*The period of Internship is the period of training not only in Medicine, Surgery and Obstetrics but what is, we think, far more important, training in the relationship of doctor to patient and doctor to doctor. We would wish that as much attention (if not more) is paid to this aspect of Intern training as is given to the imparting of profound Medical and Surgical dogma.*

# SPINAL CORD COMPRESSION

*By*

**DR. (MRS.) T. THURAIRAJASINGHAM, M.B.B.S. (Cey.), D.M.R.D. (Eng),**

*(Radiologist, General Hospital, Jaffna).*

THERE has been a fairly high incidence of spinal cord compression in Jaffna as observed during the past two years. About one hundred patients have presented with neurological signs and symptoms due to cord compression. The majority of these patients were treatable. In some instances, white patients were wheeled into the clinic or X-Ray room before treatment, have walked out of the ward after treatment. If diagnosed early and treated, spinal cord compression is one of the most rewarding pathological conditions to treat. Despite severe incapacitation of long duration, effective cure or great relief of symptoms is the rule in many cases. Hence it was thought proper to deal with this subject in this paper, with special reference to Intraspinal Tumours.

**CAUSES OF CORD COMPRESSION:**

1. Prolapsed intervertebral disc and cervical spondylosis.
2. Vertebral Injury.
3. Diseases of the vertebral column such as
   (a) Primary or secondary neoplasms.
   (b) Spinal osteitis.
   (c) Paget's disease.
4. Aneurysm of the aorta eroding vertebrae (Rare).
5. Intraspinal tumours.
6. Pachymeningitis.
7. Parasitic cysts

This paper is confined to causes no. (1) and (5) only, as we encountered hardly any examples of the others.

The lesion causing the compression may be (1) Extradural; (2) Intradural Extramedullary; (3) Intramedullary.

**SITE OF COMPRESSION:** This varies with the aetiology and will be discussed with the different types of lesions.

**AGE INCIDENCE:** The most susceptible age is between twenty and sixty years, though occasionally patients outside this age group are seen. In our series we had only two patients under twenty years and none over sixty years.

**SEX INCIDENCE:** The large majority of disc protrusions are seen in males. Both sexes are equally liable to spinal cord tumours.

**SYMPTOMATOLOGY:** The cord is affected in many ways.

(1) Direct compression which interferes with conduction in the cord and spinal roots.

(2) Pressure on veins leading to oedema of the cord below the compression.

(3) Pressure on arteries leading to ischaemia of the cord.

(4) Obstruction to the flow of cerebrospinal fluid.

(7) and (3) lead to degeneration of ganglion cells and white matter which causes softening of the cord (Compression myelitis)

The following structures may be involved:—

1. Posterior nerve roots.
2. Anterior nerve roots and anterior horn cells.
3. Long sensory tracts.

and 4. The Pyramidal tracts.

In considering signs and symptoms, one must take into consideration, (a) root pain (b) sensory loss (c) motor symptoms (d) reflexes (e) sphincter disturbances.

Root pain if present, gives a reliable indication of the level of the lesion; sensory loss seldom corresponds to the level, except in the latest stages, owing to the overlap of adjacent sensory areas; also sensory loss is difficult to elicit.

Any lower motor neurone paralysis is not recognizable early as most muscles receive a double nerve supply. Reflexes are of value in localisation especially if one or more reflexes are diminished and others are exaggerated.

In the early stages, the sphincters are not affected except in lesions of the cauda equina, conus medullaris and sometimes in intramedullary tumours.

**EXTRADURAL LESIONS:** are (I) Disc protrusion (commonest) 80% occur in the lumbar region; 19% in the cervical region; and 1% in the dorsal region. (II) Tumours—meningioma and neurofibroma; extradural cysts, lipomas, lymphogranulomas and angiomatous malformations have been described. About 20% of intraspinal tumours are extradural.

**INTRADURAL EXTRAMEDULLARY LESIONS:** These form about 60% of intraspinal tumours. These are again neurofibroma and meningioma. Occasionally, arachnoid cysts, dermoid cysts, teratomas and lipomas and even gliomas may be seen in these situations. The neurofibroma may take the form of a "dumbell" tumour, which is partly extradural and partly intradural or it may be a part of Von Recklinghausen's neurofibromatosis.

Meningioma may be single or multiple or may form a plaque over several segments of the cord.

Both these types of tumour are small in size, occur in relation to posterior nerve roots on the postero-lateral aspect of the cord (only about a quarter of them are seen on the anterior aspect), easily accessible and can be completely removed except for a few diffuse meningiomas.

About 15 to 20% occur in the cervical region, 60% in the dorsal region and 20% in the lumbar region.

They cause (1) early root pains.

(2) Hardly any sensory loss.

(3) Ascending distant sensory loss, the longest sensory fibres being affected first and the shorter ones later so that whatever the level of the tumour, the sensory loss starts in the legs and spreads up the body.

(4) Late upper motor neurone paralysis.

(5) No lower motor neurone paralysis (except when C8 and T 1 are affected.)

and (6) sphincter disturbances (very late.)

Spinal compression is usually bilateral and symmetrical if the lesion is extradural while intradural lesions compress half the cord mainly and produce unilateral root pains.

**INTRAMEDULLARY TUMOURS:** Are gliomas and ependymomas. Tumours of vascular origin-angioma, haemangioma and lipoma and dermoids are less common. They comprise 20% of intraspinal tumours.

Gliomas are solid or cystic and may extend through several segments of the cord. 50% of these occur in the cervical region. They cause (i) Late root pain. (ii) Wide local sensory loss by involvement of the posterior horn cells. (iii) Dissociated distant sensory loss by the involvement of the spinothalamic tract. (iv) Wide lower motor neurone paralysis (anterior horn cells). (v) Upper motor neurone paralysis and bladder symptoms earlier than extramedullary tumours. Motor symptoms are usually bilateral.

**TUMOURS OF THE CONUS MEDULLARIS:** These occupy the region of the 11th and 12th dorsal and 1st lumbar vertebrae. They press upon the lumbar and sacral nerve roots and tip of the cord and produce (1) Root pain (2) Sensory loss and (3) Lower motor neurone paralysis in the distribution of the lumbar roots which supply the front of the thigh and the legs, pain in the thighs, wasting of the Quadriceps, footdrop and reduction of kneejerks while the ankle jerks are increased and due to pressure on the cords, the plantar response extensor and there is spastic paralysis of the legs. The abdominal reflexes are preserved while the cremastric reflexes are lost or diminished.

**TUMOURS OF THE CAUDA EQUINA:** These are of variable size. Any of these tumours mentioned above in addition to cysts of the filum terminale may occur. Only one or two nerve roots may be involved by a small tumour for a long time or a giant tumour (ependymoma) may involve the cord. The lower roots are compressed earlier than the upper ones, by a tumour at a lower level while a tumour at a higher level compresses all the roots.

(i) Pain in the back extending to perineum and back of the thighs is the earliest symptom. It is exacerbated by jerky movements.

(ii) Saddle shaped anaesthesia and analgesia over the buttocks and back of thigh (the lower the tumour is situated, the smaller the area of anaesthesia). If the L5 and upper sacral roots are caught, there is sensory loss over the foot and posterior and outer aspect of the leg; when the lower sacral segments are involved, the genitals become anaesthetic and the patient may be unaware of the passage of a catheter but the bladder usually retains some sensitivity.

(iii) **Lower motor neurone paralysis** in the distribution of the sacral roots—wasting and weakness of the hamstrings, glutei and calf muscles. The tibialis anterior may escape.

(iv) **Loss of the ankle jerk and plantar reflex** but the knee-jerks are normal or even increased due to weakness of the opposing hamstrings. Anal and bulbo cavernous reflexes are preserved till the third and fourth sacral segments are involved.

(v) **Bladder and bowel function disturbances** occur early; precipitancy of micturition, retention of faeces due to unopposed contraction of the internal sphincters although the external sphincters are paralysed, and finally incontinence and complete loss of control over the bladder and rectum and impotence in males.

(vi) **Trophic symptoms** in the lower limbs which may be cold and cyanosed. There may be dependent oedema analgesia to injuries leading to sores which heal slowly and leave permanent scars.

## INVESTIGATIONS

**Examination of the Cerebro-spinal fluid:**

I. **Pressure:** The pressure of the CSF is usually subnormal below the level of the obstruction and pulse and respiration have no effect on it, although coughing and sneezing still raise the pressure. Quickenstedt's test gives an idea of whether or not there is a block and if there is, whether it is partial or complete. The snag is that a normal Quickenstedt's Test does not exclude a spinal block as 20% of cases of spinal compression show a rise of pressure on jugular compression.

II. **Chemical Changes:** There is a rise in Protein content from the normal of 20 to 40 mgms. up to 400 mgms., even without any block. Once obstruction occurs transudation takes place, between the loculated CSF below the block, and the blood vessels; proteins and pigments diffuse into the CSF, turning it yellow in colour and may clot

spontaneously, the protein content rising to as much as 5000 mg%. (Froin Syndrome). The protein content of CSF may be rarely raised in a block at the cauda equinqa. This was seen in one of our cases. Cervical cord compression may not produce any appreciable chemical change.

Extradural compression causes obstruction to the circulation of CSF and the protein content is usually between 40 and 150 mgms %. Intradural tumours rarely produce a block of the CSF but its (extramedullary) protein content is raised to very high levels. Intramedullary tumours cause a late block and a rise in the protein content is slight. CSF may not be obtained if Cauda equina tumours fill the spinal canal.

**RADIOLOGY :**

(I) **Plain films**: may be normal or may show one or more of the following changes :—

(a) Narrowing of the disc spaces.

(b) Flattening or erosion of one or more pedicles.

(c) Increase in the interpedicular distance (angiomatous malformation) subarachnoid cysts cause widening of the spinal canal without erosion of the pedicles.

(d) Destruction of the vertebral body or lamina.

(e) Deformity of the necks of the ribs which may be splayed out. This is difficult to demonstrate but if seen, it is diagnostic of spinal meningioma.

(f) In special cases like Von Recklinghausen's disease (neurofibroma) the limb bones may show deformity.

(II) **Myelography**: gives an accurate idea of the site and position of the lesion. This consists of injecting a radio-opaque substance (Myodil) into the spinal canal by either lumbar puncture or cisternal puncture and observing the passage of the substance up and down the spinal canal. Films are taken where a block or a filling defect is seen. In such instances, the film is repeated in a few hours or 24 hours later to prevent any false check. Myodil causes mild irritation of the spinal cord on the first and second day. Patients develop pain at the site of the lumbar puncture, pain down the legs and less commonly, neck rigidity and a positive Kernig's sign. In our experience, only one boy had an appreciable reaction with vomiting, neck rigidity and a strongly positive Kernig's sign. His temperature was normal and all signs cleared up after four days of treatment.

Myodil is said to cause arachnoiditis. Two patients who had had Myodil in their spinal canals were screened but no evidence of arachnoiditis was seen. In one, the Myodil had dispersed; in the other, it remained as a continuous shadow.

**Myelographic appearances :**

The cord shows as a column of lighter density surrounded by a dense shadow of contrast medium in the subarachnoid space. This space is narrowest at the cervical and lumbar enlargement and widest below the second lumbar vertebra where the cord ends. Radioluscent nerve tissue may be seen traversing the opaque column. One or two blood vessels may leave their impress on it. Mild constrictions of the column may be seen at the intervertebral disc spaces. In the cervical and lumbar regions, little triangular peaks or even tube like extensions are seen as the contrast medium passes down the sleeves of the arachnoid and dura which surround the nerve roots at each of the inter vertebral foramina.

**Extradural lesions**: Prolapsed disc may show as a filling defect or a complete 'cut off' of the myodil column causing partial or complete block to the passage of Myodil. The transverse margin of the block may resemble a bundle of sticks. Extradural tumours strip the theca off the pedicles and displace both the cord and the theca.

Erosion of the neck of left 6th rib and left pedicle of D6.

Enlargement of intervertebral foramen between D6 & D7.

**Intradural, extramedullary tumours:** Displace the cord but the theca lies against the pedicles. Myodil forms a cap around the tumour.

**Intramedullary tumour:** The cord remains central but is gravid, thinning down the dense Myodil column on either side. The theca lies against the pedicles.

## CASE HISTORIES

Within the last two years, one hundred patients have called at the Jaffna Hospital for neurological disorders of spinal origin. These patients had to be investigated fully including myelography. The aetiology of these cases was as follows:

| | | |
|---|---|---|
| Disc Protrusion — Cervical | | 8 |
| — Dorsal | | 2 |
| — Lumbar | | 50 |
| | | 60 |
| Intraspinal Tumours | — | 10 |
| Cervical Spondylosis | — | 4 |
| Spondylolisthesis | — | 2 |
| Arachnoiditis | — | 2 |
| Adhesions/bands due to a knife injury. | — | 1 |
| Abscess of the spinal cord. | — | 1 |
| Normal myelographic appearance | — | 1 |
| TOTAL | | 100 |

Regarding the sex incidence, disc protrusion was seen only in male patients as was arachnoiditis, cervical spondylosis, adhesions and bands.

Spinal cord tumours occured equally in males and females. The two cases of Spondylolisthesis were both females.

Among the ten spinal cord tumours, three were angiomata of the spinal cord though they are said to constitute only 4% of all spinal cord tumours.

CASE

I

**Disc protrusion and Cervical Spondylosis**

Mr. T, 31 years complained of numbness of the lower limbs of three months duration. This was followed by numbness of the right hand of three weeks duration. He had backache off and on and the pain radiated down the left leg.

On examination, the positive physical signs were 1. Weakness of the right hand and left lower limb. He walked with a drag of his left lower limb. 2. Blunting of pain and touch in the right hand distal to the wrist and below the left ankle. 3. The reflexes were equal on both sides; the

plantar reflex was flexor on the right side but not elicited on the left.

There was no wasting of muscles; No posterior column sensory loss no sphincter disturbances.

C. S. F. showed a protein content of 100 mgm %; V. D. R. L. was negative. Radiology—Plain films—1. Thoracic scoliosis, convex to the right. Anterior and posterior osteophytosis at C5 and C6. Fig. II (a) & (b). Myelography. Normal cervical and dorsal myelogram. Block at L4/L5 due to a prolapsed intervertebral disc.

Disc impression on myodil column

Filling defects caused by disc prolapse of the spinal cord.

**Comment.** Unlike in other forms of compression, muscular wasting is absent; sensory loss is slight.

CASE

II

S. Male. 13 years. Complained of pain in the dorsal region left side and a lump over the mid-dorsal region of three months duration. He had no root pains, neurological signs nor any evidence of neurofibromatosis. CSF showed no abnormality.

Plain X-Ray of the dorsal spines revealed:

1. Erosion of the left pedicle of the 6th dorsal vertebra.
2. Pressure deformities of the left 6th and 7th ribs splaying the ribs.
3. An enlarged intervertebral foramen, between the 6th and 7th dorsal vertebra.

He had a normal myelogram. This was diagnosed as a extradural neurofibroma. He is awaiting operation.

Angioma of spinal cord.

**Comment;** The two unusual features are the age of the patient and the early detection.

CASE

III

Mrs. V, 22 years. Had infantile hemiplegia, left side with a fixed flexion deformity of her left wrist and a scoliosis of spines. The left upper limb was small and underdeveloped. She complained of weakness and stiffness and weakness of the right lower limb with loss of postural sense and some blunting of sensation in the right side below the waist. She had a tendency to fall to the right on walking and distension of the bladder which had to be catheterised.

On examination, apart from her physical deformities, she had an upper motor neurone paralysis of the lower limb (R/S) in addition to a lower motor neurone paralysis of her left upper limb.

2. Blunting of all forms of sensation below D9.

On lumbar puncture, sufficient fluid could not be obtained for examination.

Anterior osteophytosis at C5 & C6.

**RADIOLOGY:** Plain films—showed no bony involvement. Lumbar myelogram: Fig. iv (a), (b) Complete block at D9. The theca showed a tendency to get stripped off the right pedicle of D9, and the cord was displaced to the left.

A cisternal puncture myelogram showed similar features at the level of D8. An extradural tumour was diagnosed. At operation a neurofibroma of the 8th dorsal nerve 1" × ¾" situated posteriorly and to the right was removed. Since operation, this patient is asymptomatic as far as her right leg is concerned. She has regained full control over her bladder.

Posterior osteophytosis at C5 / C6 & C6 / C7.

CASE

IV

Intramedullary tumour

Mrs. M, 38 years was admitted with a history of backache, weakness of both lower limbs and inability to walk without support for 3 years. She also had numbness of the body below the lower chest for one year. This was followed by numbness of the left foot and then the right foot in addition to precipitancy of micturition which was present for six months.

O/E, the following were the findings:

I. Upper motor neurone paralysis of both lower limbs.

II. Loss of abdominal reflexes.

III. Loss of postural sense.

IV. Diminution of sensation to touch and pinprick below D7.

The CSF was under normal tension. It showed no xanthochromia. Protein could not be estimated due to insufficient fluid.

RADIOLOGY: Plain films were normal.

Myelography: Complete block of myodil at D8., where a crescentic outline was obtained. An intradural tumour was diagnosed as only the cord was deviated.

FIG. V.

At operation, a large tumour 2½" long was found lying anterior to the cord. It was removed in toto.

Biopsy revealed, meningioma. Since the operation power has returned to the left leg and sensation was present below D7 though still blunted, and there was now precipitancy of micturition.

CASE V

**(Intramedullary) Angioma of the cord**

Mrs. S, 60 years, transferred from Chavakacheri hospital for prolapsed haemorrhoids. She was found to have a distended bladder on admission and oedema of the feet, in addition to the prolapsed haemorrhoids, which were reduced. But the distension of the bladder persisted; this was followed by incontinence of urine and partial prolapse of the rectum. She had numbness and weakness of the left lower limb: she could not say for how long.

Neurological examination showed (1) Upper motor neurone lesion of the left lower limb (2) Abdominal reflexes present (3) Diminished pain and touch sensation in the left lower limb. (4) Loss of joint sense in both lower limbs.

CSF examination was normal.

RADIOLOGY Plain films-NAD. except for early osteoarthritis of the vertebrae.

Myelogram: showed an obvious angioma of the cord, which was very extensive. As the lesion was too extensive for adequate treatment, the prolapsed rectum was repaired, and she was transferred back to Chavakacheri.

Arrest of myodil column at by a tumour. D 10; "caps" formation atend the tumour and cord is displaced.

CASE VI

**Tumour of the cauda equina**

Mrs. J. 28 years, a history of severe pain starting in the right knee and spreading upwards to the right sacroiliac joint of twenty months duration. Pain was worse at rest but relieved by walking. The pain spread over to the left hip. This was followed by numbness of the lower limbs and diminution of sensation in the anal region—a feeling of a lump in the anal region as described by her. Precipitancy of micturition led to incontinence and complete loss of control over the bladder and rectum, for six months during which period she was bed ridden. She conceived soon after her illness commenced. Her baby was delivered by Caesarean Section. Her symptoms continued to progress.

O/E There was generalised wasting and weakness of all muscle groups of both lower limbs, worse on the left than on the right.

(2) Absent knee and ankle jerks on both sides.

(3) Loss of the anal reflex.

(4) Loss of sensation over the sacral area and outer borders of the feet

**CSF.** Straw coloured; Proteins more than 200 mgms%; Sugar 46 mgm%.

**RADIOLOGY** Plain films—NAD.

Myelography: Arrest of the myodil column at the lower border of the second lumbar vertebra, very likely by a tumour of the cauda equina. This patient refused operation and left hospital.

Arrest of myodil column at L2 by a tumour of the cauda equina.

**CASE VII**

**Mr. N.** 35 years C|O incontinence of urine on lying down, of two months duration. He also had loss of sensation in the legs and small of the back. No root pains. The only positive findings on examination were diminution of sensation over the right foot and saddle-back analgesia.

**CSF.** Xanthochromia; Protein 100 mgm%; Sugar 16 mgm%; RBC 45 cells/c.mm; V. D. R. L. was negative.

When lumbar puncture was done for introducing myodil, dark blood stained fluid was obtained. This was not suitable for examination.

**RADIOLOGY** Plain film showed 1. Indistinct pedicles of L5. 2. Deep concavity of the posterior aspects of the body of L5 and to a less extent of L3 and L4.

**Myelography:** Peculiar appearance was noted on screening. The myodil remained as a shapeless blob, excentrically placed in the spinal canal. On tilting the table head down, two globules passed as far as L3. It was believed that the myodil had been introduced into the cavity of the tumour.

Laminectomy from L1 to S1 showed a distended dura from L3 to S1. Within it was a bilocular cyst with a papilliferous growth in its walls. Chocolate coloured fluid and myodil were expelled from the cyst. The nerves were adherent to the cyst. The cyst was therefore de-roofed, and cleared of the contents.

Biopsy revealed a Haemangioma.

Following operation the patient initially regained control over his bladder. Later he developed retention with overflow followed by urinary infection which progressed to uraemia and inspite of treatment, the patient died; after one and a half months. Urinary infection is common on lesions of the cauda equina and it adds to the gravity of the prognosis.

## SUMMARY

I.

The main causes of spinal cord compression as seen in Jaffna are outlined.

II.

The clinical and radiological features are presented.

III.

Intraspinal tumours contributed to 10% of all cases taken up for myelography.

IV.

The different causes of spinal cord compression have been illustrated by case histories.

ACKNOWLEDGEMENTS

I wish to thank the specialist staff of Jaffna Hospital for referring these patients to me and helping me with the clinical aspects and the progress after treatment. Dr. D. F. Weinman and Dr. Jayamana of the N. S. U., General Hospital, Colombo, for the details of operations of cases referred to them and for permission to reproduce myelogram Fig. IV (b) done by the cisternal route and Dr. A. Gabriel for his help and encouragement to write this article.

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# POISONOUS SNAKES OF CEYLON

*By*

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IN the rural areas of Ceylon we are not infrequently confronted with cases of snake-bite. The victim is often frightened out of his wits. His friends begin to panic. There is a wide difference of opinion as to the best place to go for treatment. At this juncture identification of the reptile, would help to plan a definite course of action, and what to advise the patient. Of over 60 identified land snakes in Ceylon, only 5 are poisonous to man—Cobra, Russell's viper, Indian krait, Ceylon krait and Saw-scaled viper.

**COBRA**—Naia tripudians — Naya (Sinhala)—Naha pambu (Tamil)—This species is easily recognised by its hood and by the characteristic spectacle mark present on the back of the hood. It has to be distinguished from the common rat snake (garandya S. Sarai T). The average specimen is about 4 or 5 feet long and has a girth of about 4 inches. It is evenly distributed throughout Ceylon, except in the hill country above 6000 feet. The cobra hunts mainly by day. It rarely bites unless provoked. In this event, it raises its hood and strikes swiftly usually above the ankle. A full grown member of this species is said to contain enough poison to kill 15 men. However, less than 50% of humans bitten by cobras show any symptoms at all and only 5% prove fatal. This is because the cobra bites man in defence and so, rarely injects a lethal dose.

**INDIAN KRAIT**—Bungarus caeruleus—Tic karavala (S)—Yennai viriyan (T)—This is a thin black snake rarely reaching 4 feet in length and with narrow paired white lines arranged on its dorsal aspect and sides. These white lines do not stretch into the belly, which therefore, has a pearly white colour. In older snakes the white lines tend to disappear and the snake becomes oily black.

It is a very rare snake found in the scrub-by jungles of the plains of Ceylon and very often close to or even inside dwelling places. Though a very poisonous snake, it is one of the most inoffensive. It bites only when really much provoked eg. when it is crushed in bed by an unsuspecting sleeper. Its poison is said to be about 5 times as potent as cobra poison.

**CEYLON KRAIT**—Bungarus ceylonicus —Polon karawala (S)—Vari pidayan (T)—This is also a thin black snake reaching to about 5 feet in length. It has single, broad white bands running across the dorsum of its body and stretching across its belly. The belly of this Ceylon krait has alternate broad grey and white bands.

It is found less rarely than the Indian variety and inhabits the plains and hilly areas at elevations up to about 2000 feet. In all other respects its habits are like those of the Indian krait.

The two kraits are similar in appearance to the Ceylon wolf snake because this too is banded. It, too is a very common visitor in and around homes. However, enlarged vertebral scales and undivided subcaudal scales are characteristic of kraits and this, along with the different banding in the two kraits helps to identify them.

**RUSSELL'S VIPER** — Vipera russelli—Tic polonga (S)—Kannadi viriyan (T)—It is a big made snake often about 5 feet in length. The head is triangular and the neck constricted. It has a brownish body on which a black regular necklace pattern runs down along the spine. Two other similar necklaces run on either side of the spine.

The Russell's viper has to be distinguished from the python which however, has a round head, a broad neck and the markings on its body are irregular.

Though not common, it is found all over the country. It usually hunts by night and rarely attacks man. It usually carries in its poison gland one and a half times as much poison as is required to kill an adult. Its poison, however, is only one third as toxic as cobra venom.

**SAW-SCALED VIPER**—Echis carinatus —Vali polonga (S)—Surutddai pambu (T)— It is a light brown, relatively stout snake reaching to about a foot and a half in length. The head is oval and the neck is very constricted. On the head is the typical 'dagger' or 'bird's foot' marking. The body is spotted with black or dark brown markings. When confronted, this reptile is seen double coiled and in perpetual motion making a rustling noise thereby, with its saw edged scales. It is indeed the only really aggressive snake in Ceylon and will dart and attack a passer-by without provocation.

It is met with in the sandy areas towards the north of Ceylon and in the Jaffna peninsula. It roams about by day. At one bite it injects twice the lethal dose for an adult and its poison is 5 times as powerful as the cobra's.

Snake Venom is a complex which contains a series of toxic principles, of which some have local effects and others, after being absorbed, affect different vital centres. Symptoms seen in victims are the results not of any single toxic components in the venom. These toxic principles, although to some degree are similar in almost all snakes, vary greatly in the relative proportion in which they exist in the different snakes.

The neuro-toxic principle predominates in the cobra and kraits and the haemo-toxic principle predominates in the two vipers. Some of the other factors found in snake venom are the cardio-toxic factor, the haemolytic factor, the anti-coagulating factor, inhibitors of respiratory enzymes, cholinesterase and cyto-toxins.

**SYMPTOMATOLOGY**—Fear and panic are often the most prominent symptoms to begin with. Not infrequently, allergic symptoms are also met with. The actual signs and symptoms resulting from the venom itself will depend by and large, on the size of the poisonous snake and the general state of health of the victim.

Cobra Bite—Local—Often the fang marks are well above the ankle. Pain begins almost immediately. The part begins to swell in about half an hour and swelling steadily increases in size and extent till sometimes a whole limb is involved. There is a slight oozing of a sero-sanguinous fluid from the marks. The swollen limb may appear bluish and tense. Death of superficial tissues occurs later.

General symptoms of drowsiness can appear as early as in about half an hour. This is followed at variable intervals by drooping of the eye lids, difficulty in speaking and opening the mouth and weakness of the lower limbs with later, an ascending paralysis. The head falls backwards—"broken neck syndrome"—due to paralysis of neck muscles; the lower lips fall away from the teeth, causing dribbling of saliva. Owing to the paralysis of the external respiratory muscles and diaphragm, breathing becomes laboured and finally stops; but the heart often continues to beat. The blood pressure is usually below normal. Occasionally haemorrhages are seen, suggesting haemolysis.

Krait Bite peculiarly, is not associated with local oedema or discoloration. Sometimes local symptoms do not even arouse suspicion of snake bite, because the immediate smarting sensation is often followed by numbness and partial anaesthesia. Otherwise, the local as well as the general symptoms are very much like in cobra poisoning, but Krait venom is much more potent. However, a very characteristic difference

between cobra and krait envenomation, is the severe abdominal pain after Krait bite. This is said to be due to submucous haemorrhages in the gastrointestinal tract.

Russell's viper Bite — Local — Within minutes severe burning pain occurs at the site of the bite and in about half an hour, swelling begins. Within four hours it can become quite massive. In 8—10 hours the swelling can involve the whole limb. Bleeding from the fang marks continues in a steady ooze and there is considerable subcutaneous haemorrhage. If however, clotting takes place at the fang marks, it is almost positive evidence that no poisoning has taken place. The swelling, in a few days, leads on to a certain amount of local necrosis and sloughing, but the sloughing is generally of a lesser degree to that in cobra poisoning.

General—Depending upon the degree of poisoning, a general depression sets in, in about 2 hours; but if severe poisoning has taken place this can occur even in half an hour or sooner. The pulse becomes weak and rapid. Mental activity is depressed in about 3 to 4 hours time. Nausea and vomiting begins; the vomitus is sometimes blood stained even at this stage, in fact even very much earlier. Blood stained expectoration can be demonstrated on asking the patient to cough hard. Cold and clammy skin, bleeding from gums, bleeding under the skin, blood in the urine, frank blood in stools—all these are seen at any time within 5 hours.

Haemoglobin percent after a few days is found to fall. Clotting defect is the outstanding feature in systemic poisoning. Blood clotting time begins to get delayed in about half an hour. Incoagulable blood is noticed even after 10—12 days in untreated cases. The total urine out-put falls. Proteinuria is present and R.B.C. are found in urine.

Saw-scaled viper Bite—The general symptoms of its bite are identical to Russell's viper bite though many times worse and much quicker in onset. Even the neurotoxic effects are more pronounced than in Russell's viper poisoning.

TREATMENT—The patient is made to lie down and rest. The limb is immobilised preferably with a splint. A tourniquet may be allowed to stay if it has already been applied. The patient is best transported to hospital on a stretcher, thereby allowing as little movement as possible and thus delaying absorption of the poison. If ice is available the portion of limb around the fang marks may be profitably packed in ice. In hospital the limb is examined for fang marks. On seeing the fang marks with the telltale ooze, then it can be established that one is dealing with a case of actual snake bite. The procedure now adopted depends upon the degree of envenomation.

If general symptoms have developed then polyvalent Anti Venom serum (Haffkine Institute) is given without delay.

If only local swelling is seen development of general symptoms is watched for and Anti venom treatment is with-held.

If there is no local swelling, it may mean that no poison has been injected or that the patient is being seen very shortly after the bite, in which case time must be allowed for symptoms to develop.

If a tourniquet has been applied it should be released slowly and general symptoms should be watched.

The initial dose of the serum is between 60—100 c.c. intravenously of the polyvalent Anti venom serum. About half this quantity is repeated in 2—4 hours or sooner by the same route and about half that dosage is given in about 6 hours; intramuscularly in a case of severe envenomation. The quantity, speed and frequency of injections of the Anti venom serum is decided on by the condition of the patient and by the response to the initial massive dose. If

time will permit, sensitivity tests are carried out before giving the injections.

An antihistamine is given as a routine, intravenously and then continued by mouth if the patient is fit.

A 5% Glucose drip is instituted as soon as envenomation is suspected, and this may be replaced with a blood transfusion, if massive haemorrhages occur.

Along with the serum 100 mgm of prednisolone are given, to help to combat any allergic reactions due to the Venom and due to the serum. (Use of steriods, in poisoning, however, is a very controversial subject).

It cannot be over emphasised that Anti venom serum is life-saving, if given along with other supporting therapy. As long as there is even a trace of life left, heroic doses of the serum given promptly and fast are effective.

For the local condition, at the beginning, rest alone would suffice. Later, when sloughing occurs, surgical debridement and early grafting would be necessary.

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# ABO & RH BLOOD GROUPS AMONG THE TAMILS AND THE ANTHROPOLOGICAL SIGNIFICANCE OF BLOOD GROUPS

*By*

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THE distribution of the A-B-O blood group of 9000 Tamil patients (Table II) and the distribution of the Rh blood groups of 6618 Tamil patients (Table IV) admitted to the Jaffna hospital over a period of 3 years (1963—1965) is recorded here. Results of similar studies by other workers have also been included. A statistical analysis of these figures on the basis of accepted theories of inheritance has also been done. (Table I) The inheritance of the A-B-O and Rh system is discussed. The value of blood groups in anthropological studies is stressed.

| Author | Race | No. examined | phenotype frequencies A% | B% | AB% | O% | gene frequencies p | q | r | D | PE | D/PE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Hill (1937) | S | 712 | 26.26 | 24.72 | 1.97 | 47.05 | .151 | .146 | .686 | .017 | .003 | 5.66 |
| Seneviratne (1941) | S | 3606 | 22.82 | 26.51 | 4.25 | 46.42 | .146 | .169 | .678 | .007 | .0015 | 4.66 |
| Koch & Weeratunga (1953) | S | 340 | 22.35 | 25.88 | 2.36 | 49.41 | .134 | .151 | .700 | .015 | .004 | 3.75 |
| Fernando (1958) | S | 925 | 23.35 | 23.89 | 5.62 | 47.14 | .157 | .163 | .686 | .006 | .003 | 2.00 |
| Wickremasinghe Ikin Mourant & Lehman (1961) | S | 1068 | 16.86 | 29.31 | 5.52 | 48.31 | .117 | .194 | .693 | .004 | .003 | 1.33 |
| Hill (1937) | T | 136 | 13.97 | 30.8 | 4.41 | 50.74 | .094 | .194 | .712 | 0 | .006 | 0 |
| Seneviratne (1944) | T | 561 | 18.36 | 29.41 | 6.24 | 45.99 | .134 | .200 | .678 | .012 | .004 | 3 |
| Koch & Weeratunga (1953) | T | 147 | 21.77 | 27.89 | 6.12 | 44.22 | .151 | .188 | .663 | .002 | .008 | .25 |
| Fernando (1958) | T | 307 | 21.50 | 28.34 | 5.86 | 44.30 | .146 | .188 | .663 | .003 | .005 | .6 |
| Wickremasinghe Ikin Mourant & Lehman (1961) | T | 660 | 21.36 | 31.67 | 7.27 | 39.70 | .157 | .219 | .632 | .008 | .004 | 2 |
| Wickremasinghe | V | 254 | 7.09 | 44.88 | 2.75 | 45.28 | .051 | .279 | .671 | .001 | .004 | .25 |

**Table I.**

The distribution percent of A-B-O phenotypes and gene frequencies among the Sinhalese, Tamils and the Veddhas. Also giving a statistical analysis of these figures. S—Sinhalese T—Tamils V—Veddhas. For the notations p, q, r, D, PE and D/PE see section on statistical analysis.

| Author | Race | No. Examined | phenotype frequencies | | | | gene frequencies | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | A% | B% | AB% | O% | p | q | r | D | PE | D/PE |
| Joseph | T | 9000 | 20.56 | 31.31 | 5.36 | 42.77 | .140 | .206 | .656 | .002 | .001 | 2 |

**Table II Present Investigation**

The distribution percent of ABO phenotypes and gene frequencies in 9000 cases studied by the author. (Notations same as in Table I).

Koch (1952) studied the distribution of the Rh blood groups (a) In a group of University students (b) In patients of the De Soysa Maternity Home. Only the investigations done on the University students are reproduced in Table III since as Koch himself contends the high figure for the Rh negativity that he obtained in the second group was due to the sample being biased in favour of a higher incidence due to the selected admission of cases with previous obstetric histories of abortions, still-births and neo-natal deaths due to Rh incompatibility.

| Community | Total | Rh positive | Rh negative | %Rh negative |
|---|---|---|---|---|
| Sinhalese | 168 | 162 | 6 | 3.6 |
| Ceylon Tamils | 46 | 43 | 3 | 6.5 |
| Ceylon Moors | 6 | 5 | 1 | 17.0 |
| Burghers | 3 | 3 | 0 | — |

**Table III.**

Rh distribution among university students (Koch 1952)

| Community | Total | Rh positive | Rh negative | % Rh negative |
|---|---|---|---|---|
| Tamils | 6618 | 6307 | 311 | 4.69 |

**Table IV.**

Rh distribution among 6618 Tamils in the present investigation.

**INHERITANCE.** Blood group of an individual is genetically determined. The Chromosomes of a cell carry on them numerous loci which are occupied by genes and which determine the genetic constitution of an individual and are responsible for the passage of hereditary traits from generation to generation. In its application to blood groups, the genes confer the property for the manufacture of the respective antigens. Blood group genes differ in their antigenecity. A and B being dominant to O which is recessive. The inheritance of blood groups is by a pair of allelic genes and since these genes are not found in the sex chromosomes blood group factors are not sex linked.

**Mechanism of inheritance of the ABO system.** Bernstein in 1924 postulated that the inheritance of the agglutinogens A and B depended on 3 allelic genes A, B, O. There is only one locus for the genes determining the blood group in a single chromosome, at which locus any one of the genes A, B or O may be situated. Since each germ cell contains only one of the 3 genes, 6 different genotypes would result from the

combination of 3 possible kinds of sperms and 3 possible kinds of sperms and 3 possible kinds of ova. Of these 6 three would be homozygous and three would be heterozygous as shown in table V.

| PHENOTYPE | GENOTYPE | |
|---|---|---|
| | homozygous | heterozygous |
| AB | — | AB |
| A | AA | AO |
| B | BB | BO |
| O | OO | — |

**Table V**

From the statistical standpoint it is possible to calculate the gene frequencies from the frequencies of the phenotypes in any population. Data obtained from these studies holds out in favour of the Bernstein's hypothesis.

**Mechanism of heredity of the Rh factor.** Fisher postulated the gene linkage theory where each chromosome would carry 3 of the 6 genes C, D, E, c, d, e. Hence it would be either C or c, D or d, E or e.

Weiner 1948 has challenged the gene linkage theory of Fisher and has stated, "that the Rh groups are inherited not by a series of linked loci but by a series of 8 allelomorphic genes all capable of occupying the same locus. On the basis of the theory of multiple genes (8), not counting the more recently established alleles 36 different genotypes are possible.

## CALCULATION OF THE GENE FREQUENCIES IN THE ABO SYSTEM AND BERNSTEIN'S FORMULA AND STATISTICAL ANALYSIS

Weiner (1946) worked out a formula for calculating the gene frequencies. Let p, q, r represent the gene frequencies of genes A, B and O respectively.

Then,

$$p = 1 - \sqrt{O + B}$$

$$q = 1 - \sqrt{O + A}$$

$$r = \sqrt{O}$$

where A,B, and O are the percentages of these groups in any given population. $p+q+r$ should theoretically be equal to unity. However there is a difference and the difference D is given by $1-(p+q+r)$ and should not be greater than what would be expected as a result of chance alone.

Bernstein has also worked out a formula for the standard error of a series

$$\sigma D.\sqrt{N} = \sqrt{2(1-p)(1-q)} \quad \ldots\ldots (1)$$

where $\sigma D$ is the standard error, and N is the number in the series.

American authors prefer to use Probable Error (PE) instead of standard error and

$$PE = .6745 \times \sigma D$$

Hence substituting for $\sigma D$ in equation (1) we have

$$PE = \frac{.6745}{\sqrt{N}} \sqrt{2(1-p)(1-q)}$$

Now to verify whether deviation D is due to chance alone the factor D/PE is calculated. If this factor is less than 3 then the deviation is not significant. Hence this analysis would serve as a means of verification of the accuracy of the investigations.

Gene studies have not been undertaken in this series of cases analysed in relation to the Rh factor and hence working out of gene frequencies of the Rh system are not indicated here.

## ANTHROPOLOGICAL SIGNIFICANCE OF BLOOD GROUPS.

Various methods have been used in the anthropological classification of the human race.

Such characters as skin, hair and eye colour; skeletal traits such as stature and shape of cranium have been used for purposes of classification. But "such characters are not inherited by any simple rule and geneticists have shown that in the case of

some characters their quantitative expression is controlled by a number of independent genes". (Mourant 1954) Furthermore such characters are subject to environmental and nutritional factors.

Differences in culture are so much influenced by the environmental factors and there is such a degree of overlap that cultural characteristics are not of much significance.

Few other characters such as (1) haematological abnormalities eg. thalassaemia and the sickle cell trait. (2) biochemical characters eg. ability or inability to taste phenylthiocarbamide and the ability or inability to smell hydrocyanic acid. (3) Colour blindness, have also been used in anthropological work but have obviously limited application.

The use of blood groups in anthropological work has the following advantages.

Firstly the blood of every individual can be readily and accurately classified (grouped) without any overlap. Errors in technique in sampling etc. may be eliminated using the Bernstein equation.

Secondly the blood group of an individual is determined at the time of formation of the zygote and undergoes no change during the life of an individual due to environmental or nutritional factors.

Thirdly as "Bernstein has himself pointed out if it is known that a certain population has resulted from crosses between two races it is possible to predict the blood group frequencies in this population if the blood group frequencies of the original population are known" (Wiener 1945). Hence it is possible to test theories concerning the racial derivatives of populations.

Fourthly due to random selecting of mates the blood group distribution in a race remains constant from generation to generation in the absence of admixture with other races.

It might also be said that large volumes of accurate classified data are available in this field than in any other anthropological criterion.

Fig. 1 shows the serological composition of the Tamils (using authors figures) in relation to some of the other populations of the world represented by means of triangular co-ordinates (after Wiener).

No comment is however being made on the anthropological significance of the position of the Tamils or their racial derivation as it would be outside the scope of an article in a medical journal.

CONCLUSION: An analysis of the ABO blood groups of 9000 Tamils has been done. Statistical tests have been carried out to check the accuracy of the investigation. The results of this survey corresponds with the figures of Wickremasinghe, Ikin, Mourant and Lehman (1961) who analysed 660 cases.

Distribution of the Rh factor studied on 6618 individuals yields a value of 4.65% Rh negatives among the Tamils which is lower than Koch's figures (1952) of 6.5% where only 46 individuals were studied. Koch's subsequent analysis of a larger series was not available for inclusion in this article.

Anthropological significance of blood groups has been discussed and the position of the Tamils in relation to some of the populations of the world in terms of their ABO serological composition has been indicated in Fig. I.

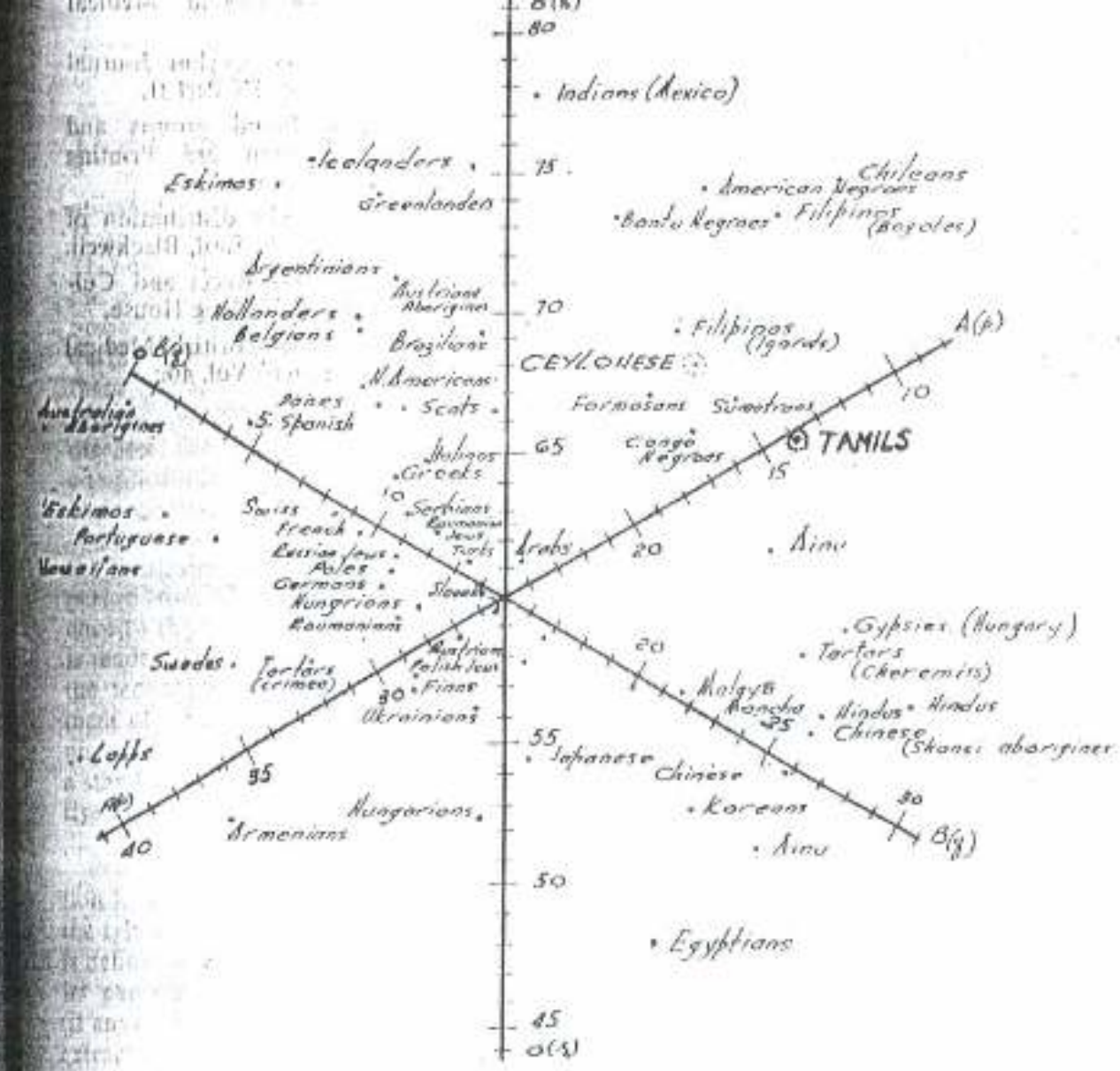


FIG. 1

ABO Serological Composition of Ceylonese and Tamils in relation to some other races. Diagram adapted from Blood Groups and transfusions by Wiener third edition ( Fernando 1958 Ceylon Journal Medical Science Vol. IV Part II) position of Tamils obtained from Author's Figures.

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# CATARACT EXTRACTION

## An analysis of 220 consecutive operations

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IN this paper it is proposed to analyse the results of 220 consecutive cataract operations done by me in the Jaffna General Hospital during the period from 1-1-65 to 31-3-66. Special reference will be made to the complications arising at the time of the operation, the complications met with during convalescence, the way in which these complications were managed, the presence of other diseases including ocular diseases, and the visual results. When the complications are discussed reference will be made to relevant cases of another series of 216 cataract extractions done by me at the Batticaloa General Hospital during the period from 1-1-62 to 31-12-64. No reference to the work of any other Eye Surgeon is made and, no claim to any newness in the technique of operation or the management of complications is made. The technique of operation, to be described later, is a standard one well tried out by numerous Eye Surgeons.

Before I describe the technique of operation let us consider a few simple facts about the types and causes of cataract. Cataract is defined as the opacification of the lens or its capsule. After-cataract is the opacity (if any) that is left after the extracapsular extraction of a lens that is cataractous.

**Types of Cataract:**—Cataract may be congenital or acquired. In this paper we will only discuss the latter type as the method of extraction to be described is not used in the treatment of congenital cataract.

Acquired Cataract:—Acquired cataract may be—

(1) Senile.

(2) Traumatic.

(3) Secondary (complicated).

(4) Metabolic.

(5) Endocrine.

This list is not exhaustive. It however includes the commoner types. In Ophthalmic parlance the secondary type is known as complicated cataract. Secondary cataract is also used to refer to the After-Cataract. Complicated cataracts are those that arise secondary to or complicating disease of some other part of the eye. The commonest type of cataract is the senile type. The senile type also carries the best prognosis. The prognosis in the 2nd, 3rd, and 4th types depends on the extent to which the vitreous, choroid, retina and optic nerve are damaged by the underlying disease or antecedent trauma.

### The Technique Employed

The conjunctival smear is sent routinely for culture and antibiotic sensitivity. The lacrymal passages are not syringed routinely. If an obstruction is thought to be present in the lacrymal passages syringing is done. Obvious cases of obstruction are treated suitably. Antibiotic drops are used for a few days prior to the operation, after the conjunctival smear has been taken. If the ABS report shows that the antibiotic being used has to be changed we do so and send the smear for another culture till the smear is clean. If the pupil cannot be adequately dilated with atropine, the patient is given a sub-conjunctival injection of mydricaine about 15 minutes before the operation.

An injection of Penicillin 1 million units is given intramuscularly (after a sensitivity

test) 12 hours before the operation. At the same time the patients are given 4 tablets of Sulphadiazine by mouth. Penicillin and Sulphadiazine are continued for five days after the operation. Phenobarbitone gr. 2 is given nocte. The eye is then covered with an eyeshade. On the morning of the operation day the patient is given gr. 2 phenobarbitone and 2 tablets (1 Gm) of Sulphadiazine at 7 AM.

Premedication of the patient is effected as follows:—Pethidine 75 to 100 mg IM depending on the age and build of the patient. Largactil (chlorpromazine hydrochloride) 25 mg IM or 50 mg orally is also given depending again on the build and age of the patient. As I have had a few of my patients collapsing after this premedication in selected patients the Largactil had been left out.

**Anaesthesia and Facial Akinesia**:— The eye is anaesthetised by the use of either Cocaine 4% drops or Lignocaine 2% (when there is a shortage of cocaine). A retro-ocular injection of 1½ cc of 2% Lignocaine is used to block the ciliary ganglion This causes anaesthesia of the itis and a lowering of the ocular tension. It also enhances the surface anaesthesia. Facial Akinesia is effected by blocking the facial nerve as it crosses the neck of the mandible (O'Brien's method). This was used in every case. There was no need to resort to any other method. The lid margins are infiltrated with Lignocaine 2% in order to facilitate the application of lid sutures which are used to keep the lids apart during the operation. The upper lid suture is retained in order to close the lid after the operation. The globe is always kept steady by the combined use of a superior rectus suture and medial rectus fixation with forceps. The section is made with a Graefe knife. This is followed by simple or combined extraction depending on the nature of the case. Slight modifications are made if complications are present or anticipated.

## AGE AND SEX DISTRIBUTION

Of the 220 cases 60% was in males and

Table 1

| Sex | Number of cases |
|---|---|
| Male | 132 |
| Female | 88 |
| Total | 220 |

40% was in females. (see table 1). In this series 83.2% of the patients were over 50 years of age when they sought treatment. The maximum incidence of cataract was in the 40 to 60 years age group. (54.5%). (see table 2).

Table 2

| Age Group | Number of cases |
|---|---|
| Below 20 yrs. | 1 |
| 20 to 40 yrs. | 13 |
| 40 to 60 yrs. | 120 |
| 60 to 80 yrs. | 85 |
| Total | 220 |

## OTHER FEATURES

In 50.5% of cases the patient was blind in both eyes owing to cataract. In 41% of cases there was vision of over 3/60 in one eye and blindness in the fellow eye. In the remaining 8.5% one eye had already been operated on. (see table 3).

Table 3

| Blind | Number of cases |
|---|---|
| In both eyes | 111 |
| In one eye | 90 |
| In one eye with aphakia in other | 9 |

**Type of operation:**—In 75 patients intracapsular extraction was done. This means that

**Table 4**

| Type of Operation | Number |
|---|---|
| Extracapsular | 145 |
| Intracapsular | 75 |

the lens was taken out while still enclosed in its thin capsule. In 145 patients extracapsular extraction was done. This means that the capsule was incised and only the lens cortex and nucleus were removed. The lens had to be removed with a vectis in 5 patients. Of these 4 patients had good vision. One had no sight after operation.

## COMPLICATIONS

**1. Hyphaema:**—Bleeding into the anterior chamber of the eye may take place at the time of the operation either from the wound or the iridectomy, if one is done. It is best to avoid an iridectomy in patients who are diabetics, hypertensives or who are very old and arteriosclerotic. Bleeding that takes place at this time usually stops as soon as the anterior chamber is irrigated. These patients should be well sedated at night and the head end of the bed should be raised. Vitamin C and K may be given IM. In some cases the bleeding into the anterior chamber takes place during the period of convalescence. This bleeding usually takes place on the 4th or 5th day after the operation. The patient usually states that he felt pain in his eye and was awakened from his sleep. In these cases the blood is probably from torn capillaries in the healing wound. Some patients may admit that they had hit the eye accidentally while sleeping. In my cases I observed that the presence of a conjunctival flap increased the likelihood of a hyphaema. If on the 3rd post-operative day a heavily vascularised conjunctival flap was seen the patient was given Pethidine 75 mg IM for the night and his eye was not dressed for two to three days. There were 24 patients with hyphaema out of the 220 in this series. (11%). Most hyphaemas clear up in 3 to 4 days. At times hyphaema persists for 14 days, or even longer. Out of 216 cataract extractions done at the Batticaloa Hospital 12 patients had hyphaema (5.5%). Though the incidence was lower than in the present series, 2 of these 12 patients lost their sight entirely owing to glaucoma secondary to the hyphaema. They also developed a condition known as corneal staining. No treatment was of avail. Let us consider these cases for a moment. The first patient had cataract secondary to uveitis. He also had a conjunctival flap. He bled from both the iris and the flap. The blood was present from the second post-operative day onwards. The second case was in a Female Attendant who had reached the retiring age and was almost blind in both eyes. These and domestic worries had made her emotionally very unstable and mentally depressed. After the operation she, unknown to me, was in the habit of discussing her troubles with the ward staff and weeping. I have no doubt this made matters worse for her, because the congestion caused by weeping would increase the oozing into the eye. The story does not end there. This patient was admited to the Jaffna Hospital and the other eye was operated on. She was put on drugs to prevent depression of the mind and the ward staff was warned to avoid talking to her about her troubles. An iridectomy was not done. Her recovery was uneventful. She had a vision of 6/6 on discharge. She had not wept over her troubles this time. There was no hyphaema.

**2. Vitreous loss:**—In the Jaffna series there were 8 cases of loss of vitreous. In no case was much vitreous lost. No eyes were lost as a result of vitreous loss. All patients with vitreous loss are nursed with the head raised. No dressing is done on the first day; it is put off for a day or two. At the time of dressing the patient is asked not to look downwards.

**3. Prolapse of Iris:**— Prolapse of Iris through the wound was seen in 8 patients.

(3.6%). In the Batticaloa series there were 6 (2.8%). The iris prolapse is best abscised or cauterised after the eye has settled down. Earlier intervention has not been of value. This often needed a further operation. I have often done a broad iridectomy when a prolapse has been anticipated. A lax iris that keeps on floating into the wound during irrigation of the anterior chamber, is one that might become at the least adherent to the wound. In these cases I convert the iridectomy into a broad iridectomy preferably before the lens is extracted.

**4. Delayed reformation of the Anterior Chamber :**—This was met with in 9 patients. I have counted only those cases in which the AC remained flat for over 6 days. Diamox orally and the dilation of the pupil if need be with a sub-conjunctival injection of Mydricaine has been sufficient to reform the AC. Avoidance of straining is desirable. In one patient I had to resort to the injection of sterile air into the AC.

**5. Irido-cyclitis :**— This complication was seen in 4 patients. One of these was probably endogenous in origin. This patient had bronchiectasis. Of the 4 patients two had no vision, after operation.

**6. Infection :**—In the Jaffna series 3 patients had post-operative infection. The first two belonged to the same batch. In one case the infection was very acute and treatment was not effective. He had 0.5 Gm of streptomycin subconjunctivally at first. Later he was given oral chloramphenicol. He was very restless and even slept on the floor at night. The other patient got over the infection. He had sub-conjunctival Penicillin and streptomycin. This was followed by oral chloramphenicol and prednisolone. The patient had a membrane occluding the pupil. On the 19th post-operative day the membrane was needled. The patient had one more sub-conjunctival injection of ½ Gm streptomycin. He was also given prednisolone orally. Recovery from this was smooth. He had a vision of 6/9 when he was discharged. I would like to stress the fact that prednisolone was given only when it was very clear that the infection was under control, and the patient had adequate antibiotic cover, during the administration of prednisolone. Both infections were exogenous in origin. It is of interest to note that the patient just described is now in the ward and has survived the 6th post-operative day with no sign of infection, after cataract extraction in the other eye. The 3rd patient belonged to a later batch and the infection was very mild. He responded well to oral chloramphenicol. He left with a vision of 6/18 after a stay of 35 days.

In the Batticaloa series there were 6 cases of infection. Of the 6 two were endogenous in origin and, 4 were exogenous. Three of the four cases of exogenous infection responded to antibiotic therapy. Two of them had good vision when discharged. The two endogenous infections failed to respond to any treatment. It is worth describing one of the two endogenous infections. The patient was an Indian Muslim male 65 years of age. He was first seen by the General Surgeon for a renal complaint. A nephrectomy was done. The patient was then sent to my ward for cataract extraction. A combined extracapsular extraction was done. The patient had a fair amount of sight when discharged. One month after the operation on the eye he was back at the eye clinic with pain in the eye that was operated on. There was a hyphaema and the vitreous was opaque. He never regained sight in that eye. He insisted that I should operate on the other eye. I sent him back to the surgeon who removed a large renal calculus from the only kidney he had. He was sent back to my ward certified free from renal infection. The operation on the second eye was successful. He had 6/60 vision and continued to have good sight for months after. The second operation was also extracapsular.

In Batticaloa conjunctival smears were not examined for lack of facilities. In Jaffna though the smears were cultured there were 3 infections. Infections have

occured in spite of Antibiotic and chemotherapeutic cover.

7. **After-cataract** :—This in its commonest form is the remnants of the capsule of the lens. It has to be expected to a varying extent in all cases of extracapsular extractions In 18 cases the capsule was considered to be thick enough to impair sight. In one case it completely covered the pupil and caused the sight to be very poor. (see table 5).

Table 5

| Complication | Number |
|---|---|
| Hyphaema | 24 |
| Iris prolapse | 8 |
| Shallow AC | 9 |
| Vitreous loss | 8 |
| Iridocyclitis | 4 |
| Infection | 3 |

8. **Chorodial detachment** :—seen in 6 cases.

9. **Delayed wound healing** :—seen in 2 cases.

10. **Mental changes** are seen in 4 patients.

## SYSTEMIC DISEASES

These were present when the patients were admitted to my ward. The VDRL was positive in 5 cases. They were all given PAM by the VD Specialist of this Hospital. All 5 had good sight when discharged. One patient had to be in Hospital for 46 days on account of persistent chemosis and lid swelling, the cause of which may have been an infection of the nasal sinuses. He left with a vision of 6/6. The second patient had to remain in ward for 39 days, but he had a vision of 6/9, post-operative. The 3rd patients had slight bleeding into the anterior chamber. None had uveal reactions following extraction.

Diabetes mellitus.—Six patients were found to be diabetic and had to be treated prior to cataract extraction. All 6 patients had good vision after the operation. Four of them had vision of the order of 6/9 to 6/6. Two of them had a vision of 6/18 each. One of them had to stay in ward for 37 days, owing to delayed wound healing. Two patients had a slight hyphaema though in both patients no iridectomy was done. The diabetics who had had an iridectomy did not bleed into the anterior chamber.

Pulmonary Tuberculosis.—Two patients in this series gave a history of having been treated for pulmonary tuberculosis. In one patient the operation was done well but he had no sight. There was a large mass in the fundus. The other patient had a prolonged hyphaema. The membrane that was left after the blood had been absorbed was needled. The bleeding recurred. The patient had no sight. These two patients were not diabetic.

CO-EXISTENT OCULAR DISEASES.—Secondary glaucoma was met with in 8 cases and had to be treated prior to extraction of the cataract. Four of the 8 cases had no sight after extraction. Two had optic atrophy owing to CRV thrombosis. Subluxation and anterior dislocation of the lens was present in 6 of the Jaffna cases. All 6 of these patients had fair to good vision after operation. In two cases the lens was removed by a vectis.

VISUAL RESULTS.—A visual acuity of 3/60 or less is taken as blindness. Vision of 6/60 and above is to be regarded as being fair to good. In this series 88% had a vision of 6/60 or more. A vision of 3/60 or less was recorded in 10% of patients.

A refraction was not done in most of the cases. The testing of vision in illiterate patients is not an easy task. They do not co-operate or make an effort to read. In

all cases the vision had been tested long before the eye had settled down to normality. This takes 6 weeks. (see table 6).

Table 6

| Vision | Number of cases |
|---|---|
| Less than ?/60 and 3/60 | 23 |
| 4/60 to 6/60 | 27 |
| 6/36 to 6/18 | 85 |
| 6/12 to 6/6 | 85 |
| Total | 220 |

STRENGTH OF CORRECTING GLASSES.—A correction of + 10 dioptres spherical was required by 78 (38% of those receiving glasses). A correction of + 12 dioptres spherical was required by 45 patients. (21.9%). A correction of + 11 dioptres S was taken by 41 patients (20%). A correction of + 10 DS/+ 2DC at 180 degrees was taken by 15 patients (7.3%). A correction of + 13 dioptres S was taken by 8 patients (3.9%). Other non-Standard corrections was taken by 18 patients (8.8%). A total of 205 glasses were prescribed. The 15 remaining patients had no need for glasses at the time of discharge.

CAUSES FOR NON-RESTORATION OF SIGHT.—These may be divided into those that were present and suspected to be present before the patient was taken up for cataract extraction and, those that developed subsequent to the operation. Table 7

Table 7

Causes of non-restoration of sight

| Cause | Number |
|---|---|
| Amblyopia | 2 |
| Optic atrophy | 2 |
| Macular degeneration | 1 |
| CRV thrombosis | 2 |
| Uveitis | 1 |
| Choroidal mass? TB | 1 |
| Total | 9 |

gives the causes falling into the first group. Table 8 gives the causes falling into the second group. Notice that a vision of 3/60 or less is taken as blindness. There were

Table 8

Causes of non-restoration of sight

(Developed after the operation)

| Cause | Number |
|---|---|
| Iridocyclitis | 2 |
| Vitreous floaters | 2 |
| Updrawn pupil | 2 |
| Hyphaema | 1 |
| Vitreous haemorrhage | 1 |
| Infection | 1 |
| Capsule | 1 |
| Not known (vision not recorded) | 4 |
| Total | 14 |

9 in the first group and 14 in the second group. In 4 of the second group vision had not been tested. At least 3 of the 14 in this group would improve with a second operation. Three more may be expected to improve with the passage of time. Four in this group had no chance at all of restoration of sight even after further surgery.

ACKNOWLEDGEMENT.

I wish to thank Dr. Mrs. Viswaratnam, Dr. Rupasingham, Dr. Mrs. Anandarajah, Dr. Miss. Rasathungham and Dr. K. Puvanendran who helped me in this work and in maintaining records. Thanks are due to the Department of Pathology for the culture and ABS reports. I must also thank the nursing staff of both eye wards for the patient and efficient care of my patients.

# CASE REPORTS

## REPORT OF TWO CASES OF MORBIDLY ADHERENT PLACENTAE

*By*

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MORBIDLY adherent placenta is supposed to be an extremely rare condition, its frequency being variously estimated at between 1 in 6,000 and 1 in 40,000 deliveries. Two such cases which occured within the space of two months in the maternity wards of the General Hospital, Jaffna are recorded.

CASE I

Mrs. K. P. 35 years old, gravida 4 para 3, was admitted on 19-5-66 at 12 midnight, in the 38th week of pregnancy with a complaint of generalised abdominal pain of 6 hours duration.

**Obstetric history:** The first pregnancy ended in a forceps delivery, under general anaesthesia-birth weight 6 lbs. The second pregnancy also ended in a forceps delivery but was a still-birth. She had severe postpartum haemorrhage due to the placenta being retained. It was removed manually under general anaesthesia and she was transfused with three pints of blood. The third pregnancy ended in a lower segment Caesarian Section for second degree posterior placenta praevia.—birth weight 7 lbs.

**History of complaint:** The onset of the pain was insidious. The pain was not colicky in nature and she was sure that she was not having labour pains. She complained of dysuria.

**On examination:** There was no oedema of feet and no albumin in the urine. Her pulse was 72/minute, and the blood pressure 120/80mm. of Hg. The heart and lungs were clinically normal. The abdomen was soft and no definite tenderness could be elicited. The height of the fundus of the uterus corresponded to that of a 38 week pregnancy. There were no palpable contra tions and the foetal heart rate was 144/minu and regular. There was no tenderness ov the lower segment of the uterus. She w kept under observation.

**Management:** The pain gradually creased in severity until 12 hours later it w much worse. There was marked pallor the tongue. The pulse rate gradually creased to 120/min and the blood press dropped to 90/60mm of Hg. She aga described the pain as being different fr labour pains. There was no evidence commencement of labour. The abdom was tender all over and there was generalis guarding. The foetal heart rate was s 144/min. regular and heard very clearly.

**Vaginal Examination:** The cervix not taken up and the os was closed. T head was at the pelvic brim. The pelvis clinically adequate.

An immediate laparotomy was deci upon, in view of the signs of intraperito haemorrhage. The cause of the haemorrh was not known, and a provisional diagn of silent rupture of the former Caesar Section scar was made.

Laparotomy was carried out under gene anaesthesia (induced with pentothal, m tained with gas oxygen and suxamethoni at 12-30 p. m. on 20-5-66.

A right paramedian incision was ma The peritoneal cavity was found to be of about 2—3 pints of dark coloured blo The scar on the lower segment was found be intact. The uterus appeared to be norm A lower segment Caesarean was done an live child was extracted. Attempts

deliver the placenta failed as it was morbidly adherent. Subtotal hysterectomy was performed.

There was a haemorrhagic area of about 1" in diameter on the peritoneal surface of the anterior aspect of the fundus, through which part of the placenta had eroded. A careful inspection of the rest of the viscera showed no other source of haemorrhage. The patient had post operative abdominal distension which responded to gastric suction and intravenous fluids. She was discharged three weeks later.

**Histology**: Section shows chorionic villi lying directly in contact with muscle tissue with no decidual cells. The chorionic villi are seen to go between muscle fibres. The appearance is compatible with that of a placenta increta.

Fig. No. 1. Shows outer aspect of uterus with pointer indicating the site of erosion of the placental villi.

Fig. No. 2. Shows inner aspect of uterus with morbidly adherent Placenta.

CASE II

Mrs. K. N. aged 24 years was transfered to our wards from a rural hospital at 10-45 a.m. on 8-4-66 with a history of having retained the placenta.

This was her second pregnancy which had ended in a stillbirth 5 hours earlier at the 38th week of gestation. The foetus was macerated. Controlled cord traction had been attempted but the cord had snapped. There was no post partum haemorrhage.

**Obstetric history**: The first pregnancy resulted in a premature delivery at the 7th month. The placenta was retained and had to be manually removed under general anaesthesia. The baby weighed 3 lbs. and died on the 7th day.

**On examination**: The patient was conscious and rational. The conjunctivae and tongue were extremely pale. The temperature was 99.4 deg. F. The pulse rate was 128/min. and the B.P. 80/40 mm. of Hg. There was no evidence of pre-eclamptic toxaemia. The heart and lungs were clinically normal. The abdomen was soft and the fundus of the uterus was at the level of the umbilicus. There was no bleeding per vaginam.

**Management**: It was decided to remove the placenta manually as soon as resuscitation of the patient was carried out. A pint of 4% dextrose was given as an intravenous drop and followed up with a pint of group AB Rh positive blood. 100 mg. of hydrocortisone was given intravenously on admission.

Manual removal of the placenta was attempted under general anaesthesia. (Nitrous Oxide, Oxygen and Cyclopropane) at 11-30 a. m. There was no plane of cleavage and the placenta was found to be morbidly adherent. Attempts at manual removal resulted only in piecemeal removal of ragged portions, protracted efforts at manual removal were desisted and hysterectomy was considered. The patient refused to give her

consent and pleaded that her uterus be conserved.

It was therefore decided to observe her carefully in the ward and carry out a hysterectomy in the event of any deterioration in her condition taking place. Her pulse rate was 104/min. and the blood pressure 100/60 of Hg. She had a rigor which lasted for two minutes. She was given 1 bulb of antistine I.V. She was given "Reverine" 275 mg. intravenously for three days followed by Tetracycline 250 mg. orally six hourly for five days.

Her convalescence was afebrile. There was very slight bleeding per vaginam on the 10th to 14 days, which did not cause alarm as it was very small in quantity. She was kept in the ward for 3 weeks and her anaemia treated with daily injections of Imferon for 10 days.

The uterus involuted slowly, being 2" below the umbilicus at the end of the 1st week, 4" below at end of the second week, and 1" above the symphysis pubis at the end of the 3rd week. She attended the follow up clinic at weekly intervals for a month, at the end of which the uterus had involuted completely. The lochia was more profuse than normal and remained tinged with red for 3 weeks. It was not offensive.

**Comments:** Morbidly adherent placenta is a condition resulting from an entire or almost entire absence of the decidua basalis, in consequence of which the uterine muscle is exposed to the invasion of the trophoblast and to penetration by the chorionic villi. The union between the placenta and the uterine wall is very intimate and it thus differs radically from simple adhesions of the placenta.

Its aetiology is unknown. It is a condition that recurs in the same patient often enough to presuppose the existence of some inborn or acquired factor. It is generally noticed to follow previous manual removal (as in the two cases reported), Caesarean Section or curettage.

Three varieties are recognised of this rare condition. Fully formed chorionic villi can be found in direct contact with the muscle bundles of the uterine walls (placenta Accreta), or are present between the muscle bundles (placenta increta), or may even penetrate the uterine muscle completely (placenta percreta). Case 1 was one of placenta percreta, and presented with signs of intraperitoneal haemorrhage.

Case 2 presented in the usual way but the management was unusual. The safest treatment in these cases is to do a hysterectomy without any greater delay than may be required for resuscitation of the patient. To leave the placenta in-situ as was done in this case carries a great risk. There is every possibility of infection of the placenta. While such infection will usually be saprophytic, the possibility of invasion of the necrotic tissue by Clostridium Welchii always remains uneasily in mind, and the humoral products of long retained placental tissue are suspect in regard to the production of hypofibrinogenaemia. Nevertheless this step may be justified in a patient for whom it is necessary to preserve the child bearing function. Numerous cases have been recorded where a placenta accreta has been left in-situ with subsequent survival of the patient and complete autolysis of the placenta. In some such cases successful pregnancy has occured (Gemmell 1947: Muir 1948; Mc Keogh and D'Errico 1951).


### Acknowledgment

I wish to thank Dr. Gnanapragasam, M.R.C.O.G. for his valuable advice. Dr. Gabriel, F. R. C. S. for the photographs and Dr. Rajapillai, M. R. C. P. for report on the specimen.


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# RETROPERITONEAL LIPOMA CAUSING TRANSVERSE DISPLACEMENT OF ONE KIDNEY

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RETROPERITONEAL lipoma is not uncommon. In the case reported here one such tumour had displaced the right kidney across the midline, so that it lay in front of the left kidney.

## CASE HISTORY

A. C. a 46 years old house wife attended the gynaecological clinic on 27th May, 1966, complaining of a lump in the right side of the abdomen, of five months duration. She had discomfort in the right side of the abdomen, and burning sensation on micturition.

On examination she looked slightly anaemic, and appeared to have lost weight recently. The large lump distending the right side of the abdomen was plainly visible. It extended from the costal margin to the right iliac fossa, and medially, extended across the midline. The lump appeared cystic and was ballottable.

A P.A. X-Ray of the abdomen showed a large soft tissue shadow pushing the right half of the colon downwards and to the left. An intravenous pyelogram (Figs I & II) showed the right kidney displaced across the vertebral column to the left, overlapping the lower half of the left kidney. There was a moderate degree of hydronephrosis and hydroureter on the right, probably due to pressure on the ureter from behind, by the vertebral column. There was no distortion of the pelvicalyceal pattern.

Fig. I.

Fig. II.

The patient was referred to us, and a diagnosis of retroperitoneal cyst pushing the right kidney over to the left was made.

Laparotomy was performed on 20th June 1966, by a transverse incision starting at the anterior end of the right 12th rib and extending across the midline to the lateral border of the left rectus abdominis muscle. The lump occupied the right side of abdomen with all the bowel pushed to the left. Superiorly the lump was in contact with the inferior surface of the liver with the fundus of the gall bladder just visible between the two. On the left the lump overlapped the right kidney which in turn rested on the lower half of the anterior surface of the left kidney. The peritoneum was incised on the right of the ascending colon. The lump was separated from the right kidney by blunt dissection, after which the lump shelled out without difficulty and was removed. The right kidney fell back into the right paravertebral gutter, but remained low with its superior pole just below the inferior surface of the liver. The incision on the posterior parietal peritoneum was closed and the retroperitoneal space was drained. Post-operative recovery was uneventful and the patient remains asymptomatic.

## PATHOLOGY

The tumour was oval in shape and measured 15" vertically, 9" from side to side, and 6" from before backwards. The cut surface was greenish yellow in colour and showed loculation. Histological section showed that the tumour was a lipoma with a mixture of myxomatous tissue.

## DISCUSSION

Lipoma is the commonest retroperitoneal tumour, and in some long series account for some 60 per cent of all retroperitoneal tumours. (Aird 1957). Aird also says that the majority start in the perirenal fat or may sometimes start in the kidney itself. In the present case the tumour probably started developing in close proximity to the lateral aspect of the kidney, and therefore pushed the kidney over to left as it grew bigger.

Crossed dystopia is a congenital abnormality in which the misplaced kidney lies below the normal kidney and is fixed. In the present case the crossed kidney slid back to its own side on removing the offending tumour. One was led to make a diagnosis of a S-shaped kidney on the pyelographic appearances but the presence of the tumour made this unlikely.

## SUMMARY

A case of retroperitoneal lipoma displacing the right kidney across the midline is presented. Though congenital misplacement of both kidneys on the same side is commonly reported, displacement by tumour to give the same pyelographic appearance is rare.

## ACKNOWLEDGEMENTS

I should like to thank Dr. R. Ramalingam, Resident Obstetrician for referring this patient to us, and helping us with the investigation and treatment of this patient: and Dr. A. Gabriel Visiting Surgeon for his help with the photographs.

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# MALIGNANT PLEURAL EFFUSIONS

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PLEURAL effusion or collection of fluid in the pleural cavity is a very common clinical entity.

Most cases are post pneumonic or tuberculous.

Post-pneumonic effusions are the sequelae of pneumonia and are usually thick, greenish or yellowish and bacteriological investigation readily reveals the causative organisms. Tuberculous effusions can occur without concomitant lung disease and have the characteristics of being clear, straw coloured form spider-web clots and do not readily reveal the Tubercle bacillus. Both culture and guinea-pig innoculation are arduous and time consuming. It is also customary to look upon most pleural effusions as Tuberculous, so that one hardly pays much attention to malignant pleural effusions.

Contrary to popular belief, malignant pleural effusions are by no means uncommon. The commonest cause of such an effusion is Bronchial Carcinoma. The growth can cause an effusion in one of two ways viz. (1) By direct infiltration of the pleura (2) The growth can occlude a bronchus leading to collapse of an pulmonary segment; this becomes the seat of infection which spreads to the everlying pleura and causes a pleural effusion. Although a rare entity has been described called a pleural Endothelioma, recent studies have shown that this is an undifferentiated Carcinoma of the Lung. Malignant effusions are usually blood stained and readily re-accumulate after aspiration.

The clinical behaviour of malignant pleural effusions can be illustrated by the following case-reports of patients who came under our care during the year 1965.

**Patient I** A. K. Male 27 years.

Presented with a history of cough, dyspnoea on exertion and hoarseness of voice of 8 months duration. Smoked about 30 "beedies" a day. On examination, he had a right pleural effusion; thick blood stained fluid was aspirated. Histology showed red blood cells and endothelial cells but no malignant cells. As subsequent chest Xrays showed reaccumulation of fluid, in spite of repeated aspirations, an exploratory thoracotomy was performed whence a large, cystic and very vascular tumour was seen, and biopsy of which revealed spindle shaped cells with large hyperchromatic nuclei and with large numbers of mitotic figures—"Undifferentiated Carcinoma of the lung" Patient was sent to the local hospital.

**Patient II** Nith. Male 36 years.

Presented with pain in the right chest of two weeks duration. Cough and dyspnoea on exertion of two months duration. He was a non-smoker.

On examination, he had a right pleural effusion and a hard, palpable Scalene node on the right side of the neck. Aspiration revealed thick bloodstained fluid which on histology showed red blood cells and malignant cells. Scalene node biopsy was performed and this showed secondary deposits from a bronchial carcinoma. A few days after admission, the patient developed ecchymoses and intense flushing of the skin and died soon afterwards. Unfortunately, the relatives refused a post-mortem examination.

**Patient III.** Aru. Male 39 years.

Presented with sudden central chest pain and dyspnoea. Queried by the admitting

officer as a case of myocardial infarction. He was a non-smoker. On examination, heart was clinically normal except for left sided displacement; he had right sided pleural effusion which on aspiration revealed thick bloodstained fluid which on histology showed red blood cells only. As this report was not helpful, a pleural biopsy was performed and this showed large polyhedral cells with hyperchromatic nuclei.—"malignant endothelioma of the pleura". As the patient was becoming more and more dyspnoeic, an emergency thoracotomy was performed whence the pleura was found to be grossly thickened with complete collapse of the right lung. There was a hard fleshy growth in the Right lower lobe bronchus infiltrating the pleura and part of the pericardium. A pleuro-pneumonectomy and pericardiectomy was done. Biopsy of the specimen revealed large hyperchromatic nuclei with distinct nucleoli and a large number of mitotic figures, "undifferentiated carcinoma of the lung". This patient is recovering at the time of writing this article.

## DISCUSSION

From the foregoing illustrations, it seems reasonable to presume that bronchial carcinoma is the commonest cause of malignant pleural effusions and that the causative condition namely, lung cancer is not so uncommon in Ceylon as previously supposed. Our patients presented for the first time with pleural effusions so that this could be looked upon as a common mode of presentation in lung cancer. Our Patient II, who developed flushing of the skin prior to death, exhibited the "Carcinoid Syndrome" which is said to be due to the production of 5-hydroxy tryptophan by the tumour. (PRACTITIONER, Dec. 1965)

In the light of these findings, it is mandatory to investigate and ascertain beyond doubt, the aetiology of every pleural effusion, as lung cancer seems to be on the increase even in our country, inspite of the absence of those predisposing factors prevalent in the Western countries.

ACKNOWLEDGEMENT. I wish to thank Dr. S. J. Stephen, MS FRCS for permission to publish details of his patients' case histories.

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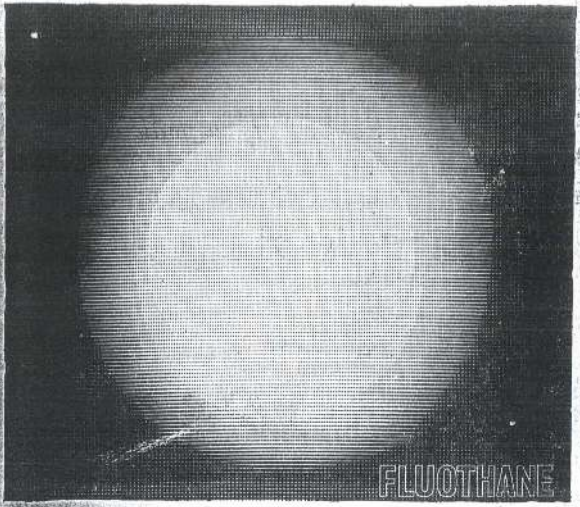
We lost the services of our energetic secretary, Dr. A. D. Mariadason who left us at the end of May, to take up a new appointment as Visiting Physician, Kuliyapitiya. We wish him all the best in his new post. Three well attended clinical demonstrations were held, since the last issue of the journal.

**K. Thuraisingham,**
*Secretary.*

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