Coxa Plana (Pseudo-coxalgia).

This is another acquired disability of the hip, and is so named from the flattened appearance of the head of the femur. The chief symptom is lameness without definite cause to account for it. Movement at the hip is often free and painless, although sometimes rotation and abduction are limited. There may be little or no shortening of the limb. It is usually unilateral. Legg, who first described the disease in 1910, believes the condition to be due to a traumatic disturbance of the blood supply of the epiphysis. Infective epiphritis is another theory, staphylococci being found in some reported cases.

Tuberculous Hip.

Tuberculosis of the hip is the remaining disability to be considered, and it is the commonest. Here again an early symptom is lameness with a varying degree of pain at the joint, and often a history of night cries with loss of sleep. On examination the child is wasted, pale, and possibly anxious and fretful about any examination of the hip. This is in strong contrast to the other disabilities already discussed. There is often protective spasm of the muscles about the joint, and movement in all directions is restricted because of this. Or there may be little spasm and hardly any restricted movement; frequently the only resentment occurring when the limb is passively rotated, due to the rubbing of inflamed synovial or bony surfaces against each other. In the more advanced stage, marked wasting of the thigh muscles with flexion and external rotation of the limb, and possibly abscess formation occur, leading ultimately to internal rotation and shortening. But the disease then offers no difficulty in diagnosis.

The Disabilities Summarised.

Taking now these four disabilities of the hip-joint, congenital dislocation and coxa vara resemble each other clinically, and to a less extent so do coxa plana and early tuberculosis. In congenital dislocation of the hip the head of the femur can be felt on the dorsum ili and telescoping is present. Trendelenberg's sign is unreliable; it may be present in both dislocation and severe coxa vara. Traumatic coxa vara presents less difficulty owing to the history of a previously normal hip, whereas dislocation and congenital coxa vara were each noticed as a disability when the child began to walk. A radiograph should always be taken in any disability at the hip-joint. It will often settle the diagnosis beyond all doubt. In congenital dislocation of the hip it will also give some indication of prognosis from the point of view of treatment. The radiograph will show first the extent of the acetabulum, and whether or not after reduction the roof will prove an effective barrier to ensure stability; and, secondly, the amount and nature of the deformity of the head and neck of the femur. In coxa plana the symptoms are much milder than in tuberculosis. There is none of the anxiety, fretfulness, spasm and wasting of tuberculosis, and manipulation of the joint is more tolerable. The grosser signs of fixed flexion and rotation are entirely absent in coxa plana. The radiograph is conclusive. In coxa plana the epiphysis is flattened and frequently broken into two or three fragments, and in the unilateral case presents a striking contrast to the sound hip. The joint is otherwise well defined. In early tuberculosis there may be a gross bony lesion, but only a cloudiness of the joint and a general lack of definition. This is often attributed to a fault of the radiograph, but the repeated exposure and a comparison with the opposite joint will readily convince one that the radiographic appearances are due to pathological changes in the joint. A further stage in the process will exhibit a small erosion at the surface of the femoral head or the acetabulum or a small cavity in the epiphysis or the femoral neck. Beyond this the radiographic appearances increase gradually through various pathological stages up to complete disorganisation and ankylosis.

CASES FROM WARDS AND OUT-PATIENTS.

By J. A. RYLE, M.D. LOND., ASSISTANT PHYSICIAN TO GUY'S HOSPITAL.

The two cases to be first described presented themselves at Medical Out-patients on the same morning, both complaining of severe attacks of epigastric pain coming on at varying intervals and for no apparent reason. In both the alternative diagnoses of biliary colic and tabetic crises were discussed, and in one case the latter possibility had already been carefully considered elsewhere. In both, however, there was good reason for reviewing other possibilities.

Case 1.—The patient, a waiter in a café, aged 35, complains of attacks of bad epigastric pain, "drawing" or "gripping" in character, and sometimes referred through to the back. He first experienced these attacks five years ago, and describes them as arriving with varying frequency, sometimes two in a week, sometimes with intervals as long as six months. Latterly they have been more frequent. The pain bears no relationship to meals or any other event in his daily life. It is sometimes accompanied by nausea or vomiting. He has never been jaundiced. He thinks that there has been some loss of weight. Latterly he has also complained of a feeling of great weakness. There is a history of syphilis treated by injections 17 years ago. Two years ago he was investigated at a hospital for nervous diseases and had both the blood and cerebro-spinal fluid examined with a negative result. Radiograms of the stomach, duodenum, and gall-bladder region are reported negative.

Examination of the central nervous system reveals no signs of tabes dorsalis nor are there any signs pointing to organic disease in heart, lungs, or abdomen. The patient looks ill, is hollow-cheeked, and has rather a dingy complexion with profuse freckling. Brownish pigmentation with a freckle-like distribution extends on to the mucous of
the lips. On close inspection of the mouth patches of grey or black pigmentation are clearly discernible on the inner aspect of both cheeks. The extremities are unduly cold for the time of year. The pulse is thready and weak, and we are surprised to find that the sphygmomanometer registers so high a systolic pressure as 120 mm. of mercury. On the occasion of his next visit to out-patients a week later patient reports further attacks of pain and, without any questioning, again lays stress upon the feeling of extreme weakness. The systolic blood pressure is still 120 mm. of mercury.

**Suggested Diagnosis.—Addison’s disease.**

Of the four important manifestations of Addison’s disease—namely, pigmentation, asthenia, gastric symptoms, and low blood pressure—the first three are present in this case. The gastric symptoms usually described include both vomiting and pain, and the latter is occasionally reported to be so severe as to suggest biliary colic or gastric crises. Although an abnormally low blood pressure is regarded as an essential finding in Addison’s disease, its development may be preceded by the other symptoms, and we are able to recall clearly a case in which the systolic blood pressure was registered as 125 mm. of mercury 25 days before the patient’s death. The length of history might be held to militate against a diagnosis of Addison’s disease, for the majority of cases die within two years of the first recognition of symptoms. Histories of five and seven years have, however, been recorded. Fagge confirmed at autopsy in 1865 a case diagnosed by Sir William Gull in 1860.

The patient is to be admitted for further observation. A falling blood pressure during the next few weeks will help to confirm the diagnosis of Addison’s disease.1 In recent years we have been trying to obtain radiograms showing the diseased suprarenals. It seems reasonable to suppose that in a thin subject and in the presence of fibrocaceous changes the glands might sometimes prove to be as opaque to the rays as caseous thoracic glands are commonly shown to be. The position of the glands in close proximity to such dense organs as the liver and kidneys, however, makes it difficult to obtain successful pictures.

**Case 2.—The patient is an inspector, male, aged 37.** He was admitted as an urgent case to another hospital a few weeks ago in a collapsed condition and complaining of intense pain in the upper abdomen. He was then reported to have a slow but weak pulse, a subnormal temperature, and great pain, rigidity, and tenderness in the upper abdomen. A diagnosis of perforation of a hollow viscus was made and he was prepared for operation. In about an hour, however, the pain ceased and the abdominal signs disappeared. On discharge from hospital a week later he was referred to a surgical colleague at Guy’s for diagnosis, who then referred him to Medical Out-patients. Patient is anxious and obviously in dread of a return of his attacks of pain, of which he has had several though none so severe as the one described. He does not look seriously ill. His abdomen is still stained with the picro solution applied on admission to the other hospital in anticipation of operation.

On inquiry into this previous history, it appears that he was invalided after active service during the war “on account of his heart,” and that he has drawn a pension for cardiac disability ever since. He admits, however, to no cardiac symptoms before the war, and has not had rheumatic fever. He states that he is now short of breath, even when walking on the level, and that he cannot “talk and walk at the same time.” He first began to have attacks of epigastric pain a few months ago during a period in which his heart was bad, and this was tried with severe coughing. The attacks of pain have not been induced by effort, and may come on at any time. He has not had jaundice. His pupils react to light and his knee-jerks and ankle-jerks can be readily elicited. His pulse is regular and not accelerated. There is a well-marked crescendo presystolic murmur and a presystolic thrill. The liver is slightly enlarged and there is tenderness on palpation below the right costal margin. There is no cyanosis or oedema of the feet, and breathing at rest is normal and comfortable.

With such well-marked mitral stenosis and poor cardiac reserve, the patient is perhaps to be congratulated on having escaped an anaesthetic and a laparotomy. In advising future treatment we shall obviously have to be guided by a knowledge of his cardiac lesion. It is much more difficult to decide whether his attacks of pain should in any way be associated with the only organic lesion of which we have clear evidence. Anginal pain may be referred to the epigastrium and simulate acute abdominal disease, but angina pectoris is an exceedingly rare accompaniment of mitral stenosis. The patient, due to a failing heart may give rise to considerable pain in the upper abdomen, but hardly in the form of severe, transient attacks. Patient is to be admitted for further observation. The possibility of biliary colic is under consideration, and for the present we must content ourselves with a decision to regard his case with a medical rather than a surgical eye.

**Case 3.—The patient, a male, aged 38, is at present undergoing treatment in hospital, and is another example of Addisonian pigmentation.** His own medical adviser had very reasonably concluded that he was suffering from Addison’s disease.1 He was registered for Addison’s disease (suprarenal tuberculosis). His appearance is very striking. His face shows a uniform dingy pigmentation, and there is a deeper pigmentation of the back of the neck and the trunk, particularly about the waistline. The nipples are deeply pigmented, and there are several dark moles on the face and trunk.

On examination of the buccal mucous membranes numerous patches of red pigmentation were observed. The symptoms which he described on admission included great weakness and trembling in the legs. Nevertheless, a closer inquiry into his past history and present symptoms and signs enables us to establish a diagnosis not of Addison’s disease but of Addison’s (pernicious) anemia, and this has since been confirmed by other investigations. The patient reports no serious illness until 18 months ago. He then became very weak and ill, and was advised to have all his teeth extracted on account of pyorrhoea. This advice was followed and he slowly improved, and in three months was able to return to his occupation as a cinematograph operator. He remained at work until shortly before his admission when the old symptoms returned and he became short of breath on the slightest effort. He eats and sleeps well; has no diarrhoea or sore tongue, and has, if anything, gained a little weight since last summer.

A closer inspection shows that his dingy pigmentation covers an anaemic pallor, the general effect being that of a dirty yellow, not quite characteristic either of Addison’s disease or of Addison’s anemia. The mucous membranes are very pale. The spleen is palpable. There is a soft systolic murmur audible both at the apex and the base of the heart. His temperature occasionally rises above the normal line. His history of a remission after the dental extractions last year, the addition to his general anaemia of pronounced diarrhoea and pallor (a few symptoms which do not obtain in Addison’s disease), a palpable spleen, and a systolic blood pressure which varies from 115 to 130 mm. of mercury and does not tend to fall, all serve to exclude caseous disease of the suprarenals, a diagnosis which his pigmentation otherwise so strongly suggests.

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1 Two weeks later the systolic blood pressure has fallen to 90 mm. of mercury.
Pernicious anæmia is one of the diseases in which Addisonian pigmentation, involving both skin and mucous membranes, may occasionally be found. It is interesting to speculate whether the pigmentation depends upon some degenerative change involving the suprarenals and produced by the same toxins which affect the heart muscle and more rarely the central nervous system. By a curious chance in the next bed to this patient there is yet a third case of Addisonian pigmentation, developing during observation, in association with malignant disease of the stomach. The hemoglobin in the case under discussion was 29 per cent. on admission. There is a high colour-index. Films show well-marked anisocytosis and poikilocytosis with a preponderance of large forms. There is a typical Price-Jones curve. Test-meal shows complete achylia. Treatment is being carried out along the lines suggested by Hurst and includes transfusions, full doses of hydrochloric acid, iron and arsenic, and sour milk. Some improvement has been registered and the hemoglobin has risen to 41 per cent., but we hesitate to give a good prognosis. Of the three cases which we have personally seen recover, apparently completely, after treatment along these lines, all three were aged 60 or more, and were afebrile. Youth and pyrexia in Addison's anæmia would at present seem to be adverse prognostic features. 

Case 4.—A girl, aged 21, was referred to Medical Outpatients with a history of repeated haemoptyses during the previous two months. She has lost a few pounds in weight, and complains of slight impairment of appetite and taste in the mouth on waking in the morning. The sputum has already been examined for tubercle bacilli, but none were found. Fortunately (for the diagnosis depends largely on the character of the sputum) she has brought this morning's specimen with her. In quantity it is about three drachms; it is fluid, chocolate-red in colour, and when it cools resembles the pus in a liver abscess; it contains no mucoid element, and has a very foul odour. It is, therefore, quite unlike the blood-stained sputum of phthisis, and the foul smell at once suggests the possibility that it has come from some abscess communicating with the mouth, or (less probably) from a lung abscess, case. Examination of the chest reveals no physical signs of disease. The patient has no pyrexia, and does not look ill.

On further inquiry it appears that she only brings up sputum in the morning, and that she expectorates every morning. She is asked to remove an upper denture and we can now see extensive but quite superficial ulceration of the palatal aspect of the upper alveolus on both sides. She is asked to suck a gum, and spits the spume brightly stained with blood. It appears that she wears her dentures all night as well as in the day.

We conclude that the bad taste in the mouth and the foul watery sputum have been produced by decomposition under the dentures of blood-stained exudate from her ulcerated palate, and that this ulceration has been caused by an ill-fitting denture and neglect of simple hygiene precautions.

Reviews

Diseases of Children.

A Short Introduction to their Study. By Hector Charles Cameron, M.A., M.D., F.R.C.P., Physician in charge of the Department for Diseases of Children, Guy's Hospital, London; Humphrey Milford, Oxford University Press. 5s.

An undergraduate learns most of his medicine and surgery from a study of adults, and this is as it should be, for they form easier subjects for investigation and treatment than do children. His curriculum is now so extended that it is impossible to lengthen it, nor can we put more into it; for it is already full. Nor is the student taught anything that he need not know. We think it is, therefore, useless for those whose work lies exclusively among children to urge that the undergraduate should devote more time than he does at present to their study. In any subject, whether it be medicine or not, when a student first becomes a graduate in it, he has merely arrived at the position of having learned how to study the matter further. The taking of an average degree in classics does not imply that the newly fledged graduate has studied all branches of his subject, nor does the taking of a qualification in medicine mean that the newly qualified man knows all about it. Probably that department of it in which he feels most ignorant is diseases of children, and consequently there is no post-graduate work that is more essential for the student and for the community among whom he is going to work.

Every man or woman who is going to practice the profession of medicine must, as soon after qualification as possible, begin to re-study diseases of children—and that for many reasons. There are so many children; as they cannot describe their illnesses, the right interpretation is much more difficult than in the case of adults; the children have or ought to have a long life in front of them, and, therefore, they must be as healthy as they can be; and, lastly, they are frail so that an injury or an illness that would hardly affect a young healthy adult may be fatal to them. Beyond its mere outlines, the study of diseases of children is a post-graduate subject, and we know of no better book with which to begin than this small work by Dr. Cameron, for it is not a text-book, but an admirable introduction to a difficult subject. As the author says, "The doctoring of little children can never be an easy matter. The symptoms of disease in childhood are so little differentiated that formidable disorders are apt to pass under the guise of trivial. Intestinal obstruction, for example, or peritonitis, may be hard to distinguish from dyspeptic disturbance. No branch of medicine requires greater experience, in none is experience harder to come by." All this is perfectly true, therefore again we see children's diseases must be carefully studied by every doctor. We would strongly urge that the already qualified should carefully read Dr. Cameron's admirable book and a perfectly clear, perfectly readable. With it as a guide he should be a real pleasure to take up the subject of which treats, which can be studied for its own sake without the bogey of an examination. Children's hospitals and clinics must be visited, and when the practitioner feels himself quite at home with a difficult illness in a child, he will have attained a knowledge that will make him happy in doing good, and he will have become so interested in the subject that he will be able to play worthily his part in the modern movement of infant welfare.

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