A Clinical Lecture ON RENAL DROPSY.

DELIVERED AT THE ADDENBROOKE'S HOSPITAL IN
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BY

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[The first part of the lecture consisted in the diagnosis of the case as one of Chronic Parenchymatous Nephritis, and in the discrimination of this from other diseases of the kidney, illustrated by pathological specimens: this part is omitted.]

On March 7th, 1918, was admitted L. G. H., aged 19, complaining of swollen legs and body, dating back to the previous August; also of dyspnoea, nausea, and vomiting—he vomited last the morning before. Nothing in the previous history worth recording, except the absence of any recognized infection, and a positive story of several severe wettings and chills before he fell ill. He presented the usual aspect of renal dropsy: face puffy and pallid; attitude propped up in bed; some cough and shortness of breath; body and legs enormously swollen—face, back, loins, legs, and private parts—and pitting deeply on pressure everywhere. Heart probably large and dilated; some crepitations in the backs of the lungs. Systolic pressure about 140. As pink spots with some dew upon them had appeared on the legs it was decided to tap the legs at once. After careful disinfection and greasing of the skin Southey's trocars were introduced into both legs and during the next three days about 45 pints of water drained off, with great relief; the penis and scrotum also were punctured and drained. No local irritation of importance followed these measures. The urine contained much albumin, and "many granular casts were seen under the microscope." The quantity was deficient. Vomiting recurred occasionally.

This phase of the illness lasted till May 11th. Medicinal means of reducing the dropsy had failed. A free flow of urine had been obtained by various gentle diuretics, for many days reaching some 45 to 50 oz. in twenty-four hours. Diaphoretics, including hot air baths, laxatives, and the rest of the ordinary means, were tried in vain; the dropsy reaccumulated to the fullest extent after each tapping, although at four or five operations large quantities of water had been successfully drawn off.

At the bedside, Gentlemen, I impressed upon you, and now I repeat, that this huge anasarca is not, as generally supposed, a mere mechanical overflow of water into the subcutaneous tissues because of an obstructed filtration from the kidneys. At first this obstruction had perhaps something to do with the condition, but by a little management, and by means of sundry diuretics, we found no great difficulty in bringing about quite a good urinary flow. Moreover, as you have seen, we were able to supplement any defective renal filtration by large discharges of water in other directions. You will remember that, out of the patient's hearing, I had led you to form no great expectations of success by these means, for the secret of relief did not lie there. For the most part we were expelling plenty of water from the patient's system; yet the enormous dropsy continued as bad as ever, and after each tapping returned in all its volume within a few days. On the other hand, I told you that in cases of anuria of eight or ten days' duration no oedema appears. I argued that the fault lay in some abnormal holding up of the water by the tissues, probably by some physical, or chemico-physical perversion. We speculated on osmotic relations (Starling), on acidosis (Fischer), on various perverse affinities, on surface tensions, and we tried methods calculated to check such aberrations, attempts in which we had the valuable co-operation of Mr. Sydney Cole of our biochemical laboratory. The saltless diet was tried; the patient's system was alkalinized for a time, a Blaud's pill was given with each meal; but all in vain.

Happily one morning I received a paper from Dr. Epstein of New York, whom I had met at the Sinai Hospital some years ago, and who had since kindly sent to me his papers, which chanced to be on the very subject of the causation and alleviation of dropsy in chronic

parenchymatous nephritis.¹ From what I thus learned of Dr. Epstein's researches I determined at once to make a great change in the diet of the patient. Many of you know that I have not been in the habit of strictly reducing the protein diet in these cases, but rather of allowing such protein diet as the patient seems to require, remembering that these patients are always anaemic, and under the anasarca always emaciated. But Dr. Epstein's diet had gone far beyond any liberty I had taken. It was as follows: Lean meat, lean ham, whites of eggs, (oysters), (jelly, lentils, split peas), green peas, rice, (oatmeal), skimmed milk, (coffee), and tea; salt in moderation, and fluid as reasonably desired. We allowed also fish, an ounce of bread, and half a pint of unskimmed milk. The articles in brackets we omitted.

Unfortunately the patient had hardly begun the new diet when he was attacked by something like influenza; the throat was sore, the head ached, and the temperature rose—an additional burden which almost made an end of him. All through June he was very ill. However he weathered this storm, and the diet was pressed on again. Fortunately it was acceptable to the patient. During July his progress was remarkable; by July 24th a steady fall of the anasarca had been recorded, and on that day it was noted that the swelling of the legs and thighs was less than it had been at any time since admission; less, indeed, than after the tappings, although the catarrhal attack was not quite over and a cough and muco-purulent spitting still distressed him. On July 27th he had been up on a couch and was feeling much better than he had been since his illness began. August 2nd: "Legs practically free from oedema; general condition good. He passes up to 90 and 100 oz. of urine a day, which however is still very albuminous." On September 10th he was still on the Epstein diet, with excellent result. "He is not now passing an excessive quantity of urine, but there is no sign of dropsical fluid anywhere in the body." A cursory examination of a drop of the urinary deposit which I placed under the microscope showed nothing important save one epithelial cell (renal). On September 28th he received his discharge; not of course as cured, but as entirely relieved of his burden.

And now, Gentlemen, with Dr. Epstein's help, I will try to explain to you the reason of the method which in this case—a severe one of its kind—has proved so successful.

First of all, you must understand that I am speaking only of chronic parenchymatous nephritis, and that what I have to say about this disease does not apply directly to any other disease of the kidney—not, for example, to the primary contracted kidney. This being understood, we will turn to our dietetic treatment—from Galenism to biophysics. You have seen that the successful diet was high in protein value, low in fats and carbohydrates, being thus the contrary of the ordinary diet in this disease. Yet it seems to have been brilliantly successful. How? You have seen also that in this case the dropsy was not due to deficient flow through the kidney. Again, as to the salt retention, while admitting that this is probably important, we remark that salt retention is found also in renal diseases in which there is no dropsy, or but little. Now in his researches Dr. Epstein has found that, in the disease before us, the blood is very poor in proteins; moreover, that of these the globulins are in great excess, sometimes in extreme instances making almost all of the protein volume.*

It seems highly probable that this poverty of the blood in proteins is due to the very large losses of albumin by the urine which are characteristic of this particular disease, and that this impoverishment of the blood is proportionate to the degree of the albuminous discharge. For instance, such a patient may thus lose 25 grams a day, or more, of albumin, while the average total content of the blood is but 210 grams. It may be replied that the poverty is but apparent and due to the aqueous dilution (hydraemia), but this source of error has been controlled. What then must be the effect on osmotic pressure of this impoverishment of the colloid content of the blood? Professor Starling has calculated that the normal osmotic pressure of the blood is about 4 mm. Hg per 1 per cent. protein, and thus the passage

* I have tried to ascertain the normal proportion, but failed. At last I find in Luciani that the relative proportions of serum and globulin are difficult to determine, and in fact have not yet been determined.

c^f saline water is, of course, from the tissues to the blood vessels. Should the protein or colloid content of the blood fall sufficiently, the drift may well turn the other way; and the tissues attract the water and hold it. Accordingly, if we analyse the dropsical fluid, we shall find it to be little more than this saline water, its protein content being practically *nil*; the tissues, indeed, may become stronger in colloid content than the vessels, and the osmotic relations be quite upset. If, for control, we take a dropsical effusion from a case of primary contracted kidney, or of cirrhosis of the liver, we shall find it to contain as much as, or more than, three parts of protein per 100 c.cm. (Epstein). You see then how idle it is under these conditions to rely on expelling the dropsy by diuretics, diaphoretics, and the like; we can expel but the surplus after the tissues have saturated themselves.

In the next place, Dr. Epstein discovers a great excess of lipid in the blood of these cases. This he attributes in great part to starvation; and if derived largely from mobilization of fat, it is also a product of breaking down of the tissues. Now abstraction of lipoids from tissue cells causes imbibition of fluid with consequent swelling of the cells—cell oedema.[†] The diet for this dropsy is therefore largely exclusive of fat.

Once more: I need not remind you that one main product of carbohydrate food is water; we may fear therefore that large amounts of such food may promote the accumulation of water in the system. Moreover we desire to make room rather for the protein constituents, and to secure their full assimilation without rivalry.

These, then, are Dr. Epstein's principles, which, you will admit, seem reasonable, and are based upon careful research. Further experience only can establish or invalidate his conclusions. So far as our one case goes you have seen that the results—we are tempted to say the consequences—were very striking. In conclusion I have to acknowledge the able assistance of our house-physician, Mr. Gittens, in the management of the case.

To bacteriology, Gentlemen, we owe indeed an unspeakable debt; but you have heard me say again and again that, this territory being largely won, we have now to look for the next great conquests to biochemistry and physical chemistry. But these and such advances demand a very different system of clinical investigation from that we now have in England; experience is good, sagacity is good, but now we have penetrated so far that chairs of clinical research, armed with skilled staffs and laboratories, must be established in tardy England as already they are provided in every considerable medical school in Scotland and on the European and American continents.

REFERENCE.

¹ A. A. Epstein: *Amer. Journ. Med. Sci.*, November, 1917; and *Journ. Amer. Med. Assoc.*, August 11th, 1917.

ACUTE ULCERATIVE GINGIVITIS.

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DURING the past year or two a number of observers have written on the subject of acute ulcerative gingivitis. Little attention has, however, been paid to the signs on which an early diagnosis can be made, while no facts have been available regarding the extent to which the disease is prevalent among our armies in France. With regard to treatment, chief emphasis has hitherto been laid on the local use of drugs having a more or less specific action on the organisms found in this condition.

In the present paper, which is a modification of a report drawn up at the request of certain army medical authorities, special attention has been given to the methods of early diagnosis and the incidence rate in troops in France. The treatment has been dealt with on more general surgical lines than in previous communications, less reliance being placed on the action of anti-spirochaetal drugs, and more on thorough surgical cleansing of the infected areas. This method of treatment has been adopted as the result of personal experiences dating from March, 1915, when I saw for the first time cases in France.

[†] On the harm which cholesterol may do to the body see Dr. Epstein (*loc. cit.*), and my *Diseases of Arteries*.

(In passing, it is interesting to note that between November, 1914, and March, 1915, no cases came under my notice.) Since March, 1915, numerous cases of the disease, in all stages, have come under my personal care.

Signs and Symptoms.

Acute ulcerative gingivitis is an acute inflammatory condition of the margins of the gums, which spreads rapidly, and leads to sloughing of the interdental papilla; sloughing of the gums around the necks of the teeth; ulceration of the adjacent mucous membranes (in the more severe cases); rarefying osteitis and destruction of the bone around the teeth, in chronic cases.

The general symptoms are as follows: Malaise; slight elevation of temperature, rarely over 100° F., but occasionally 102° F.; glandular swelling, the submaxillary glands being chiefly affected; general depression.

The local symptoms are: Haemorrhage from the gums, either spontaneous, or as a result of pressure; pain in the gums, especially at night, and often so severe as to make sleep impossible; a foul taste; very offensive breath; mastication difficult and extremely painful, the teeth becoming very tender and loose.

Clinical History.—The onset is gradual. A small area of gum becomes painful, and this pain spreads to the interdental papillae of the adjacent teeth. By the seventh to the tenth day the pain and local symptoms are severe, and the patient may report sick.

Local Signs.—In the earlier stages the only signs are a slight oedema of the interdental papillae; these rapidly become congested and are bluish-red in colour, and are distinctly swollen; if touched they bleed readily. Later the gums appear to be separated from the teeth, this being due to the swelling and not to any destruction of the periodontal membrane. This stage is generally present for two or three days. An acute necrosis of the papillae then follows. The slough formed is non-adherent, of a characteristic brownish colour, and very friable. There is no attempt at formation of a true membrane until the process of healing has commenced. If left untreated, sloughing gradually spreads until a considerable area of gum is destroyed. The gums in the infected region are deeply congested. The teeth are usually loose.

Distribution of the Lesion.—The gums around the upper incisor teeth are commonly affected first, but in a number of cases the disease commences in other situations; an erupting lower third molar, for instance, is a common site, or any spot where food tends to lodge. When one jaw is affected, the other soon shows signs of infection in the corresponding situation. The spread is usually in direct continuity along the gum, the interdental papillae on either side of the primary lesion becoming attacked. It is rare for a large area of gum to slough before several teeth have become implicated. Occasionally, on the other hand, the lesion begins at distinct points.

Pathology and Bacteriology.

In the early stages the tissues attacked by the specific organisms become deeply congested, and after two or three days necrose, making a series of cup-shaped depressions between the teeth. The sloughing does not extend deeper than the bottom of the periodontal sulcus. At first there is little or no bone destruction, although this is to be found in advanced or chronic cases. Tartar is always present on the necks of the teeth exposed by the sloughing of the gum.

[*Note.*—In periodontal disease (pyorrhoea alveolaris), which is the disease most commonly mistaken for acute ulcerative gingivitis, the gums become slightly congested, but *do not slough*. Ulceration proceeds at the bottom of the periodontal sulcus, thus destroying the periodontal membrane. This is accompanied by a rarefying osteitis, which finally leads to the destruction of the bony tooth socket. The gum is not destroyed as quickly as the periodontal membrane, and so a pocket of varying depth is formed between the tooth and the gum. In severe cases this pocket is filled with pus. Periodontal disease is extremely chronic, and in contrast to acute ulcerative gingivitis, may be present for ten or fifteen years.]

The characteristic organisms found in films are a mixture of *Bacillus fusiformis* and spirilla. In well-marked cases, especially when occurring in a clean mouth, these may appear to be present in almost pure culture, giving a very striking picture under the microscope. In other instances where periodontal disease or simple gingivitis is present