(intracavitary nitrogen mustard, thiotepa, etc.), but it still has a place where alternative methods fail, as they often do.

The comparative merits and demerits of the various preparations of yttrium have still to be determined and checked against clinical experience. In the colloidal silicate we appear to have a substance of promise, worthy of further investigation.

It is proposed to publish elsewhere full technical details of the above investigation, including physical data on dosage, post-mortem assays, activity in blood, urine, and cavitary fluid, etc.

Summary

As an alternative to radioactive gold for control of malignant pleural and peritoneal effusions, the use is described of a new compound, colloidal radioactive yttrium silicate ($^{90}Y_2(SiO_3)_3$). A preliminary report is made of its therapeutic use in 15 cases. Out of nine cases surviving over one month, six received good palliation for 5 to 19 months. The advantages of yttrium over gold are discussed, especially the superior surface penetration of the beta-particles and the lesser radiation hazards owing to absence of penetrating gamma-radiation.

Thanks are due to my physicist colleagues, on whom this work depended, in particular Mr. J. C. Jones (now at the Royal Marsden Hospital, London), and Mrs. Monica Fisher; to my colleague, Dr. Frank Neal, for helpful liaison; to Mrs. Ruth Martin for secretarial assistance; and to Miss D. Wemm for the Figure.

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"The aims of the Association of Social Workers and also present-day thought in social work show that there are elements common to all social work practice whatever its setting. To discern these common elements, recast them as a discipline and find effective ways of teaching them is a major challenge which merits special study and research. Evaluation studies of techniques in casework are needed and the criteria of success and failure in casework practice require to be explored. So far only small sporadic studies of this kind have been made in Britain. The probation field with its preponderance of unwilling clients would seem to offer a unique opportunity for comparative studies into the histories of such clients and for analysing the ways in which they can be enabled to draw help from the relationship with the worker and from the resources of the community. Such studies might well have a bearing on future social planning and could usefully have a wider reference to include analyses of existing training, work studies, and job evaluations in each of the social work specialties." (*Evidence Submitted to the Departmental Committee on the Probation Service*, Association of Social Workers; price 1s. 6d.)

DYSTROPHIA MYOTONICA IN CHILDHOOD

BY

T. M. VANIER, M.B., M.R.C.P.

Assistant Medical Registrar, Hospital for Sick Children, Great Ormond Street, London

The clinical development of dystrophia myotonica in adults is commonly the following: a family history of senile cataracts in an early generation, of presenile cataracts in the next, and finally the full picture of the disease in the third. The early development and evolution of the disease in infants and children, of whose parents one may show only certain features of dystrophia myotonica, is less familiar. Six such cases have been seen at the Hospital for Sick Children in the past five years. Only three cases of myotonia congenita are recorded over the same period, and yet this disease, while it, too, is rare, is reputedly of earlier onset than dystrophia myotonica. Though the two diseases present differently and have a widely different prognosis, there is no doubt that they are interrelated. There are records of patients with myotonia congenita diagnosed in childhood who many years later developed stigmata of dystrophia myotonica; also of families in which the two diseases have occurred in different individuals (Maas and Paterson, 1950; Walton and Nattrass, 1954).

Because dystrophia myotonica is not widely recognized as a cause either of flaccid paresis in infancy (amyotonia congenita syndrome of Oppenheim) or of congenital facial diplegia the following cases are reported.

Case Histories

The diagnosis was made while the child was under the care of the physician whose name appears in parentheses, and at the first age mentioned. When a second figure for age is given this indicates the length of follow-up. In no family is there parental consanguinity.

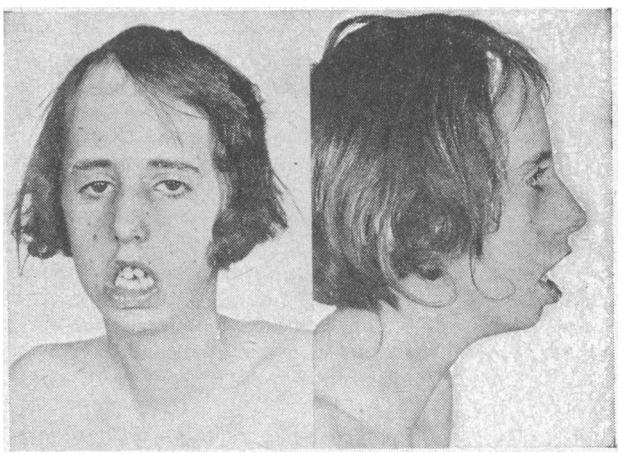

FIG. 1.—Case 1. Dystrophia myotonica in girl aged 13.

Case 1.—Girl (Dr. P. H. Sandifer) 13 years (Fig. 1). Breech delivery after normal pregnancy. Bilateral talipes noted shortly after birth. Difficulty with sucking. Floppy infant. Delayed motor and speech development. Assessed as ineducable. No history of deterioration in performance. On examination: mentally retarded. Complete bilateral facial paralysis, ptosis, wasted temporal muscles but strong

masseters, nasal speech, poor palate movement, string-like sternomastoids. Receding hairline. Myotonic grip and percussion reaction of thenar, pectoral, and deltoid muscles. Generalized feeble muscle power, especially in pelvic girdle, peronei, and tibialis anterior. Kyphoscoliosis. All tendon reflexes present and brisk except ankle jerks. Below third percentile in weight. Skull *x*-ray examination: small pituitary fossa, large frontal sinuses. Her mother, who denies any disability, has ptosis, myotonic grasp, and percussion reaction of thenar muscles. The eldest sibling, aged 20, is normal, but two older brothers are mentally retarded and have foot deformities. They have not been examined.

Case 2.—Boy (Dr. P. H. Sandifer) 11 to 13 years. Normal pregnancy and delivery. Difficulty with sucking. Floppy infant. Delayed motor and speech development; walked alone at 2 years. Attends school for educationally subnormal children. Recent history of deterioration in gait from progressive weakness of peroneal and anterior tibial muscles. Examination disclosed complete bilateral facial paralysis, weak masseters, wasted temporal muscles, string-like sternomastoid on right, absent on left. Myotonic grasp and thenar percussion reaction. Generalized muscle feebleness, most pronounced in upper arm, peroneal, and anterior tibial muscles. Foot-drop gait. All tendon reflexes brisk except absent left ankle-jerk. Dorsal scoliosis. Normal testicles. Weight at 13 years below third percentile. Skull *x*-ray examination: thickened calvarium and small pituitary fossa. Only sibling (Case 3) had dystrophia myotonica. Mother (who died from the effects of rheumatic heart disease) had ptosis, sternomastoid wasting, myotonic grip, and thenar percussion reaction without being aware of these abnormalities.

Case 3.—Girl (Dr. P. H. Sandifer) 4 to 8 years. Normal pregnancy, breech delivery. Bilateral talipes noted at birth. Normal sucking. Floppy infant. Retarded motor and speech development. Attends school for physically handicapped. No subjective or objective deterioration. Examination showed marked facial weakness, most obvious in the forehead, ptosis, wasted temporal muscles, string-like sternomastoids, nasal speech, poor palate movement. Receding hairline. Myotonic grasp and thenar percussion reaction. Generalized muscle feebleness, most marked peripherally. All tendon reflexes brisk except ankle-jerks. Fixed talipes equinovarus deformity. At 8 years below third percentile in weight and tenth in height. Skull *x*-ray examination: small pituitary fossa. Serum aldolase, wrist bone age, and E.C.G. normal. I.Q. 60 (all at the age of 8).

Case 4.—Boy (Dr. R. Lightwood) 6 to 10 years. Normal pregnancy and delivery. Normal sucking. A floppy infant, but was sitting at 9 months and walking at 15 months. Late speech development. Attends school for physically handicapped. No subjective or objective deterioration. On examination, facial weakness most pronounced in forehead, wasted temporal and sternomastoid muscles, nasal speech, and poor palate movement. Myotonic grip and percussion reaction of thenar muscles. Generalized feeble muscles, especially peripherally. Tendon reflexes absent in upper but brisk in lower limbs. Normal testicles. Weight between third and tenth percentiles at 6 and 10 years. Electromyogram (Dr. A. T. Richardson): right wrist flexors; evidence of myotonia. Skull *x*-ray film: normal (6 years); thickened vault, normal pituitary fossa (10 years). Bone age (10 years) normal. E.C.G. normal (6 and 9 years). I.Q. (6 years 6 months), 80–90 on revised Stanford Binet scale, 70 on Merrill Palmer scale. At 6 years 11 months, 85 on revised Stanford Binet scale. At 8 years 7 months, 78 on revised Stanford Binet scale. At 9 years 2 months, 73 on revised Stanford Binet scale. At 10 years 5 months, 70 on revised Stanford Binet scale. One normal sister aged 4. Mother severely affected, including cataracts.

Case 5.—Boy (Dr. P. R. Evans) 14 months to 3 years 10 months. Normal pregnancy but feeble intrauterine movements. Breech delivery. Difficulty with sucking. Bilateral talipes noted at birth. Floppy infant. Retarded motor and speech development. Continues to gain skills. On examination, bilateral facial paralysis, wasted temporal muscles, weak sternomastoids. Floppy infant with generalized muscle feebleness especially in peroneal and tibialis anterior muscles. No myotonic phenomena noted until 3 years old, when relaxation of calf muscles was noted to be slow. Leg tendon reflexes and supinator jerks brisk, biceps and triceps reflexes absent. Left testicle normal, right undescended. Normal height and weight throughout. Electromyogram (Dr. E. D. C. Campbell) normal. Serum aldolase and skull *x*-ray film normal. Urine creatine coefficient, 11.4 (raised). Urine creatinine coefficient, 13.2 (normal). Muscle biopsy of tibialis anterior, using Cöers and modified Koelle techniques (Dr. M. Bodian): sacrolemmal nuclei increased in number, and rows of these lined up on the periphery but not in the centre of muscle fibres. Nerve-endings normal. (All at 14 months.) Mother has minimal signs of dystrophia myotonica, but her brother is severely affected. Of two children of the mother's previous marriage, one is retarded in motor development and is said to have a myotonic grip.

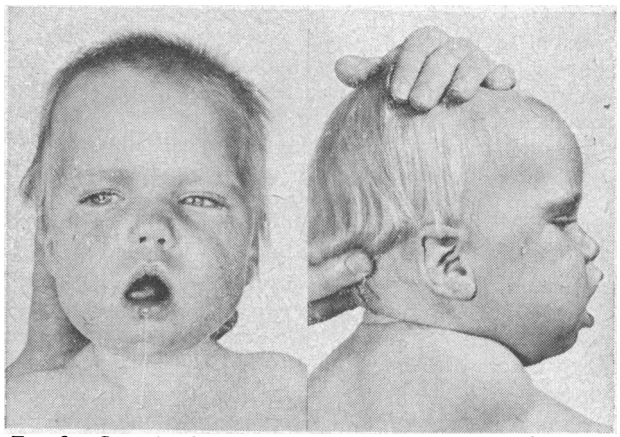

FIG. 2.—Case 6. Dystrophia myotonica in female infant aged 9 months.

Case 6.—Girl (Dr. P. R. Evans) 9 months (Fig. 2). Normal pregnancy and delivery. Difficulty with sucking. Floppy infant. Not lifting head or sitting up. On examination, poor facial movements, wasted temporal muscles, weak sternomastoids, poor tongue movements, high arched palate. Floppy infant; all reflexes present. No clinical evidence of myotonia. Electromyogram (Dr. E. D. C. Campbell): tibialis anterior and extensors of forearm; typical myotonic pattern with minimal dystrophic (myopathic) signs. Serum aldolase, 20.2 μmol. per 100 ml. per hour (slightly raised). Urine creatine coefficient, 9.5 (raised). Urine creatinine coefficient, 6.1 (low). Mother has myotonic grip, of which she is aware but which is not a cause of severe disability. Her temporal muscles are wasted. Her brother is severely affected and has cataracts. The maternal grandfather's sister had cataracts. The patient has an older normal male sibling.

Clinical Features of Dystrophia Myotonica in Childhood

Muscle-wasting.—As in adults, wasting of the temporal, facial, and sternomastoid muscles is an early and characteristic feature. All these children had obvious wasting of the temporal muscles. Four had facial paralysis at birth, causing difficulty with sucking. Two (Cases 3 and 4) had no such difficulty, but when first seen, at 4 and 6 years respectively, there was obvious lack of facial expression with paresis, most pronounced in the upper half of the face. The string-like sternomastoids complete the characteristic appearance: a long, swan-like neck, a long face tapering towards the jaw, with hollow temporal fossae. Ptosis

was noted in Cases 1 and 3. In three children palate movement was poor and resulted in nasal speech, as in some adult cases. In no case was myotonia or wasting obvious in the tongue, though one patient (Case 6), at 9 months, is noted to have poor movements of the tongue. The other muscles selected for atrophy have shown a variability at least as great as that reported in adults (Maas, 1937). In Case 1, for instance, the greatest weakness was in the pelvic girdle, whereas in Case 2 the distal leg muscles were most severely affected. All these children were limp and floppy infants (see below). It is noteworthy that three children were found to have bilateral talipes at birth. In only one (Case 5) has early correction given good results. He has, however, marked weakness of the peroneal and tibialis anterior muscles. Ford (1952) mentions the case of a woman whose history went back to infancy, during which she was noted to have an immobile face and club-feet.

Myotonia.—This was present in the four older children. The two children examined in infancy showed no evidence of myotonia clinically, though the electromyogram was characteristic in Case 6. One patient (Case 5) at the age of nearly 4 years has a suggestion of slow relaxation of the calf muscles. In none of the children was myotonia the cause of symptoms (unlike myotonia congenita). As in adults, the degree and distribution of myotonia bore no relationship to muscle weakness and wasting.

Tendon Reflexes.—These were all strikingly brisk in Case 6. In the other children knee-jerks were brisk but other reflexes varied. The ankle-jerks were absent when talipes deformity was marked.

Skeletal Abnormalities.—Various types have been reported in cases of dystrophia myotonica. Klein (1958) found a high arched palate in 40% of his cases. This was present in Case 6.

Bodily Dyscrasia.—A generally wasted appearance, premature baldness, cataracts, and testicular atrophy are features of the disease in adults. The three older children had this wasted appearance and were below the third percentile in weight, though only Case 3 was below the tenth percentile in height. Cataracts have not been detected, but slit-lamp examinations were carried out only in Case 6. Cases 1 and 3 have a receding hairline. The three male children have apparently normal testicles, except that in Case 5 one is undescended.

Speech.—In adults this may become slow, nasal, and indistinct from a combination of facial, palatal, and lingual weakness and/or myotonia. In the five older children speech has been delayed, and in three it is nasal in quality.

Mental Equipment.—Dystrophia myotonica is often associated with defective intelligence. It has been suggested that a severe form of the disease with oligophrenia is the extreme manifestation of the responsible gene. The evidence in favour of a dementing process, however, is less overwhelming. Maas and Paterson (1937) studied 29 cases and found 17 to be of low intelligence. In 11 the intellectual defect had been present since childhood and apparently before the obvious onset of the disease. The others seemed to show evidence of dementia. Those with severe muscle-wasting were the most seriously affected intellectually. Disorders of affect are said to occur, ranging from lack of energy and initiative to complete inertia. One wonders, however, in how many cases this impression is conveyed by oligophrenia combined with physical disability. Estimate of mental age is particularly difficult in young children affected with dystrophia myotonica because retarded speech and motor development can be attributed to physical handicap. Error may result from interpreting the lack of facial expression as evidence of mental retardation (Evans, 1955). Of the older children, one (Case 1) attends an occupation centre, one (Case 2) attends a school for the educationally subnormal, and (Case 3) had an I.Q. of 60 at 8 years. Only in Case 4 have repeated intelligence tests been carried out. He has been seen five times over the past four years by the same educational psychologist (Mr. L. Gardner). During this period there has been a steady fall in his verbal I.Q., so that betwen 6 and 10 years it has fallen by 15 points. It is not easy to interpret this apparent deterioration, but the number of tests carried out and the steady fall militate in favour of this being a true fall in I.Q.

Course and Prognosis

In adults the onset is usually insidious and deterioration slow. We know that sometimes an adult history goes back to infancy. The immobile face and sucking difficulty dating from birth seem to indicate that the degenerative process started *in utero*. In Case 6 there was marked wasting of the temporal muscles at 9 months; if, as seems likely, this was present to some degree at birth, then these muscles, too, started degenerating *in utero*. The same comments apply to the presence of talipes in these children. We know that on occasion in adults localized muscle-wasting can be remarkably rapid at the onset without the disease progressing further at the same speed (Klein, 1958, Case 53). None of our cases have been followed up long enough for an accurate assessment of the course in childhood. Only in Case 2 has clear evidence of deterioration been shown. The others, while being slow to develop skills, have nevertheless continued to improve in performance. The dubious mental deterioration in Case 4 is discussed above.

Family History and Mode of Inheritance

Dystrophia myotonica is inherited as if due to a dominant gene with variable manifestations. The disease is said to show "anticipation" in that the children are affected at an earlier age than are the parents and there is an increase in severity in succeeding generations. This is almost certainly due to the tendency for the more mildly affected individuals of one generation to transmit the gene to the next generation; on the whole the severely affected do not bear children.

It is noteworthy that the affected parent in all our cases is the mother and that with one exception she has minimal features of the disease. Maas (1937) has remarked on the greater number of women with a mild form of the disease, but Klein's (1958) figures suggest that transmission is commoner through a mildly affected father. In two of the families here reported, the mother's brother is severely affected. Caughey and Brown (1950) have pointed out that females do not exhibit the striking hypogonadism obvious in males with dystrophia myotonica.

The genetic problem is well reviewed by Thomasen (1948) and Klein (1958).

Ancillary Aids in Diagnosis

Electromyogram.—This is the only investigation likely to provide diagnostic proof if there is clinical doubt. It may of course, at some stage and in some muscles, be normal, as it was in Case 5 at 14 months. In Case 6 at 9 months, it provided conclusive proof of myotonia even though this was not evident clinically. Depending on the stage of the disease and the muscle tested, it may show at rest the grouped, repetitive high-frequency discharges and " myotonic dive " of myotonia or on volition the myopathic pattern of a muscular dystrophy. Both changes may of course appear in the same muscle.

Serum Aldolase.—This has been normal or slightly raised as in adults (Evans and Baker, 1957; Pearson, 1957).

Urine Creatine and Creatinine Coefficients.—As in other muscular dystrophies the creatine may be raised and creatinine reduced. This was the case in the two children on whom the test was carried out.

Skull X-ray Examination.—This was normal in two children at the ages of 14 months and 6 years respectively. The brother and sister (Cases 2 and 3) have small pituitary fossae, and Case 2 has some thickening of the calvarium; Case 1 has a small pituitary fossa and large frontal sinuses. At 6 years Case 4 had a normal skull *x*-ray picture, but by the age of 10 years this showed thickening of the calvarium. The changes noted in adults are: small pituitary fossa, thickening of the calvarium, internal hyperostosis of frontal and parietal bones, enlarged sinuses (Caughey, 1952).

E.C.G.—This was normal in the two children tested. The commonest abnormality reported in adult cases has been prolongation of the P–R interval and QRS complex (Fisch, 1951).

Muscle Biopsy.—This was carried out in one case only, but failed to show " diagnostic features " of dystrophia myotonica. These are thought to be enlargement of scattered muscle fibres and the presence of long rows of centrally placed sacrolemmal nuclei in otherwise normal muscle fibres. Other changes consistent with a muscular dystrophy may occur (Adams *et al.*, 1953).

Urinary Excretion of 17-Ketosteroids.—These have been reported as below normal in adults (Caughey and Brown, 1950). Estimations have not been carried out on our cases.

Discussion

Amyotonia Congenita Syndrome.—Early in life all these children presented as " floppy infants " backward in motor and later in speech skills. The possible causes of infantile flaccid paresis are numerous (Walton, 1957). Broadly speaking, those children who show steady progress are likely to be examples of benign congenital hypotonia. Much more rarely the very early onset and remission of polyneuritis or polymyositis may follow a similar course. As a rule one is justified, on seeing a limp and floppy child steadily acquiring new skills, in giving a good prognosis. Dystrophia myotonica is an exception to this generalization. All our cases have continued to acquire skills in early childhood, but, of course, they carry the eventual prognosis of the progressive disease. A history of difficulty with sucking, facial paresis, and possibly club-feet should make one search further for signs of dystrophia myotonica in the infant and his parents.

Möbius Syndrome or Congenital Facial Diplegia Syndrome.—Most cases are probably due to a congenital defect of muscle (Evans, 1955). Congenital facial diplegia has been described as occurring in combination with weakness of external ocular muscles, masseters, or muscles of the palate, pharynx, larynx, or tongue (Henderson, 1939; Evans, 1955). It has been personally observed, moreover, with sternomastoid weakness and wasting of the temporal muscles. Four cases of this syndrome, which illustrate certain similarities between it and dystrophia myotonica, are briefly reported in Appendix I. One patient (Case D) is of particular interest in this connexion: she had palatal and facial paresis dating from birth, while her father has the facies of dystrophia myotonica and a myotonic reaction on thenar percussion. This child is being closely watched in case she develops any signs of dystrophia myotonica herself. Children with facial diplegia do not usually show universal hypotonia, but in Appendix II three brief case reports are given which indicate that this may occur. They also illustrate the similarity with the early clinical picture of dystrophia myotonica in infants. The individual muscles mentioned are weak out of all proportion to the general muscular feebleness. Congenital talipes, which not uncommonly occurs in combination with Möbius syndrome, has been present in three of the six children with dystrophia myotonica.

Differential Diagnosis in Older Children.—These children are likely to be seen (often in orthopaedic clinics) because of residual talipes deformities, difficulty in walking, or postural defects. At this age many stigmata of dystrophia myotonica may be present. Peroneal muscular atrophy, myopathy, amyotonia congenita, and even a progressive bulbar palsy may all be considered unless the distinctive features of dystrophia myotonica are noted.

Summary

Six cases are reported of children with dystrophia myotonica whose symptoms date back to early infancy. The youngest in whom the diagnosis was made was aged 9 months.

These case reports illustrate that the onset of dystrophia myotonica may be earlier than is generally recognized, that dystrophia myotonica is occasionally responsible for the syndrome of amyotonia congenita (Oppenheim's syndrome); and that dystrophia myotonica is occasionally responsible for the presence of congenital facial diplegia.

Four case histories are briefly outlined in Appendix I because they illustrate certain similarities between dystrophia myotonica and Möbius syndrome).

Three case histories are briefly outlined in Appendix II because they illustrate similarities between dystrophia myotonica and facial diplegia occurring as one feature of benign congenital hypotonia.

The clinical features of dystrophia myotonica in childhood are outlined and the use of ancillary aids in diagnosis is mentioned.

APPENDIX I. CONGENITAL FACIAL DIPLEGIA ASSOCIATED WITH WEAKNESS OF CERTAIN OTHER MUSCLES

Case A.—Boy now aged 12. Facial diplegia, bilateral external rectus paresis, left sternomastoid atrophic and weak, poor palate movement, unilateral talipes. Not a floppy infant; milestones of development normal except speech.

Case B.—Boy now aged 11. Facial diplegia; weak masseters, temporal muscles, and sternomastoids; wasted tongue. Generalized poor muscular development, but not a floppy infant and milestones of development normal.

Case C.—Boy now aged 7. Facial diplegia, bilateral external rectus paralysis, poor palate movement, fibrillating tongue. Unilateral talipes, with wasting of muscles of anterior compartment of lower leg and some shortening of limb. Normal electromyogram and intensity duration curves of facial, lingual, and limb muscles. Not a floppy infant. Development delayed though he has shown steady improvement.

Case D.—Girl now aged 8. Facial diplegia, palatal paresis. Father has facies of dystrophia myotonica and myotonic reaction over thenar muscles.

APPENDIX II. CONGENITAL FACIAL DIPLEGIA WITH BENIGN CONGENITAL HYPOTONIA

Case E.—Girl now aged 5. Facial diplegia, poor palate movement, bilateral talipes, delayed milestones with universal hypotonia and absent reflexes.

Case F.—Boy now aged 5. Facial diplegia, poorly developed sternomastoids, absent clavicular heads of pectoralis major, delayed milestones with universal hypotonia and absent reflexes.

Case G.—Girl now aged 11. Facial diplegia, delayed milestones with universal hypotonia and sluggish reflexes.

I am grateful to the physicians of the Hospital for Sick Children, Great Ormond Street, for permission to publish details of cases under their care, and to the department of medical illustration for the clinical photographs. I would particularly like to thank Dr. P. H. Sandifer for much helpful criticism and advice.

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"A notable innovation, now a little over a year old, is the Flying Surgeon Service sponsored by the Queensland Government. This is designed to provide surgical facilities for 21 towns in Western Queensland. Regular monthly visits are paid to 16 of these, all of which have one or more resident doctors. Towns which are without a resident doctor are visited by the flying surgeon only if the doctor of the Flying Doctor Service requests assistance with a patient who needs a surgical opinion or operation and cannot be moved. The service is provided by a surgeon, an anaesthetist, and a pilot, all based on Longreach, which is approximately in the centre of the area. The hospitals in the scattered towns are equipped with routine surgical instruments and materials, but the flying surgeon brings with him whatever else is needed in the way of instruments and equipment. The anaesthetist carries a portable anaesthetic machine. . . . The service is essentially consultative, as patients are seen only at the request of another doctor. If the flying surgeon considers a surgical operation necessary he may recommend transfer of the patient to a larger and better-equipped centre, he may perform the operation with the resident doctor as assistant, or he may act as assistant while the resident doctor operates." (*Med. J. Aust.*, October 15, 1960.)

GASTRIC TUBERCULOSIS

BY

B. I. CHAZAN,* M.B., M.R.C.P.
Late Senior House Officer

AND

J. D. AITCHISON, M.B., F.R.F.P.S., M.R.C.P.
Consultant Physician
Western District Hospital, Glasgow

Tuberculosis of the stomach is a rare disease. Palmer (1950) has estimated from previously published material that 159 cases were found among 96,251 necropsies (0.16%) and 117 among 20,585 necropsies on subjects whose primary disease was tuberculosis (0.57%). He states further that 19 instances of tuberculosis were found among 17,542 specimens obtained from stomach operations (0.1%).

The diagnosis is difficult to establish clinically, and the condition may be confused with peptic ulcer and with neoplasm of the stomach. Accordingly, the following case seems worth recording.

Case Report

The patient, an Indian aged 45, was first seen in hospital in 1951, complaining of epigastric pain, lethargy, and loss of weight. He had come to Glasgow from the Punjab in 1948 and had worked as a manual labourer. His knowledge of English was limited. Abnormal findings on physical examination were epigastric tenderness and slight enlargement of cervical, axillary, epitrochlear, and inguinal lymph nodes. No abnormality was found clinically in the lungs, but radiographic examination showed a small area of opacity in the right upper lung field, thought to be due to primary tuberculous infiltration. Tubercle bacilli could not be found in the sputum. E.S.R. was 65 mm. in one hour (Westergren). Haemoglobin was at first 12.1 g./100 ml., but during the next two weeks a moderate degree of anaemia developed from loss of blood into the gastro-intestinal tract. Faecal occult blood tests were repeatedly positive. Neither ova nor cysts could be found in the stools. Blood W.R. was negative. Barium-meal examination revealed nothing abnormal. Orthodox medical treatment for duodenal ulceration was given and the symptoms subsided. The haemoglobin and E.S.R. returned to normal in three weeks. The patient was discharged on an ulcer regime. He was asked to attend the follow-up clinic for review and further chest *x*-ray examination, but did not do so.

In 1954 he was again admitted to hospital with post-prandial epigastric pain and frequent vomiting. Barium-meal examination showed evidence of active duodenal ulceration; there was no sign of pyloric obstruction. This examination was repeated two months later with the same findings. A normal acid curve was obtained by fractional test meal. Slight pleural thickening was seen over the upper part of the right lung on the chest *x*-ray film. This has persisted unchanged on subsequent radiographs. E.S.R. was normal. Again there was a good symptomatic response to medical treatment for peptic ulcer.

He had a similar episode in 1955. In 1956 he was in the medical unit of the hospital for three periods of several weeks with epigastric pain and vomiting. Barium-meal examination again showed signs of duodenal ulcer. The intervals of relief produced by strict ulcer regime with bed rest became progressively shorter and surgical treatment was at length undertaken.

Operation (Mr. F. Smith).—On September 13 a hard ulcer crater penetrating the pancreas was found in the pylorus.

*Now at Beilinson Hospital, Petah Tikvah, Israel.