

association of fatal so-called "non-obstructive" anuria with widespread necrosis of the secreting cells of the renal convoluted tubules, but it differs from the others chiefly in the absence of obvious microscopic signs of thrombosis of the interlobular arteries of the kidneys. The Malpighian corpuscles showed little or no acute changes. It seems to me that the one essential change in all the above described cases is the widespread necrosis of the secreting cells of the renal convoluted tubules and that this change is merely an extreme degree of the change known as "granular degeneration" or (in its earliest form) as "cloudy swelling," a change which is probably present to a greater or less extent in all cases of acute nephritis and in many toxæmic conditions. I suppose that when for any reason (e.g., violence

may be remembered that acute parenchymatous degeneration of hepatic cells is known occasionally to supervene in previously diseased (cirrhotic or enlarged and fatty) livers.

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ADDISONISM AS A FAMILY DISEASE.

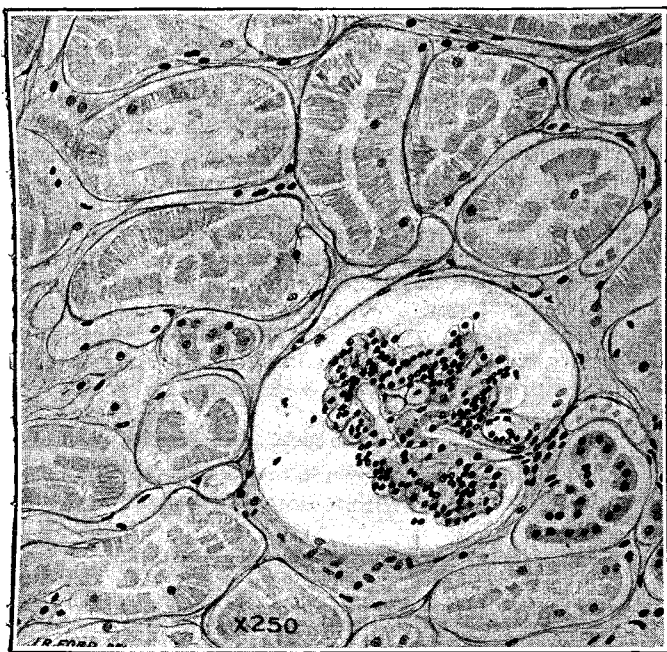
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WHILE the disease associated with the name of Addison is now recognised as comparatively common, it is surprisingly rare in children. In a recent paper published in the *New York Medical Journal* Felberbaum and Fruchthandler in describing a case in a girl, aged 12 years, mentions that in a summary of the literature they have only succeeded in collecting 25 cases occurring in children under 13 years of age. Of the 25 cases 12 occurred between the ages of 10 and 13 years, 4 between five and 10 years, while 9 occurred below the age of five years. Under the circumstances a report of the following three cases, members of the same family, which came under my observation as physician to the Royal Dispensary, Edinburgh, is not without interest.

CASE 1.—The patient was a girl, aged nine years. The father and mother were alive and well, and of four sisters the eldest was 17 years of age, the second had been still-born, the third was six years of age, and the fourth was aged three and a half years. There was no history of previous similar trouble in the family, but a grandmother and uncle had died from pulmonary tuberculosis. The child, who was healthy at birth, had been breast-fed. There was no history of congenital syphilis. She had suffered from chicken-pox at the age of one and a half years but from no other infectious diseases. Pneumonia had been experienced at the ages of nine months and 18 months, she had been treated three times subsequently in the Royal Hospital for Sick Children for "chest trouble," and during the last three years had been feeling very "tired." For the last year her skin had been becoming very dark brown and from time to time this had been more marked. The patient was often too tired to do exercises at school, did not run about at play, and at times felt very sick and vomited occasionally. On examination she was found to be thin and appeared to be poorly nourished. The skin was dark in colour and at certain sites the pigmentation was more intense; round the hips, where the pressure of the clothes fell on the body, the pigment was very dark. The popliteal spaces, where the garters rubbed up and down with each movement of the knee-joint, were very dark. Pigmentation was marked at the umbilicus, in the folds of the axillæ, and in the creases of the hands, but it was interesting to note that those parts of the axillæ where no pressure fell failed to show pigmentation. There was a scar on the left malleolus round which there was deep pigmentation. A more recent scar under the instep showed signs also of commencing pigmentation. A point of great importance was the absence of involvement of the buccal mucous membrane. In addition to the cutaneous pigmentation already noted, there were numerous black spots of about the size of a pinhead scattered about the body. There was nothing abnormal to note with regard to the circulatory system. A blood count gave the following results: red cells, 4,820,000; hæmoglobin, 80 per cent.; and white cells, 8200. A differential count gave the following percentages: Polymorphonuclear cells, 68; lymphocytes, 29; large mononuclear cells, 1; eosinophiles, 2; and mast cells, 0. The measurement of the blood pressure (Riva-Rocci) gave a maximum systolic pressure of 90 millimetres of mercury. With regard to the respiratory system, there was nothing abnormal to note except that the note on percussion over the left apex was slightly higher in pitch than the right. The patient's weight was 3 stones 5 pounds. Pirquet's cutaneous tuberculin reaction gave a negative result, while the result given by Calmette's tuberculin reaction was positive.

I am much indebted to Dr. G. A. Gibson and Dr. F. D. Boyd for having admitted this case to their wards in the Royal Infirmary and the Deaconess Hospital respectively and so enabling me to study the case more thoroughly than otherwise would have been possible.



The figure is drawn from a portion of the cortex of the left kidney (magnification 250) and shows a Malpighian corpuscle surrounded by convoluted tubules, the epithelium of which is necrotic. The secreting cells are mostly detached or broken up and scarcely take on the logwood stain at all. Their nuclei have disappeared (karyolysis) or are hardly to be distinguished from the cytoplasm.

of the local or general exciting cause, want of cell vitality from previous disease, or otherwise) the cloudy swelling and degeneration rapidly pass on to actual cell necrosis the convoluted tubules become mechanically blocked and more or less complete anuria results.⁵ At a later stage perhaps the interlobular arteries and their glomerular branches become thrombosed (as in the three cases first referred to) and then the glomeruli likewise degenerate. As I have already explained, it is, I believe, more rational to regard thrombosis of all the interlobular arteries, when it occurs, as secondary to the parenchymatous necrosis than to regard the parenchymatous changes as altogether secondary to the arterial thrombosis. Perhaps the necrosis of the glandular cells produces a substance having a hæm-agglutinative action which favours thrombosis in the neighbouring blood-vessels.

Hæmaturia at the commencement of attacks of acute nephritis may have a beneficial effect by relieving pressure—that is to say, it may represent an automatic conservative measure on the part of the organism. Probably all cases of so-called non-obstructive suppression of urine (non-obstructive anuria) should be regarded as representing an exaggerated form of the functional and morphological changes in the renal cells that occur in all cases of acute nephritis or acute exacerbations of chronic nephritis or as the result of the actions of various toxic substances on the kidneys. What was the immediate exciting cause of the acute parenchymatous degeneration in the present case is not quite clear, but certainly the kidneys were already diseased; and in regard to another abdominal viscus—namely, the liver—it

⁵ Perhaps temporary anuria may sometimes be a mechanical result of swelling (without actual necrosis) of the secreting cells of the convoluted tubules. I do not, however, wish to imply that all cases of so-called "non-obstructive anuria" are really obstructive and due to mechanical blocking of the convoluted tubules from swelling (with or without actual necrosis) of their epithelium.

CASE 2.—The patient, aged six years, was well nourished but listless, and did not run about at play but preferred to sit still. Diarrhoea was occasionally suffered from. There was a band of pigmentation round the neck. The posterior folds of the axillæ were also slightly pigmented and there were a well-marked band of pigmentation round the waist and a slight darkening behind the left popliteal space, but there was no involvement of the mucous membrane. There was no other abnormality of note. The blood pressure as measured by the Riva-Rocci instrument showed a maximum systolic pressure of 105 millimetres of mercury.

CASE 3.—The patient, aged three and a half years, was poorly nourished and very listless. Pigmentation involved the neck very slightly and was fairly well marked round the waist, but was not present in other situations. The maximum systolic pressure was 98 millimetres of mercury. The blood showed no abnormality. The eldest sister, aged 17 years, showed no abnormality.

From a study of the three cases, it can hardly be doubted that at any rate Case 1 was a true case of Addison's disease. The prostration, the asthenia, the distribution of the pigmentation, the low blood pressure, and the occasional vomiting go to make a typical picture of the disease, and the only important negative point in this case (common to the three cases) is the absence of pigmentation of the buccal mucous membrane. The second and third cases, while showing only one of the cardinal signs of Addison's disease—namely, pigmentation, must with a knowledge of the elder sister's condition be viewed with suspicion. The low blood pressure, especially well marked in Case 3, is also a point of great significance. To revert to Case 1, an especially interesting feature is the fact that a positive result was obtained by Calmette's tuberculin reaction as pointing to a possible tuberculous condition of the suprarenals. The lesions found post mortem in the adrenals in Addison's disease are in a large majority of cases tuberculous—namely, fibro-caseous. Thus Rolleston in the Goulstonian lectures in summarising the causes of Addison's disease says: "The first [fibro-caseous due to tuberculosis] is the only common cause of Addison's disease." The absence of any definite signs of tuberculous disease, glandular or respiratory, would point to the possibility of a similar lesion present in the case described. The cases as a whole present three main features of interest: (1) the age of the patients; (2) the family character of the disease; and (3) the low blood pressure in each case. How rare the condition is in children under 13 years of age has been shown, as mentioned by the statistics of Felberbaum and Fruchthandler.

The family character, if we accept Cases 2 and 3 as true cases of Addison's disease, is even more rare, for the only published record is that given by Fleming and Miller,¹ where a mother and four of her children were affected.

Before discussing the third main point of interest—namely, the low blood pressure—it may be well to review briefly some features in regard to the structure and function of the suprarenals as detailed by Rolleston. The suprarenals are composed of (1) the cortex derived from the coelomic epithelium in the Wolffian ridge and closely related to the sexual glands; and (2) the medulla derived from the sympathetic and neuro-ectodermal in origin. As regards the cortex, our imperfect knowledge of its functions points to the following inferences: (a) that it is related to growth and development; (b) that it has the power of neutralising poisons; and (c) that it is in some way concerned with the internal secretion of the medulla. The function of the medulla is briefly to produce an internal secretion which raises blood pressure by acting on the terminals of the sympathetic.

In the light of this knowledge it is significant to note that the main symptoms in Case 1 are (a) pigmentary changes, (b) asthenia and prostration, and (c) low blood pressure, while in Cases 2 and 3 the pigmentary changes are not so marked but the blood pressure is low. A study of these facts seems to suggest that in cases similar to Case 1 both cortex and medulla are affected, while in those similar to Cases 2 and 3 the medulla is primarily affected and the cortex only slightly so. This view of the cases is in accordance with a hypothesis formed by Dr. Gibson in his address to Guy's Hospital Physical Society in 1907. He says: "It may be supposed that Addison's disease may result in part

from inadequacy of the medullary portion leading to asthenia and hypopæsis by loss of adrenalin and by failure of the cortex to maintain certain as yet unknown, but probably important, functions particularly associated with pigmentary excretion and toxin destruction. On this view there may be a most variable assemblage of symptoms, according as the one or the other portion of the suprarenal body is mainly affected." It is impossible to dogmatise from three cases, but as a pure speculation this hypothesis is interesting and the cases recorded appear to add weight to it.

It would be interesting to know in what part of the suprarenal gland the tuberculous lesion begins in Addison's disease—namely, whether the cortex or the medulla is first affected and whether the course of the symptoms is affected differently according as to where the lesion commences. So far as I am aware no definite observations on this point occur in literature, the reason probably being that by the time the cases reach the post-mortem table the lesions involve the whole gland.

The value of a knowledge of the blood pressure in the disease has been emphasised recently, and Cases 2 and 3, if accepted as Addison's disease, would appear to show that it may be possible to still further differentiate types of the disease and to diagnose a case as such when the pigmentary changes are slight or absent. Stursberg has recently published cases in which the diagnosis of Addison's disease has been made with an almost entire absence of pigmentation and Cases 2 and 3 with low blood pressure, general weakness, and slight gastric and intestinal symptoms may well be associated with the characteristic lesions of the disease.

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INJURY TO THE SEMILUNAR CARTILAGES OF THE KNEE.

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SINCE William Hey more than a century ago published¹ an essay on "Internal Derangement of the Knee-joint," surgery has advanced so materially that knowledge of its pathology through operations on the living has now cleared up many things that were then matters of opinion only. The knee-joint has not escaped this advance, but it is because I think it is possible to make a further small addition to our definite knowledge that I am induced to offer these notes based upon 75 cases operated upon by me from January, 1900, to September, 1908, on injury to the semilunar cartilages.

It appears to be assumed by authorities that the pathology of these cases is mainly a displacement of the semilunar cartilage, due to a relaxation of the coronary ligaments, but the cases I have seen and operated upon prove that this is wrong, that the injury is a fracture of the cartilage and that displacement without fracture, if it occurs at all, is a very rare event. Of the 75 cases 27 were split longitudinally, 14 were split and detached circumferentially, 8 were split transversely across, 6 were split both transversely and longitudinally, 5 were reported loose, and 5 were reported rupture only. Of 10 the pathology is not recorded. (See illustrations, Figs. 1 to 6.)

It is of some importance to note that all of the cases reported loose were done in the period when I was unable through an imperfect incision to make a satisfactory examination of the joint, and that during the last two years, since the fact that fracture of the cartilage was the true pathology occurred to me, no case of my own or of my colleagues has been seen to refute this view. On a recent occasion I would have missed the fracture had I not been convinced that it could be found if searched for; and found it was, but so far back in the joint that it could not be seen even

¹ Brit. Med. Jour., April, 1900.

¹ Practical Observations in Surgery, 1803.