LOWER NEPHRON NEPHROSIS.

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DURING World War II attention was refocused upon a condition which had been described following the previous World War, but which had been neglected during the interval. This is the syndrome variously described as crush kidney, ischaemic compression nephrosis, or hemoglobinuric nephrosis. It was noticed by Bywaters in air raid casualties, and in such instances was associated by him with the precipitation of the muscle pigment, myohemoglobin, in the kidney tubules of patients who suffered prolonged crushing injury of large muscle masses. Since then numerous publications on isolated instances and groups of cases have clearly substantiated the correctness of the clinical pattern and pathological picture described by Bywaters. They have also enlarged the scope of the syndrome to embrace conditions which, although not owing to crush and release of myohemoglobin, involve precipitation of the hemo-compounds, abnormal proteins and drug crystals in the renal tubules with a sequence of clinical and pathological events similar to those of the "crush syndrome".

This condition is induced by a variety of circumstances, which have sometimes little in common, except that they share a similar type of renal pathology. These inciting causes include such diverse processes as hemolytic transfusion reactions, myohemoglobinuria, sulphamamide intoxication, burns, blackwater fever, mushroom poisoning, arterial thrombosis, uteroplacental infarction, and alkalosis. In all these there is circulation in the blood of some abnormal substance which has a low renal threshold value, and is eliminated rapidly by the kidney, where it is precipitated in the tubules. Subsequent to appearance of the substance, such as myohemoglobin, hemoglobin, or sulphamamide, within the circulation and its excretion into and plugging of the renal tubules, a similar clinical picture ensues in each instance. There is oliguria, progressing to anuria, associated with casts and traces of albumin in the scanty urine, which is of low specific gravity; the onset of anuria is usually within 24 hours. After a few days the patient manifests the symptoms and signs of uremia, which is substantiated by the raised blood urea, uric acid and elevated serum potassium, with depression of the alkali reserve. Vomiting, pulmonary edema and raised blood pressure are frequent. Unless relief is procured from the anuria death ensues by the tenth or eleventh day after onset. The course is unpredictable, and does not respond uniformly to any type of treatment, of which there are many; but if urinary excretion does not commence before the seventh day the issue is fatal. A synopsis of a case history is included as an illustrative example of a typical course.

CASE OF LT. T. G. (P.O.W., Germany)

The patient was wounded in action, when he suffered multiple gunshot wounds, with the following distribution:

1. G.S.W. right tibia and fibula, middle and lower third, severe;
2. Comminuted compound fractures, left foot, metatarsals and phalanges;
3. G.S.W. left arm, lacerated, severe.

He was admitted the following day to an advanced hospital unit, where both pulse and blood pressure were unobtainable. Within five minutes transfusion was com-
menced with a pint of whole blood (Group O), of which a further two pints were administered during the next twelve hours. There was no transfusion reaction. In addition there were also given 1,800 c.c. of plasma, 720,000 units of penicillin and 20,000 units of tetanus antitoxin. No sulphad drugs were given. During this 12-hour period the blood pressure reached 112/72 mg. Hg., and the pulse rate averaged 112-132 per minute. Fourteen hours after admission he voided 400 c.c. of dark urine. The next day a guillotine amputation of the right leg was performed. On the sixth day after injury the patient arrived at a general hospital, when he was amnior, irrational and restless. He was given glucose intravenously, 500 c.c. of whole blood and 2,000 c.c. of plasma. The next (seventh) day he was catheterized and 30 c.c. of urine obtained, which contained a few granular casts, albumin ++ +, and had a specific gravity of 1012. The blood urea level at this time was 180 mgm. per cent. He expired late on the seventh day.

An autopsy was performed. The pertinent pathology was in the lungs and kidneys. The lungs were heavy, rubbery in consistency and exuded a plentiful frothy pink fluid; microscopically there was extensive pulmonary edema. The kidneys weighed over 200 grams each, were pale, and bulged through the cut capsule. In the medulla there were many fine, greyblack streaks, which were identified by low power microscopy in the fresh specimen as pigment casts. Histologically the changes in the kidney were typical of a severe lower nephron nephrosis, and included degeneration of tubules, pigment casts in the distal convoluted and collecting tubules, interstitial edema with considerable round cell infiltration.

This condition has been reviewed and admirably discussed by Lucké, who has exhaustively surveyed the literature and added 583 fatal cases encountered in army personnel. He includes this disease of varied exciting etiology under the comprehensive name of "lower nephron nephrosis", which avoids the restrictive limitations of the previous titles. In his work Lucké quotes the personal communication of the figures and incorporates the material analyzed by Angevin and Harman which were derived from cases of the syndrome encountered in autopsies performed upon military personnel in the European theatre of war. The inclusion of these in such a comprehensive survey, however, does not permit the clarification of certain lessons, which closer study of the more limited series might permit. For this reason the results of experiences with the categories of cases encountered in battle casualties are presented here.

The tissues of 3,223 autopsies were studied grossly, when so available, and invariably by histological technique. Of those autopsies 1,065 were battle casualties who died in hospital subsequent to evacuation from forward units, and exclude deaths in battle and in forward echelons. The survival time of such patients was, therefore, sufficient to permit manifestations of the clinical features of the syndrome. This exclusion of rapidly fatal casualties enhances the significance of the incidence of the syndrome by rendering the series more comparable to the type of traumatic case encountered in a large civilian hospital.

The material at our disposal, which rarely included fresh specimens, was usually fixed in 10 per cent. formalin and was accompanied by protocols and clinical summaries composed by the individual prosectors. Despite the individual variability in the composition of the histories, they were adequate in most instances to permit a clinical assessment of the degree of renal insufficiency. The clinical data usually included the results of laboratory studies on blood and urine constituents, when pertinent alterations were found. Care was taken in each case to determine if blood transfusions and sulphad drugs had been administered, because of the importance of these therapeutic measures in the etiology of the syndrome.

Histological studies were made on the tissues in all cases, by examina-