"FIELD NEPHRITIS" IN THE GERMAN ARMY

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NEPHRITIS as a disease of active service first came into prominence during the American Civil War, when about fourteen thousand cases were recorded. During the last war the disease assumed serious proportions on both sides. In the British Forces it commenced on the Western Front in the spring of 1915. In the German army it commenced in 1916 on the Eastern Front, and only later, and to a lesser degree, affected the armies in the West.

During the second World War nephritis never became a serious cause of wastage amongst the American or British Forces, but the German army appears to have suffered much more severely than during the previous conflict. The disease commenced in the East about the time of the opening of the Russian campaign. Until June, 1941, the monthly incidence of nephritis in the field army had fluctuated around 0·4 per 1,000. From then onwards the curve of incidence rose steadily, to reach a peak in March, 1943, of just over 1·4 per 1,000; this was followed by a decline during the summer, and a further rise during the following winter (fig. 1). In the Balkans cases first occurred in large numbers during the winter of 1943-44, but this outbreak was not nearly so severe as that which followed in the earlier months of 1945, during the retreat from Greece. Between February and April of the present year more than 1,000 serious cases were admitted to one hospital in Zagreb alone.

The majority of German authorities regard the "field nephritis" of this war as an entity, distinct from the acute nephritis of civil practice. Some are inclined to the view that it may be a "new" disease with only a superficial resemblance to the "war nephritis" of the last war. Gutzeit, principal consulting physician to the German army, terms it "infectious nephritis" and describes it as such in a military handbook [8]. Another writer, however, in summing up the views of various authorities [4], remarks that "it is quite plain that many clinical observations made during the present war and regarded as new had been recognized in the first World War."

Facilities for medical research were much more limited in the German than in the British
Army, and although much has been published on "field nephritis" the biochemical and pathological evidence is very incomplete. The scope for speculation was correspondingly wide. Despite the emphasis laid by German physicians on certain features which, they insist, give a distinct character to this malady, its resemblance to the acute nephritis which affected the British Armies on the Western Front in the last war is very close. Bradford, in his account of the latter disease in the Official History of the War [3], expressed the view that it was merely the nephritis of civil practice seen under war conditions, and that its peculiarities were due to the hardships to which its victims were exposed. There is no convincing evidence that the case of field nephritis is in any way different.

**Clinical Features.**

Bradford's description of the nephritis which he saw in France from 1915 onwards coincides very closely with the clinical picture of "field nephritis."

The basic syndrome of field nephritis consists of dyspnoea, oedema and albuminuria.

The onset was usually sudden, but might be gradual. Most patients gave a history of having been perfectly fit until the typical symptoms began, but many upon questioning recalled a period of feverishness some three or four weeks previously. The first symptoms were those of dyspnoea or oedema. The onset of dyspnoea was often remarkably sudden, usually in the course of physical effort, the patient being seized with such intense shortness of breath that it was impossible for him to carry on. This symptom was so frequent and characteristic that the condition was often recognized at the outset by the soldier's comrades.

In other cases the disease began insidiously, and the shortness of breath was worst on lying down, interfering with sleep [6]. The onset of oedema was, however, the most usual symptom causing the patient to report sick, being present from the beginning in four out of five cases.

It generally began rather gradually, in which case it was usually first noticed in the ankles, becoming gross and more typically nephritic in distribution in the course of the next twenty-four or forty-eight hours. A dry cough was often present at the beginning, but sore throat was conspicuous by its absence. Headache, sometimes very severe, was a frequent symptom, which in many cases persisted for several weeks. Vomiting was not very common, and there was seldom any marked loss of appetite.

When first admitted to a medical unit all degrees of the condition might be seen, from mild dyspnoea and transient oedema to patients critically ill with severe pectoral and cardiac distress, with anasarca, or even—and not very uncommonly—uraemic coma with convulsions. To a certain extent the severity of the condition depended directly upon the handling of the case; the effect of transportation over any distance was invariably bad, and patients who commenced a train journey with only mild dyspnoea and no oedema often developed severe waterlogging before reaching their destination. This was attributed partially to the physical conditions of the journey and partly to lack of control over the patient, who could not be kept on the strict regime of absolute starvation which was considered so important.

About one-third of all cases were febrile during the first day or two after admission to hospital. The oedema was generally moderate in degree, and ascites was uncommon except in cases with generalized anasarca. In severe cases pleural and pericardial effusions occurred, and pulmonary oedema, which might be partially interstitial in type with a radiological picture similar to "virus pneumonia" (presumably the same as our atypical primary pneumonia), was found occasionally. Soft swelling of the spleen was said to be an invariable feature of the disease.

The blood-pressure was usually raised to a systolic reading of 140-200 mm. and a diastolic of 100-120 mm. These figures were seldom exceeded; and the systolic pressure was more generally under 180 mm. than above. Bradycardia was the rule, and was regarded as an important distinguishing feature from cardiac insufficiency in cases without albuminuria. Capillary pulsation was marked.

\[1\] Rudisser[13] stated that the diastolic pressure was lowered in some cases.
The daily urinary output was always diminished. Anuria was not very rare, and not always of serious import. At the outset the specific gravity was often fixed at 1,010, the urine being strongly acid. There was generally a heavy albuminuria, up to 20 to 30 Esbach units per 1,000, and this was usually very persistent; but, as mentioned below, cases without albuminuria also occurred. Erythrocytes were generally present but not in great numbers, although in serious cases gross haematuria was not uncommon and sometimes persisted for a week or ten days. Blood casts were practically never found in uncomplicated cases, and epithelial and granular casts seem to have been less frequently present than would have been expected in ordinary acute nephritis.

The blood showed a transient polymorph leucocytosis with a shift to the left in the Arneth count, followed by a lymphocytosis. In the initial stages there was a monocytosis with the presence of plasma cells. Some degree of anaemia developed sooner or later in most cases, but was usually proportional to the loss of red blood corpuscles in the urine. In protracted cases an anaemia of toxic origin supervened, and in some patients with marked oedema the anaemia was attributed to direct injury to the bone-marrow.

The blood showed a diminution in albumin and a very marked increase in serum globulin. The total blood protein figure was increased, and this feature was regarded as characteristic. Unfortunately only a few cases appear to have been investigated fully. Non-protein nitrogen was increased in severe cases, but the urea and uric acid levels were only raised in cases with the greatest renal damage. The blood sedimentation rate was generally increased.

The oedema fluid was very rich in albumin and relatively weak in globulin. In many cases the oedema fluid coagulated spontaneously when drawn into a test tube.

**Varieties of the Disease.**

The characteristic triad of dyspnœa, oedema and albuminuria was only found in fully developed form in something less than 50 per cent of cases [14]. In about 10 per cent the urine showed no abnormality, the only signs being oedema and a raised blood-pressure; these cases were termed "nephritis ohne nephritidis"—nephritis without kidney inflammation. In other cases, which were however rare, the blood-pressure was normal or there was no oedema while in other respects the picture was complete.

**Complications.**

On the Eastern Front in 1943 as many as 30 per cent of cases occurred in combination with trench fever [14], which was very common in the German army. Other cases were complicated by typhus; in such circumstances the mortality was naturally high.

The commonest complications were cardiac failure from pericarditis or myocarditis, cerebral oedema with convulsions, true uræmia and pneumonia. The last was supposed by some to be the same as "virus pneumonia," but the evidence on this point is not convincing.

**Course and Prognosis.**

The case mortality was not high. A pathologist stated that of about 800 cases which he had seen during the Balkan campaign (when the disease was particularly severe) 20 died, but 8 of these were suffering from typhus in addition. The outlook in all cases depended principally upon the facilities for hospitalization and treatment.

Pilgerstorfer [11] gives the following results, based on two years' experience in a hospital for field nephritis in Vienna (number of cases not stated):

<table>
<thead>
<tr>
<th>Complete cure</th>
<th>Per cent</th>
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<tbody>
<tr>
<td>Trace of residual albuminuria or haematuria, still under treatment</td>
<td>21.8</td>
</tr>
<tr>
<td>Discharged with persistent traces of blood and albumin</td>
<td>11.6</td>
</tr>
<tr>
<td>Left with unimportant elevation of blood-pressure</td>
<td>3.8</td>
</tr>
<tr>
<td>Residual damage to the kidneys</td>
<td>3.6</td>
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<tr>
<td>Transition to chronic nephritis</td>
<td>1.2</td>
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With rest in bed and absolute starvation the edema usually disappeared in a surprisingly short time. Estimated quantities of edema fluid of up to 20 litres had completely gone in less than a week. The average duration in hospital varied from a few weeks to several months. It was generally four or five months before a case was fit for duty, and even then he was generally placed in a low medical category and employed on home establishment. Cases with persistent albuminuria were never sent back to the line, but if a patient remained free of albumin and had no relapses for six months he was generally considered fit for front-line service.

The ultimate prognosis may not be so good as the figures given above may appear to suggest. Pilgerstorfer [12] cites Gros, who in 1928 followed up 211 cases of trench nephritis from the last war. He found that 24.7 per cent had chronic nephritis (including 9 per cent with "contracted kidneys") ; 26 per cent had residual signs in the urine and nothing else; 44.6 per cent were free from signs or symptoms.

**TREATMENT.**

Rest in bed, warmth and absence of movement were the first essentials. The deleterious effects of transportation have already been noted.

In the first three days food and drink of every kind was absolutely forbidden, luminal being given freely to allay hunger and thirst. This regime was continued for as much as eight days if necessary. It was followed by a salt-free diet consisting of fruit, rice and potatoes, with butter prepared by kneading in water to reduce its salt content, the fluid intake being restricted. The renal function was estimated by Volhard's method from time to time, and if the results were satisfactory salt was gradually added to the diet, beginning with 3 grammes per diem. Some physicians considered that the flushing of the kidneys in Volhard's test had a beneficial action, and repeated the test at frequent intervals partly as a method of treatment [7].

In the early stages circulatory distress was relieved by giving strophanthin once or twice daily, 1/4 to 1/2 mgm. intravenously, and venesection, from 400 to 800 c.c., was repeated once or twice at intervals of three or four days. Complications such as coma and " eclampsia " were treated in orthodox fashion. In cases in which edema persisted for several weeks testosterone, thyroxin and potassium acetate were exhibited with apparent good effect. The Vienna school employed urea and potassium acetate in the acute stages, but this was not favoured generally.

**MORBID ANATOMY.**

Pathological material is scarce, but we were fortunate enough to obtain sections of kidneys from four fatal cases. These were examined by Colonel H. L. Sheehan, to whom we are indebted for the following report:—

"Death was stated to have occurred in these cases after 2, 3 1/2, 29 and 33 days. The kidney lesions are very similar to those of trench nephritis in the last war. The condition is a true glomerulitis, not a simple famine oedema.

The earlier specimens (2 and 3 1/2 days) show a very early acute glomerulitis which might easily escape notice on cursory examination. The glomeruli show undue lobulation of the tufts which are somewhat swollen and occasionally show pouting into the tubule lumen; these changes are more marked in the second specimen. There is a slight increase in the number of endothelial nuclei in the glomerular loops. The arterioles and tubules appear normal and contain no casts.

In the third specimen (29 days) the glomerulitis is more advanced, and the lesions are quite obvious. The glomerular tufts are all swollen and show a considerable increase of endothelial nuclei. There are numerous light non-cellular adhesions between tuft and capsule. The convoluted tubules are moderately dilated and a few contain hyaline casts. The disease was presumably progressing up to the time of death. If the patient had recovered there would have been some permanent histological lesions in the glomeruli."
The last specimen (33 days) shows what appears to be an almost fully recovered glomerulitis. Many of the glomeruli are apparently normal, others show a slight increase of endothelial nuclei and occasional light adhesions to the capsule. The first convoluted tubules are moderately dilated. Hyaline casts are present in a number of second convoluted tubules and in a few of the collecting tubules. The acute disease seems to have lasted only a few days, healing apparently having been in progress for three or four weeks. (This specimen also shows gross haemorrhage into many nephrons and some interstitial cellular infiltration, probably due to some intercurrent disease causing death; possibly typhus.)

The histological appearances seem to be very similar to those described by Shaw, Dunn and McNee in the trench nephritis of the last war.”

Ætiology.

The great majority of cases amongst our troops in the last war occurred in individuals who had no preceding disorder of the kidneys. Exposure to cold and wet was a factor in practically every case, but occasional instances were recorded in which hospital orderlies and nurses living under good conditions developed the disease. No definite evidence could be adduced in support of the infective theory, and the remarkable freedom from nephritis of Indian troops on the Western front, under conditions which produced large numbers of cases of bronchitis, pneumonia and trench foot amongst them, was considered by Bradford as an argument against the disease being due to an infection [3].

On the German side in the last war some writers (Müller, Jungmann et alia) were definitely of the opinion that the condition was due to a specific organism, others considered that wet and cold were the principal factors, others laid stress on focal sepsis or dietary deficiencies. One clinician (Töpfer) suggested that a relationship existed between lice, trench fever, war nephritis, and exposure, and this opinion was resurrected during the recent conflict [4].

Studying the literature on field nephritis, one finds that the only ætiological factor which is beyond dispute is the importance of cold, damp and hardship. The disease was exceedingly
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rare amongst civilians, and few cases appear to have occurred except in formations in the line. Of the three factors mentioned, hardship was the most important. It was characteristic that when Divisions were transferred from the west to the east cases only began to occur in large numbers after two or three months of heavy duty in Russia, whereas there was no constant relationship (fig. 2) between the incidence of frost-bite and the incidence of nephritis; and in the Balkans, although the winter of 1943-44 was more severe than the following winter, there were not nearly so many cases of nephritis, a fact which was attributed to the hardships of the arduous retreat from Greece in the second year. There can be no doubt, however, that cold played an important part. One writer [10] lays stress on "cold trauma," and makes the rather naive statement that when questioned 94 cases in 100 always remember one particularly cold night.

The disease showed no particular age incidence; although the majority of cases in base hospitals appeared to be over the age of 35. This was explained by the statement that the disease was milder, and recovery more rapid, in the earlier age-groups, but the age distribution of the German army may have been responsible. The majority of patients were infantry soldiers, but all arms were represented, depending on the degree of physical hardship to which the individual had been exposed. The disease was uncommon in the Navy and the Air Force, but we saw one case, for example, in a member of the Luftwaffe who had been employed in the line as an infantryman.

Dietary deficiencies seem to have played no special part, and although many cases occurred in association with trench fever (up to 30 per cent on the Eastern Front in 1943) the louse can neither be implicated nor excluded on available evidence; all German soldiers admitted to hospital from the front line were lousy, and in the comparatively rare instances where nurses and orderlies developed nephritis the possibility of louse infestation can hardly be ruled out.

In many cases there appears to have been a transient mild pyrexia about three or four weeks before the onset of typical symptoms, but tonsillitis was not a feature. The vast majority of cases occurred in men who had no history of previous illness of any kind and most authorities agree that focal sepsis was never a factor. Where an acute nephritis was preceded by a sore throat clinicians insisted that these were cases of glomerular nephritis of classical type which must be distinguished from a true field nephritis. Such cases were relatively uncommon; the distinction, they admitted, was difficult at the bedside, but the two conditions differed widely in their epidemiology.

Graphs of the incidence of sore throat and nephritis in various formations offer considerable support to the contention that there is no causal relationship between the two conditions. Fig. 2 shows the incidence of nephritis, sore throat and frost-bite in an army on a southern sector of the Eastern Front from August, 1941, to June, 1943. Similar graphs for divisions transferred from the west to the east showed seasonal peaks of incidence of sore throat, which had no obvious relationship to the occurrence of nephritis, the incidence of which rose sharply in the third month after transfer to Russia whatever the season.

The theory that the condition was due to a specific organism (probably a virus) is widely held amongst German medical authorities. The evidence in favour of this view is as follows:

(a) In Norway, in particular, it was noted that the numerous cases occurred in certain units, while other units at the same time and under similar conditions of weather and duty remained completely free.¹

(b) Gutzeit [8] states that the curves of incidence of civilian cases and of cases in the army were closely similar, suggesting a seasonal wave of an infectious disease. He also records that he saw a considerable number of cases who developed nephritis while in hospital for wounds, about four weeks after leaving the front line.

(c) Cases occurred amongst medical orderlies, who had not been exposed to cold or wet, employed in hospitals where field nephritis was being treated; we saw one case, and were

¹Against this it might be argued that a similar observation could have been made in the Apennines last winter in relation to trench foot which nobody believes to be due to infection.
given details of another, in which the patient was a medical orderly in a special kidney centre, and developed the disease while living under good conditions.

(d) Certain storemen of the German Ordnance Corps are stated to have developed nephritis while employed in clothing stores, although they were not exposed to cold and wet.

(e) Clinically and at post-mortem some enlargement of the spleen was always present.

(f) Every third case had an elevated temperature at the beginning of the illness although it is a feature of the disease that it is not preceded by tonsillitis or focal sepsis.

Attempts to prove the infective theory by experiment were unsuccessful. Blood from cases of field nephritis was injected intravenously into healthy individuals without results [4], and efforts to transmit it to experimental animals also failed; but the amount of research carried out on these lines was very small. Rudisser, who quoted Russian doctors as having stated that a few cases had occurred in Russian civilians which they attributed to spread by infected water supplies, believed that the epidemiology of the disease was closely allied to that of infective hepatitis [13]. Others, on the basis of the radiological picture in cases with pulmonary œdema, tried to link it with "virus pneumonia": the two diseases occurred at the same time in some formations of the German army in Greece.

Allergy was, of course, invoked by some [6]. Others believed the disease to be a complication of pyodermia (Sylla, Korth and Pankow) and claimed to have found identical haemolytic streptococci and staphylococci in skin lesions and in the urine [4]. (In the last war Wilson found virulent organisms in catheter specimens of urine from fourteen out of a consecutive series of a hundred cases, but regarded them as saprophytes [3].)

**DISCUSSION.**

This paper has been based upon the available literature, which is sadly incomplete in many important details, discussions with German physicians and pathologists who have seen many hundreds of cases, and on clinical examination of the comparatively small number of patients still under treatment in the British zone in Austria. There have been no fresh cases since the middle of May, and it seems probable that the opportunities for first-hand study of field nephritis have now disappeared, perhaps for ever. We have thought it worth while to collect together what evidence we could obtain now, because the loss and destruction of records and data in the final débâcle was so extensive that it seems unlikely that much more of value will ever be forthcoming. Very little biochemical research was carried out at any time, and all clinical records of the cases in the Balkans at the beginning of this year were lost in the retreat of the German army from Jugoslavia.

Most German authorities regard this condition as not, primarily, a disease of the kidneys. To quote Schäfer and Reuter [14]:

"The basic cause of the disease was seen as a general capillitis which led to an increase in permeability. In many cases the capillary damage was limited to the peripheral circulation without the kidneys being involved. The pathogenesis of the dyspncea must include the question of a pulmonary inefficiency due to the increase of permeability of the lung capillaries as well as the question of cardiac power."

This theory has, of course, been put forward in relation to ordinary acute nephritis. The occurrence of cases of field nephritis without any abnormality in the urine, and of others in which massive œdema has been present long before the presence of albuminuria, and the high protein content of the œdema fluid, may be cited in support of this view.

The suggestion that the disease was due to infection with an unknown but specific organism will naturally be received with scepticism. British experience with the disease in "epidemic" form appears to have been confined to about three hundred Italian prisoners of war who were treated in British hospitals. These cases were indistinguishable, both clinically and pathologically, from ordinary acute nephritis [2]. The question of infectivity could only be settled, however, by making a detailed study of the epidemiology of the condition in the field, and by experiment, and there is insufficient evidence on either aspect to enable one to form an
opinion. It is obvious that if there is a specific organism it must be one which nearly always requires special conditions of hardship and exposure before it can produce the disease in recognizable form.

An analogy might, we suggest, be drawn between "catarrhal jaundice" and infective hepatitis on the one hand, and the acute nephritis of civil practice and field nephritis on the other. There seems to be good reason for believing that in each case we are dealing with a disease which assumes a special form under the conditions of active service. Just as the study of infective hepatitis has shown that its peacetime counterpart is due to a specific infection, so it may be worthy of consideration whether field nephritis does not provide a clue to the true nature of acute nephritis.

If it requires a war to produce the disease in epidemic form it is to be hoped that the nature of field nephritis will not be decided in our time; but acute nephritis is always with us.

SUMMARY.

(1) In striking contrast with British and American experience in the second World War, the German field army was seriously affected from the opening of the Russian campaign onwards by outbreaks of acute nephritis.

(2) The monthly incidence in the German field force on the Eastern Front rose steadily from less than 0.4 per 1,000 in June, 1941, to over 1.4 per 1,000 in March, 1942. Thereafter it continued to be a serious factor on the Eastern Front, and in 1943 it appeared in the Balkans. Fresh cases have ceased to occur since the termination of hostilities.

(3) The basic syndrome of "field nephritis" consisted of dyspnoea, oedema and albuminuria. The clinical picture was similar to that of trench nephritis in the last war. The case mortality was low and complete clinical cure occurred in about 60 per cent of cases; but there is reason to suspect that the ultimate prognosis may not be so favourable.

(4) Treatment was based on Volhard's hunger and thirst regime.

(5) Histological appearances in kidneys of fatal cases were similar to those in trench nephritis of the last war. Most German authorities believe, however, that the disease is essentially due to a general capillitis and is not primarily a kidney disease.

(6) Cold, damp and hardship were important aetiological factors. Tonsillitis or other preceding infectious conditions seemed to play no part. The evidence in favour of a specific infective agent is discussed.

Note.—The graphs were taken from a paper read at a German medical meeting in Austria before the war ended. The author stated that he had taken his figures from articles published by Assman [1], Dietrich [5], and Pilgerstorfer [11] which we have not, unfortunately, been able to consult in the original.

REFERENCES.