on the inner side of the left arm, with weakness of the hand-grip and fingers.

On September 5 definite wasting of the left palmar muscles and loss of hand-grip were observed. He now received injections of N.A.B., bismuth, and sodium iodide. He was seen by Mr. Nightingale and Dr. Fisher, who thought this to be definitely a case of new growth at the left apex (? origin, but probably bronchial). On the 12th there was oedema and swelling of the left arm (? due to lymphatic or venous obstruction), and left Horner's syndrome. Another radiograph taken on the 15th showed that the condition was worse: there was more erosion of the seventh cervical vertebra; the first rib had disappeared, and the second partly disappeared at the head and neck (Plate, Fig. 1). From this time the patient went rapidly downhill, his temperature became elevated, and he died on September 28.

Post-mortem Examination.—A large mass was found at the left apex involving the lung, upper three ribs, and the vertebrae, and surrounding the left subclavian vein, which was thrombosed. No primary was present elsewhere, but there was terminal pericarditis and pyaemic abscess of the kidney. The growth had the consistency of an unripe pear, and had completely replaced the upper two ribs, which could be cut easily with the knife. Sections of lung, ribs, and cervical glands were made.

Report of Sections.—All sections showed squamous-celled carcinoma with cell-nest formation (Plate, Fig. 2). Catarrhal pneumonia was present in the lung.

Commentary

This case corresponds exactly to the description given by Pancoast, and is actually more complete owing to our being able to obtain a full necropsy. None of the original cases apparently were examined post mortem. The result of microscopy does not settle the vexed question of the origin of the tumour, which Pancoast suggested was in the remnants of the fifth branchial cleft. A tumour in this situation gives none of the common symptoms of carcinoma of the lung, such as cough, sputum, or haemoptysis: its symptoms and signs are due entirely to pressure and mechanical involvement of the structures in its neighbourhood; consequently the picture may be given by any tumour which encroaches upon this particular situation. Cases described by Evans, Tobias, Steiner and Francis, Jacox, and others all exhibited the typical x-ray appearances and clinical features, but were shown to be due to various tumours, such as thymomas, sympathoblastomas, secondary growths from stomach and cervix uteri, and breast and bronchial carcinoma. The last-mentioned forms the largest proportion of the cases, and probably is the usual cause of the syndrome. Browder and de Veer, in describing another case, suggested that the name of carcinoma of the pulmonary apex is more appropriate than that applied by Pancoast. Our case may therefore have been a squamous-cell type of carcinoma at the apex, originating most probably in the lung but possibly in a branchial remnant.

The differential diagnosis includes several widely differing conditions. Cervical rib and syringomyelia may be excluded by the x-ray appearances. Tuberculous fibrosis at the apex does not cause destruction of the ribs or vertebrae. Endothelioma of the pleura spreads more rapidly, and again there is no bone change. Osteogenic sarcoma of the ribs or spine in this region may confuse, but there is generally widespread metastasis to other organs, the superior sulcus tumour being circumscribed and only spreading locally.

All the patients have been unrelieved of their symptoms by any procedure, surgical or otherwise, and a feature of

the condition is its rapidly progressive and fatal course. The growth seems to be completely resistant to irradiation treatment, which was given in the majority of cases reported. Jacox suggests the frequent metastasis of bronchial carcinoma to the suprarenals as a cause of the rapid decline. Such metastasis, however, has not been demonstrated in any case.

I am indebted to Dr. M. K. Jardine for assistance with the case history, and to Mr. H. J. Nightingale and Dr. D. Fisher for permission to report the case.

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FAILED SPLENECTOMY IN ACHOLURIC JAUNDICE, AND THE RELATION OF TOXAEMIA TO THE HAEMOLYTIC CRISES

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The accepted treatment for acholuric jaundice is splenectomy, and in the majority of cases there is little doubt that the results are good. Cowen (1936) mentions "the generally accepted view that the operation brings about complete and lasting freedom from symptoms." Dawson (1931) states: "Even in the severe anaemias the recovery is rapid and permanent, and the patients are thenceforth healthy." Pemberton (1931), however, summarizes the results in 118 cases operated upon between 1911 and 1931 in the Mayo Clinic. The hospital mortality was four, subsequent deaths numbered eleven (six stated to be due to unrelated causes), eighty-two patients were alive and well, and fair and poor results were obtained in sixteen cases. Difficulties exist in the interpretation of these figures, but some degree of failure apparently resulted in at least 20 per cent., the true figure being probably higher. While the author believes that splenectomy should be advised as the safest form of treatment in all cases, these figures suggest that the results may not be so good as some writers would indicate.

Examination of the literature shows that failure, of varying degree, has been due to operative mortality, complications such as hepatic disease and other types of anaemia, and inability to achieve temporary or permanent relief from excessive haemolysis. Relief from the latter is the direct object of the operation, and in this paper consideration is directed chiefly to this question. We have failed to find any published data of its frequency.

It has been recommended that to prevent possible future haemolytic crises splenectomy should be carried out even in slight cases, especially if the family history is bad (Cowen, 1936). Probably this is effective in many cases, but it is clear from the literature (Freund, 1932) that the operation does not guarantee freedom from such crises.

There appears to be some factor which converts latent into acute acholuric jaundice, and as many of these patients are quite well between haemolytic attacks, some information as to the cause of these latter is desirable. Consideration of some of the failed splenectomies, including one of our own, and various other cases, suggests that toxæmia may be one of the factors stimulating haemolysis.

Failed Splenectomies

Freund (1932) quotes six cases from the literature, and records two others, in which operation failed to relieve the haemolysis permanently. Freund's first was a case of congenital acholuric jaundice in a boy aged 10 in whom haemolysis continued without remission after splenectomy, with pyrexia and abscesses, but necropsy gave no clue as to why the case differed from others in which splenectomy was successful. The second case was that of a girl of 11, with chronic bronchiectasis and hyperthyroidism, both of which preceded any history of jaundice. Considerable improvement followed splenectomy, but nine months later she developed a sore throat with pyrexia, and jaundice followed a few days later, death ensuing rapidly. Post-mortem examination showed bronchopneumonia with pus in the dilated bronchi. The author questions the advisability of splenectomy in this case, owing to a possible depression of the defence mechanism against infection. She also suggests that further studies should be made to ascertain whether or not chronic infection occurring in a case of haemolytic jaundice should be considered a contraindication to splenectomy. Both these cases showed increased red-cell fragility.

A. C. and A. Van Ravenswaay (1934) recorded a case of haemolytic jaundice with normal red-cell fragility associated with an infection of the paranasal sinuses and the left ear and mastoid. Multiple transfusions failed to cure, and splenectomy was followed by only temporary improvement, but remission eventually occurred. Further attacks of sinusitis were associated with jaundice and anaemia, and in each instance improvement in the sinus condition was accompanied by an improvement in the anaemia.

Lovibond (1935) records a case in which, eight days after splenectomy, the patient died from a streptococcal infection. There was a history of a mastoid operation five years previously, following which she had been treated for anaemia. Liver, iron, blood transfusion, and splenectomy all failed to improve her condition. Increased fragility was found in one out of six tests. "The probable cause of the anaemia was thought to be either acholuric jaundice or an infective process."

Relationship of Toxæmia to the Haemolysis

The question of the presence or absence of infection or toxæmia and its possible relation to the haemolysis is one of special difficulty.

Some authors accept the presence of pyrexia and leucocyte changes as evidence of infection, but the products of the rapid blood destruction might be sufficient to account for these phenomena (Joules and Masterman, 1935). Also, it is a common experience that cases of severe anaemia tend to be febrile, whatever may be the actual mechanism involved (Lovibond, 1935). While these points constitute no evidence against the toxæmic theory, they render pyrexia and leucocytic changes inadequate for establishing its causal relationship. Some clinical evidence of an association between infection and haemolysis is available in two of the cases quoted above, parallel fluctuations of each in one case being particu-

larly suggestive. Further examples of this relationship are provided by the following cases.

Dedichen (1937) records an epidemic of haemolytic crises in two families, eighteen members of which were found to have familial haemolytic jaundice. The author suggests that the acute manifestations were precipitated by what was presumably influenza. Scott (1935) records an epidemic of crises occurring at short intervals in four members of one family (increased fragility in all), suggesting an infection, although Scott does not favour this view. Infection, nevertheless, does not appear to be excluded, and the epidemic nature of the attacks is suggestive, and is not otherwise explained. It is doubtful, however, if the crises were entirely haemolytic in character. Estiu and Lenci (1931) record a case which developed a haemolytic crisis with palpable spleen following the onset of a tonsillar infection. After tonsillectomy the red-cell count rose almost to normal and the spleen became impalpable. Splenectomy was not performed.

Lederer's acute haemolytic anaemia bears a close resemblance to the crises of acquired acholuric jaundice, and Lederer (1925, 1930) in recording six cases of acute haemolytic anaemia suggested that they were due to infection, basing this opinion on the sudden onset and the presence of fever and leucocytosis. In discussing this condition O'Donoghue and Witts (1932) emphasize the slenderness of the evidence on which anaemia is attributed to foci of infection. They conclude that this acute haemolytic anaemia appears to be a specific illness due to infection, but the nature of the infection is unknown. In selecting thirty-six cases from the literature for consideration, however, they have rejected all with proved infection.

In the absence of experimental evidence the examples quoted do no more than suggest that toxæmia may be a possible factor in the haemolytic crises of acholuric jaundice. This possibility receives some support from the following case, in which splenectomy was performed during a haemolytic crisis without success, but after removal of an ovarian teratoma recovery ensued.

Case of Failed Splenectomy

The patient was a married woman of 44, and was admitted to hospital with a two-months history of shortness of breath on exertion, increasing pallor, and some loss of weight and appetite. She had had no menstrual period for five months. There was no history of previous jaundice and no family history. She was febrile, and there was a lemon tinge in the skin. Apart from a tumour in the lower abdomen, thought to be a fibroid, and a precordial murmur no other physical signs were detected. The haematological findings are shown in the table.

Microscopical examination of the blood showed the presence of the densely staining microcytes usual in acholuric jaundice, along with large numbers of reticulocytes and nucleated red cells. Free HCl was found in a test meal. A diagnosis of a haemolytic crisis in acholuric jaundice probably of the so-called acquired type was made.

COURSE OF THE ILLNESS

A transfusion was given but failed to affect the haemolysis, as did a further transfusion three weeks later. Parenteral liver treatment was tried without effect. Splenectomy was therefore carried out, a small spleniculus being also removed. Gall-stones were noted to be present. The patient's blood was now found to agglutinate the red cells of two donors of her own group (O), but a compatible donor was found and a further transfusion given after the operation.

Although the spleen had been impalpable it was seen to be enlarged on removal, the weight being 500 grammes. The cut surface was a uniform dark red colour, the Malpighian bodies being invisible. Microscopical examination showed the usual feature of acholuric jaundice—namely, well-marked engorgement of the pulp with red cells; the Malpighian bodies were small, and no pigment deposits or Gandy-Gamna nodules were seen.

Table showing the Blood Picture in a Case of Failed Splenectomy

Date	Haemo- globin per cent.	Red cells per c.mm.	Colour Index	Reticulo- cytes per c.mm.	Nucleated Reds per c.mm.	Fragility per cent.	Van den Bergh		Leuco- cytes per c.mm.	Poly- morphs per c.mm.	Lympho- cytes per c.mm.	Lobes per 100 Polys.	Plate- lets per c.mm.
							Direct*	Indirect					
17/1/36	23	812,000	1.42	Numerous	—	—	—	—	—	—	—	—	—
25/1/36	17	580,000	1.47	—	36,746	—	Neg.	Pos.	13,353	8,654	3,313	197	—
26/1/36	Transfused 720 c.cm.												
31/1/36	30	—	—	—	—	—	—	—	—	—	—	—	—
5/2/36	27	1,250,000	1.08	—	—	0.6—0.44	Neg.	Pos.	—	—	—	—	—
7/2/36	26	1,012,000	1.23	455,000	—	—	—	—	—	—	—	—	162,000
15/2/36	Transfused 600 c.cm.												
19/2/36	23	—	—	—	—	—	—	—	—	—	—	—	—
21/2/36	18	850,000	1.06	—	—	—	—	—	—	—	—	—	—
21/2/36	Splenectomy. Transfused 720 c.cm.												
25/2/36	29	1,300,000	1.12	—	—	—	Pos.	Pos.	—	—	—	—	—
28/2/36	31	1,375,000	1.13	550,000	80,376	—	—	—	21,624	17,340	3,264	194	80,000
2/3/36	38	1,830,000	1.04	604,000	—	—	—	—	—	—	—	—	100,000
6/3/36	37	1,887,000	1.00	774,000	—	—	Neg.	Pos.	—	—	—	—	280,000
11/3/36	45	2,150,000	1.05	473,000	700	—	—	—	7,900	4,977	2,054	224	251,000
25/3/36	29	1,253,000	1.12	—	17,472	—	Neg.	Pos.	18,928	—	—	—	—
15/4/36	30	1,170,000	1.28	468,000	15,038	—	„	„	27,600	20,110	5,796	222	140,000
29/4/36	26	—	—	—	—	0.6—0.44	—	—	—	—	—	—	—
25/5/36	27	—	—	—	—	—	—	—	—	—	—	—	—
3/6/36	Right ovariectomy and cholecystectomy												
22/6/36	62	3,050,000	1.02	290,000	59	—	—	—	5,900	3,038	1,888	226	533,000
29/6/36	—	—	—	—	—	0.62—0.4	Neg.	Neg.	—	—	—	—	—
10/8/36	76	—	—	—	—	—	—	—	—	—	—	—	—
12/10/36	88	4,680,000	0.94	61,000	0	—	—	—	8,000	5,600	1,920	265	340,000
14/12/36	89	4,310,000	1.03	—	0	—	—	—	7,300	4,351	2,219	276	—
15/2/37	82	4,000,000	1.02	—	—	—	—	—	7,200	—	—	—	356,000
31/5/37	96	—	—	—	—	—	—	—	—	—	—	—	—
18/10/37	90	4,175,000	1.08	—	0	0.4—0.3	Neg.	Neg.	7,500	5,025	1,875	277	—
21/3/38	89	4,240,000	1.05	70,000	0	0.45—0.3	„	„	7,300	4,818	1,861	268	389,000

* The direct Van den Bergh test refers only to the immediate reaction.

Following the splenectomy the patient was worse, the jaundice becoming deeper and of the obstructive type for a short period, with some swelling of the left leg and pleural rub and pain in the chest. Pyrexia persisted and there was no cessation of haemolysis, but there was an increase in the reticulocytosis. The haemoglobin rose to 45 per cent., but soon fell back below 30 per cent.

Eventually after four months in hospital, during which splenectomy, three transfusions, and liver therapy had failed to arrest the haemolysis, it was decided as a last resort to explore again in search of a spleniculus. This was done, but none was found: the gall-bladder and the pelvic tumour, which proved to be an ovarian teratoma, were removed. No transfusion was given. Unexpectedly the haemolysis ceased immediately, the signs of toxæmia disappeared, the temperature settled, and the blood began to return to normal, complete recovery ensuing.

ULTIMATE BLOOD CHANGES

Twenty-six days after the last operation, the excessive haemolysis having ceased, the increased fragility was still present. It was not tested again for sixteen months, when it was found to have become normal. The use of a quantitative technique and washing the red cells revealed no defect. Price-Jones curves were constructed from blood films taken before the splenectomy and transfusions (25/1/36) and after the establishment of normal fragility (18/10/37), and are shown

in the accompanying chart. The mean corpuscular diameter was originally normal, but the variability was very high.

After recovery the mean diameter was found to be above the normal range (8.136 μ), but the variability was normal and

Chart showing the Price-Jones curves before splenectomy and transfusions and after the establishment of normal fragility.

no large megalocytes were found. The mean corpuscular volume (98 c. μ on 18/10/37) was also slightly increased, with a normal mean thickness (1.9 μ , calculated). Apart from these changes and an increased platelet count the blood picture has become normal.

THE OVARIAN TERATOMA

The ovarian teratoma consisted of a dermoid cyst containing some pellets and flakes of sebaceous matter and hairs. The following tissues were present in the wall: squamous epithelium, sebaceous and salivary glands, hairs, intestinal wall including mucosa, lymph follicles, muscular tissue, nerves, and nerve ganglia. No splenic tissue was found. Degenerative changes with an inflammatory exudate and polymorph infiltration and plasma-cell foci were present in places. A bacteriological examination was not made, as its possible significance was not apparent at the time.

The gall-bladder contained numbers of dark-coloured faceted calculi. Except for pigmentation of the epithelium the wall showed no changes, but the cystic lymph gland contained considerable deposits of brown pigment, conspicuous to the naked eye. Microscopically these were found to be associated with proliferated endothelial cells lining the sinuses; the pigment gave a negative reaction for iron, and was presumably of biliary origin.

Discussion

In attempting to explain the train of events in this case the usual limitations of uncontrolled clinical material of this type restrict the drawing of conclusions, but the most probable explanation, so far as it goes, is that some substance was being absorbed from the ovarian teratoma and was, directly or indirectly, one of the causes of the haemolysis. Its mode of action can only be a matter of speculation, and we are unable to define the relation of the excessive fragility to the condition. It was present throughout the haemolytic period, persisted afterwards when excessive haemolysis had ceased, but eventually disappeared, and the mean corpuscular diameter, which had been normal, became abnormally high. The findings suggest a change from spherocytosis to a flatter cell of normal thickness and increased diameter, but it is of interest that Hawksley (1936), as the result of a study of the red-cells, suggested that familial acholuric jaundice could not at present be regarded as a primary defect either of erythropoiesis or of splenic function; and Vaughan (1937) concluded that increased spherocytosis is not the fundamental abnormality present in the red cell.

There are certain other possibilities which appear less probable. The removal of the gall-bladder at the second operation might be related to the successful result. No significant changes were found in the gall-bladder wall, and although calculi were present they are also present in a large proportion of cases of acholuric jaundice, and whether they are left or removed at operation does not appear to influence the result so far as haemolysis is concerned.

With regard to alternative diagnoses the condition most closely resembling this case is Lederer's haemolytic anaemia. The following are against this diagnosis: (1) the failure to respond to transfusions; (2) the excessive fragility and other red-cell changes; (3) the prolonged period of haemolysis (four to six months); (4) the histopathology of the spleen, which was that of acholuric jaundice. While these may appear to be good reasons for differentiation, we discuss this question further below.

The absence of a previous history and a family history (two daughters were available for fragility tests and both were found to be normal) is usually accepted as evidence that the case is of the acquired type of excessive red-cell fragility, although the existence of the acquired type is still questioned (Vaughan, 1936). There is some evidence that the two types exist, but we have no definite conclusion to offer on this question. As excessive fragility

persisted for some time after cessation of the haemolysis an additional factor or factors (besides the spleen) must have been present during the crisis.

Classification of the Haemolytic Anaemias

The classification of the haemolytic anaemias is still unsettled, chiefly owing to lack of knowledge of their aetiology. It is not even clear whether cases showing increased fragility should be classified with those in which fragility is normal. Of Lederer's six cases (1925, 1930) the fragility is mentioned in three, and in these it was normal. Other writers have disregarded this point, and many cases showing increased fragility have been classified as Lederer's haemolytic anaemia.

Lederer did not discuss the differentiation of his cases from the crises of acquired acholuric jaundice. O'Donoghue and Witts (1932), in dealing with differential diagnosis, differentiate Lederer's anaemia from acholuric jaundice by the absence of family history and the brief duration, and from chronic acquired haemolytic anaemia by the acute febrile course with leucocytosis and signs suggestive of infection, but they provide no means of distinguishing it from haemolytic crises in acquired acholuric jaundice.

We have mentioned above four reasons for differentiating our case from Lederer's haemolytic anaemia, but as the latter condition has not yet been adequately defined we are unable to estimate to what extent these differential criteria are valid. We make the following comments regarding them.

1. Failure to respond to transfusion.—Some cases described as Lederer's have failed to respond to transfusion, and some cases of acholuric jaundice (Scott, 1935) have apparently responded well. One of us has seen an acute haemolytic crisis apparently respond satisfactorily to a transfusion, but when recurrence took place, over a year later, transfusion failed. Response is apparently variable in either condition and even in the same individual.

2. Excessive fragility.—This has already been discussed. Other red-cell changes have not yet been adequately defined in Lederer's.

3. Duration.—According to O'Donoghue and Witts, Lederer's disease is of a self-limited character, tending to death or complete recovery within a period usually of a few months. The value of this criterion is nebulous.

4. Histopathology of the spleen.—This is not yet established in Lederer's anaemia, as in none of his six cases was the spleen available for examination.

It is apparent that the differentiation of the two conditions is by no means clear, if indeed Lederer's anaemia is a distinct entity. In the absence of any record of increased red-cell fragility in Lederer's original cases, haemolytic crises showing this feature appear to us to be best classified in the acholuric jaundice group until more is known of the aetiology. It is perhaps of some interest that Harvey and Janeway (1937), in describing three cases of haemolytic crises apparently due to sulphanilamide, comment on their striking similarity to Lederer's anaemia.

Treatment

The first treatment usually adopted in haemolytic crises appears to be transfusion, in spite of its special risks. When this fails splenectomy has to be considered. Some of the cases quoted and the new one recorded indicate that an alternative sometimes exists, and our case shows that this alternative may be effective where the other two methods fail. It would seem reasonable to suspect the presence of an infection or other toxæmia in any case of acholuric jaundice showing a haemolytic crisis; but that

there are other factors in the production of the latter is apparent from its rarity compared with the frequency of toxæmias.

Failed splenectomies have been recorded not only in the apparently acquired type of acholuric jaundice but also in the hereditary congenital type (Freund, 1932), and there seem to be no data available to show whether failure is commoner in one type or the other. It is doubtful at present, therefore, whether splenectomy should be regarded with greater favour in the familial congenital type.

Summary and Conclusions

A case of acholuric jaundice with a haemolytic crisis is recorded, in which splenectomy failed to cure the haemolysis.

Recovery followed the subsequent removal of an ovarian teratoma.

From a consideration of this case and others from the literature it is apparent that splenectomy does not always cure or prevent haemolytic crises in acholuric jaundice, and where radical treatment is necessary splenectomy is not invariably the method of choice.

Evidence suggesting that toxæmia may act as one of the precipitating factors in producing a haemolytic crisis, and that the latter may be cured by removal of the toxæmia, is discussed.

We are indebted to Mr. P. McEwan in regard to the surgical procedures mentioned.

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F. Hamburger (*Dtsch. Tuber.-bl.*, March, 1938, p. 53) points out that almost every tuberculous patient has tuberculous bronchial glands; there is, however, a narrower and more definite clinical entity occurring chiefly in infants under 2 years old. The general symptoms include a subfebrile daily temperature with a slight rise at night, lassitude, night sweats, and anorexia. Local symptoms are due to compression of the bronchi or trachea, and give rise to a high-pitched metallic cough and expiratory stridor. Similar symptoms may be due to mediastinal tumours or to acute bronchitis, and these conditions must be ruled out. Physical examination is of little value in the diagnosis of tuberculous bronchial glands. The tuberculin test must always be carried out; a negative result is proof of the absence of tuberculosis. An increased shadow round the hilum may be due to causes other than enlarged bronchial glands; its presence in a radiograph must be interpreted together with the other clinical data. A high blood sedimentation rate of 30 to 40 millimetres or more after an hour is in favour of active tuberculosis; a lowering of the rate is of good prognostic significance.

Clinical Memoranda

Fragment of Needle in Foot

(WITH SPECIAL PLATE)

The following case history presents some points that may be of interest.

CASE RECORD

A man consulted me on March 15, 1937, complaining that for the past two years he had suffered recurrent attacks of pain in the right ankle-joint. Latterly this symptom had increased in severity, and on exertion the right foot became swollen.

Clinically the condition appeared to be due to chronic synovitis of the ankle-joint. X-ray examination revealed a fragment of a needle lying in the soft tissues below the head of the os calcis (Plate, Fig. 1). Under local anaesthesia a Southey's tube was inserted into the sole of the foot down to the location of the foreign body, using the fluorescent screen. The trocar was removed, leaving the cannula in position. A further x-ray photograph (Fig. 2) was then taken by Dr. McDougall, who reports as follows:

"A piece of the eye end of a needle about half an inch long is visible close to the bone in the plantar aspect of the head of the os calcis. In the dorsi-plantar view the foreign body is right in the centre of the foot and about a quarter of an inch from the joint formed by the os calcis and the cuboid. A needle indicator is in position showing the relation of this guide to the foreign body."

Under local anaesthesia a vertical incision about three inches in length was made in the sole of the foot. Using the cannula as a guide, the dissection was carried through the plantar aponeurosis and deeper structures down to the head of the os calcis. The fragment of needle was found under the periosteum. The distal end of the cannula was situated about a quarter of an inch from the foreign body.

Points of interest are: (a) the use made of the cannula as a guide; (b) the patient had no history of a needle having entered his foot or any other part of his body.

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Ischio-pubic Osteochondritis: with Report of a Case

(WITH SPECIAL PLATE)

This uncommon condition has only recently been recognized clinically. Attention was first drawn to it by Van Neck in 1924. Since then a few cases have been described in America and on the Continent, but none has been reported in the British Isles. Probably it does occur with some rarity and only needs a wider recognition of its clinical and radiographic features.

The condition arises before the union between the ischium and pubis is complete. This occurs between 8 and 10 years of age. The case described below was that of a boy 8 years old when first seen. He had been complaining of pains in his lower limbs for four years. In Durham's series the average age of the five patients was 7½ years. Either sex may be affected. The condition may be bilateral or may only affect one side. It is a non-suppurative osteochondritis allied to the disturbances in growing areas met with in Perthes's and Osgood-Schlatter's disease. Durham thought that trauma might be a causative factor in some of his cases. There was no definite severe injury in the present case, but the boy was apt to trip and fall easily, and this may have been a factor.