weeks and underwent maceration. The foetus papyraceus and the macerated, older foetus were both delivered at term with the viable third child weighing 5\frac{1}{2} lb.

Mills (1949) reports eight cases of intra-uterine death of one twin. In two cases, where death was early on in pregnancy, a foetus papyraceus was formed. In the others, death occurred in the later weeks of pregnancy, the foetuses were macerated and some liquor amnii was found in the sac. The only one of these cases to show placental infarction had a velamentous insertion of the cord.

He further maintains that compression by the sac or the head of the surviving twin causes dehydration with the arrest of maceration and the formation of a foetus papyraceus. Compression may well be a necessary factor, as similar changes in the dead foetus of a single pregnancy have not been reported.

Crosman (1936) in a study of the dissolution and absorption of retained dead foetuses in rats, noted that under-developed foetuses were better preserved than more mature foetuses retained in utero for the same period of time. He contended that as a foetal life progresses enzyme activity becomes more progressive and that in retarded foetuses the reverse obtains. This may be of importance as retarded foetal development may be a precipitating factor, leading to the death and dehydration of the one foetus of a twin pregnancy.

Thompson (1927) draws attention to the fact that unless the membranes are intact, the usual process of putrefaction takes place. With intact membranes the dead foetus is in a sterile medium and so, too, are its own respiratory and alimentary tracts, from which areas putrefaction usually occurs. She quotes in evidence the fact that if the limbs are removed from a cadaver at the time of death, putrefaction is very much slower in them as compared with that taking place in the trunk.

Kindred (1944) suggests that the dead foetus is surrounded by a fluid of maternal origin which replaces the liquor amnii and preserves the foetus in a desiccated and embalmed state.

CONCLUSIONS
1. The cause of intra-uterine death of one foetus in a multiple pregnancy and its preservation in utero is controversial.
2. Pressure by the viable twin is a probable factor.
3. The protective value of the intact membranes is shown by the lack of any effect on the health of the mother and surviving child.

SUMMARY
1. Two cases of foetus papyraceus are reported.
2. A brief review of the literature is given.
3. The possible clinical complications due to this condition are mentioned.

I wish to thank Prof. James T. Louw, of the Department of Obstetrics and Gynaecology, University of Cape Town, for his advice and interest in the preparation of this paper.

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DISSEMINATED SCLEROSIS IN AN INDIAN MALE


King Edward VIII Hospital, Durban

This case of disseminated sclerosis occurred in an Indian male who has spent his whole life in Natal. Disseminated sclerosis is extremely rare in South Africa,1 though a case in a non-European female was described in Johannesburg in 1947.2 In other parts of Africa the disease is apparently also very rare, but two cases were reported from Kenya in 1946.3

As far as is known, this is the first case to be described in an Indian who has never left the borders of South Africa.

Khanda Samy, a male Indian, age 51 years, married and with four children, was admitted to King Edward VIII Hospital on 19 July 1951. He was born in Natal and has lived there all his life. He had formerly been employed ironing clothes in a laundry.

History. The patient's main complaint was inability to walk. Twenty-one years ago he developed pain in the hips and weakness in the left arm and leg, and 10 years later weakness of the right arm and leg. This weakness has been static and has not regressed. He denied at any time having suffered from blurred or double vision. There was no history of vertigo or paraesthesia and the patient had no complaints referable to the urinary or gastro-intestinal systems.

His mother is alive and well. His father died of 'fever' at the age of 70. His one brother and two sisters are alive.
and well. Five brothers have died but the cause of death is not known. The patient denied that any member of his family has or has had similar symptoms to his own.

Clinical Examination. Mentally, the patient was reasonably intelligent. He was of a contented, happy nature, but did not show euphoria.

The physical examination revealed nothing abnormal except in the central nervous system.

Cranial Nerves. The pupils were regular and equal and reacted to light and accommodation. On ophthalmoscopy the discs were normal. Vision was 6/6 in both eyes. The fields of vision were full to hand testing. There was no weakness of eye movements. There was marked nystagmus on horizontal deviation of the eyes, equal to both sides, but no nystagmus on vertical deviation. Marked dysarthria was present, speech being staccato in nature. No other abnormalities of the cranial nerves were found.

Motor System. Tremor of the head was present. There was marked weakness of the left upper and lower limbs, with marked increase of tone and left ankle clonus. The right upper limb showed some increase of tone and some weakness. The right lower limb showed slight weakness, but no appreciable increase of tone. There was no wasting and no fasciculation. Reflexes were as follows:

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Sensory System. All four limbs showed gross ataxia with marked intention tremor. Adiadochokinesia was present in the upper limbs, which also exhibited the rebound phenomenon. The arms fell away in a very short period of time on being held out while the eyes were shut. Ataxia of the lower limbs was so gross that even when the patient was supported by two assistants he could only take a few groping steps with difficulty.

On testing finger and toe movements, the sense of position was found to be normal in all four limbs. There was, however, some diminution of appreciation of the sense of vibration in both upper limbs and in the right leg. There was no loss of sensation to pin prick or cotton wool.

Lumbar Puncture. The fluid was clear and under a pressure of 120 mm. water. The pressure rose rapidly to 180 mm. water when the jugular veins were compressed.

Laboratory and Radiological Findings

Cerebrospinal Fluid: Protein 40 mg. per 100 c.c. Globulin, no excess. Sugar 85 mg. per 100 c.c. Chloride 700 mg. per 100 c.c.

The Lange colloidal gold test was not done in the absence of excess globulin.

Cells: 18 erythrocytes per c.mm.

Organisms: B. subtilis only.

Wassermann reaction: negative.

Blood count: Haemoglobin, 109% (15.25 gm.%). White blood cells: 8,400 per c.mm. Polynuclears: 52%. Lymphocytes: 41%. Monocytes: 4%. Eosinophils: 3%.

Blood sedimentation rate: 8 mm. per hour. Blood Wassermann reaction: Negative.

Urine: Chemical and microscopic examination revealed no abnormality.

Chest X-ray: Nothing abnormal detected.

Skull X-ray: There was a calcified pineal gland, which was located in a normal position. No evidence of intracranial pathology was demonstrated. Lines drawn through the hard palate and the plane of the atlas vertebra were parallel, thus excluding platybasia.

Discussion

It is felt that this is an undoubted case of disseminated sclerosis. Other diseases considered, only to be dismissed, were:

1. Neuro-Syphilis. The normal cerebrospinal fluid findings, the negative blood Wassermann reaction, the absence of Argyll-Robertson pupils, the lack of dementia and the long duration of the disease were amongst the features against the diagnosis of tabo-paresis.

2a. Friedreich's Ataxia. The onset at 30, the increased tendon jerks and lack of family history precluded this diagnosis.

2b. Other Familial and Hereditary Ataxias. Most cases in this group are not associated with upper motor neurone lesions. Ferguson and Critchley have described a form of hereditary ataxia resembling disseminated sclerosis, in which there were signs of bilateral pyramidal degeneration and cerebellar inco-ordination in the limbs with dysarthria and an ataxic gait. However, their cases were familial and associated with external ophthalmoplegia. Also there was usually some anaesthesia and analgesia present.

3. Subacute Combined Degeneration. The blood count revealed no anaemia. The age incidence was too early; there was no glossitis, no palpable spleen and there was no glove and stocking anaesthesia.

4. Cervical Cord Tumour. A lesion of the cervical cord might cause nystagmus and spastic ataxia, but would not cause dysarthria. There was only minor involvement of the posterior columns in this case with the ataxia mainly cerebellar in origin. Laboratory investigation revealed no abnormality of the cerebrospinal fluid. Queckenstedt's test gave a normal response. There was no involvement of the fifth cranial nerve to suggest an upper cervical lesion and no wasting in the arms or Horner's syndrome to suggest a lower cervical lesion.

Certain 'classical' features were absent in this case, viz. a history of remissions, bladder symptoms, temporal atrophy, a history of diplopia or blurred vision, euphoria and dementia; but then no case of disseminated sclerosis shows all the wide variation of common features.

In a special article in 1947, the National Multiple Sclerosis Society of New York presented a table of symptoms and signs in order of frequency of occurrence and this case showed the six most common features, namely:

1. Abdominal wall reflexes decidedly diminished or absent.

2. Chief complaint weakness and stiffness of one or both lower extremities.
3. Nystagmus (chiefly horizontal).
4. Babinski’s sign present bilaterally.
5. Tremor present in the extremities (tremor present also in the head is not such a common feature).
6. Ataxia and adiadochokinesis.

It is interesting to speculate on the rarity of disseminated sclerosis in Southern Africa. In an address to the Southern Medical Association of America, Dr. Shields, a neurologist of Richmond, Virginia, had many pertinent observations to offer. He drew a parallel between the geographic distribution of the disease and the farm practices carried out in various areas where the disease is prevalent. He pointed out that there is a maximum incidence of the disease in countries such as Germany, England, United States of America and the states of Northern Europe, and that it is in these countries that large amounts of inorganic fertilizers are used. He maintained that these fertilizers cannot adequately replace all the requirements of the soil after intensive cropping and, in fact, upset the chemical balance of the soil. This results in the production of food with deficiency of elements such as cobalt, manganese and iron. This may result in diseases to animals and man. Apparently in New Zealand a soil deficiency of cobalt in the pasture grasses resulted in sheep becoming paralysed. The application of cobalt to the pasture prevented the paralysis from occurring. (In this connexion it is also interesting to observe the suggested association between copper deficiency in the soil and sway-back in sheep.) He further stated that cases were first described in England and France when the soil was being depleted. He contrasted this state of affairs with the absence of disseminated sclerosis in China, Japan and India, where natural fertilizers or manures are used.

If this theory be the correct one, one wonders how long it will be before disseminated sclerosis becomes a fairly common disease in South Africa, where soil erosion is so widespread and where the feeding of a steadily increasing population will lead to the wider use of inorganic chemical fertilizers.

My thanks are due to Dr. J. L. Parker, Medical Superintendent, King Edward VIII Hospital, for permission to publish this case and to Dr. N. A. Rossiter, under whose care this case was admitted. I would also like to make acknowledgment to the Radiological Department, King Edward VIII Hospital, and to the Provincial Laboratory for X-rays and laboratory investigations.

REFERENCES

VENOUS MESENTERIC THROMBOSIS
REPORT ON A CASE WITH RECOVERY
E. KRUGER, M.B. CH.B.

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This case is presented in the hope of contributing something of value to the literature of vascular accidents. My purpose is to review briefly the subject of mesenteric thrombosis and to present a case which recovered.

Mesenteric vascular occlusion is often unrecognized before laparotomy or autopsy. It has been pointed out that mesenteric thrombosis occurs more commonly than is often appreciated but in a milder form; this accounts for many vague abdominal symptoms in older people without there being necessarily a massive abdominal accident, e.g. where the intestinal circulation is impaired gradually. There is no place for conservatism in the treatment of this condition when it presents itself as an acute abdominal emergency. Radical surgery is the only hope in saving life.

Etiology. There is a number of predisposing causes but the exciting cause is still unknown.

The predisposing causes can be divided into general and local.

General:—
1. McClenahan and Fisher reported 616 cases with 43 successful resections (or 7%) up to August 1947. Although the incidence of mesenteric thrombosis is greatest between the ages of 30 and 70 years, it may occur at any age, but is extremely rare in children. It is more common in males. The rest of the causes are either ascending or descending.

The ascending causes are:
2. Venous thrombosis is most frequently associated with infections in organs or viscera that are drained by tributaries to the portal vein. Thus appendicitis, pelvic disease or ulcerating carcinoma of the colon may lead to venous thrombosis.
3. Antecedent surgery—surgery of the stomach, gall bladder, appendix, strangulated hernia and pelvis are predisposing factors. Here there are no inflammatory lesions in evidence, but injury to the vessel wall is a factor.

Descending Causes:
4. Other conditions which have led to thrombosis of the mesenteric vein have been blood diseases such as splenic anæmia; also trauma to mesenteric vessels; mechanical causes such as portal stasis from tumours or adhesions.
5. Many cases reported in the literature confirm the idea that in the history of patients who have primary mesenteric thrombosis due to atheromatous change in the vessels of the mesentery, there often was some abdominal distress, perhaps after a heavy meal, after some exertion, or at other times when the blood supply to the gut may be impaired.
6. In some cases no primary cause of thrombosis can be demonstrated. Vickery suggests that in these cases the thrombi are bland in nature and that a partial volvulus with torsion of the mesentery could initiate the thrombotic process.