**THREE CASES OF PAROXYSMAL HAEMO-
GLOBINURIA IN SOLDIERS.**

BY

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IN view of the comparative rarity of this condition the following cases possess considerable interest. All three men were in the early stages of their military training and paraded sick owing to discoloration of the urine, which, coupled with a feeling of great exhaustion, caused them considerable alarm.

CASE I.

Private H., aged 18. No history of venereal disease. Woke up one morning after night operations feeling very weak and shivery, and noticed that his urine was red in colour. His temperature was 100.8°; no physical signs could be detected, except that the spleen was just palpable. The urine was dark brown and contained heavy deposits; it showed the presence of albumin, and gave a positive reaction to the guaiacum test. On microscopic examination a few red corpuscles, a quantity of amorphous pigment, and some bladder epithelium were seen. Spectroscope examination showed the absorption bands typical of methaemoglobin.

The patient was put to bed with hot-water bottles and given warm drinks. After twenty-four hours the urine was clear, and he felt quite well, though weak. He was given an iron and arsenic tonic, and returned to special duty in a few days. He had no recurrence while under my observation.

CASE II.

Private S., aged 32, had suffered from malaria in 1912 while in the tropics. He had led a sedentary life until enlistment. His attack started suddenly with severe lumbar pains after exposure to cold and wet.

On examination he had a temperature of 102.2°, which, however, quickly fell after rest in bed. There were no abnormal physical signs; the spleen was impalpable. His urine, which was examined both microscopically and spectroscopically, presented the same characteristics as in the preceding case, differing only in specific gravity. A blood examination showed a leucocytosis of 9,800. This man continued to pass blood-stained urine for forty-eight hours. He was given similar treatment, and was soon back at work. He is now in Mesopotamia, and when I last heard from him had had no recurrence.

CASE III.

Private J., aged 28, had had a similar attack previously in civilian life. On this occasion the onset was sudden, accompanied with great exhaustion. His temperature was 102° and the spleen just palpable. After being put to bed he complained of an intense "itchiness" all over. The urine had the same physical, microscopic and spectroscopic characteristics as previously described, and he also had a leucocytosis.

He recovered from this attack in twenty-four hours, but had a relapse about six weeks later. He was eventually put to clerical work and has not suffered since.

In all these three cases the exciting cause was apparent exposure to cold coupled with considerable bodily exertion. In none was there any admission of syphilitic taint. The Wassermann reaction was not done in any of the cases. On examining the cases admitted into Guy's Hospital during the last few years, I find that in the last three the Wassermann test was performed, and was positive in all of them. Malaria is cited by some authorities as an etiological factor, and Case II had suffered from this disease. It is interesting to note that in none of these cases was the initial temperature under 100°, and that in none of them did the urine clear up under twenty-four hours. This apparent severity was, no doubt, due to the considerable exposure and exertion undergone.

The most generally accepted theory of causation is that a haemolysis exists in the blood of these cases, the amoebocyte of which will only unite with the red blood cells in the presence of the complement, and at a low temperature. This low temperature was present in all the cases under consideration. It seems to me that these men, all of whom had lived sedentary lives prior to enlistment, when thrust rapidly into the full stress of military life, had given way at the weakest point, which in them happened to be in the blood. I am convinced that were a more gradual system of early training recruits more generally adopted many fewer would succumb and be invalided out of the service. In Cases I and II the patients were put back and allowed only the minimum of physical effort, which was increased by degrees with the view of more thoroughly hardening them and enabling them to with-

stand both cold and exposure. So far, after nearly a year in both cases, neither has to my knowledge suffered a relapse.

HEART-BLOCK IN JAUNDICE.

BY

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THE following case occurred in a hospital some months ago, where I saw him with Lieutenant Joscelyne. It was the first example that I have come across of heart-block complicating jaundice, and I now put it on record, after reading Dr. J. Davenport Windle's paper.¹ Dr. Windle states there that he does not accept the commonly-made statement that the heart tends to be slow in jaundice, and with this I fully agree. For years in civil hospital practice I have noticed this, and since coming to France, where we see a good many cases of jaundice, the point has been brought more home to me than ever. But Dr. Windle goes on to say that in his knowledge heart-block has never been demonstrated, and hence my reason for now recording the following case. It was, as will be seen from the tracings, one of partial heart-block, and, strange to say, was at once relieved completely, and, as far as we were able to observe, permanently, by a single dose of atropine.

An officer, aged 43, was admitted to hospital suffering from jaundice. It had existed for about two weeks, and was preceded by nausea and vomiting, but no pain. There was no history of syphilis, but the patient admitted having drunk too freely for years.

FIG. 1.

He looked ill and was deeply jaundiced. There was no oedema at first, but there was some before he left for England. There was some slight irregular fever. The liver was not palpable and seemed small on percussion. The spleen could

FIG. 2.—Immediately after exercise.

not be felt. The urine contained no albumin on several examinations. The pulse could be felt to be irregular in that it often missed beats, and no heart sounds occurred in the pauses. A little exertion, such as sitting up in bed several times, caused

FIG. 3.—Twelve minutes after atropine.

it to become quite regular. Tracing 1 shows the pulse while the patient was at rest. The a-c interval will be seen to get longer and longer until it is more than $\frac{2}{3}$ second, and then the ventricle misses. After this pause the a-c interval is shorter at first, but gets longer and longer until another ventricular miss occurs. Figure 2 shows the action after the exercise described. The a-c interval is still too long, but the stimulus gets through in time and the ventricle does not miss, so that the pulse is regular for a period. It will be seen that this slight exercise caused the auricular rate to fall from 72 to 68. Next day tracings were again taken and showed these same features. Then $\frac{1}{2}$ grain of atropine sulphate was given hypodermically,

and tracings were taken every two minutes for an hour. The first effect of the atropine appeared to be a slowing of the auricular rate, which fell from 72 to 64 (as measured in the tracings), and the ventricular missings were very frequent.

Fig. 4.—After fourteen minutes.

Tracing 3 shows the state of things twelve minutes after the administration of the drug. But at fourteen minutes, the auricular rate being only 60, the heart had become quite regular, although the *a-c* interval still remained much too long (Tracing 4). At twenty minutes the pulse was 68 and quite regular and the *a-c* interval was shortening, and at thirty minutes after the giving of the atropine, as seen in Tracing 5,

Fig. 5.—Thirty minutes after atropine.

the pulse was regular (76) and the *a-c* interval of quite normal length. The pulse was then taken by the sister for another three hours, and was always regular, and ran as follows:

12.50 p.m. ...	72	2 p.m. ...	72
1 " ...	72	2.10 " ...	72
1.10 " ...	74	2.20 " ...	70
1.20 " ...	76	2.30 " ...	72
1.30 " ...	72	3 " ...	76
1.40 " ...	74	4 " ...	76

The general condition became rapidly worse, and he soon developed oedema and ascites. The pulse, however, remained absolutely regular. He was very anxious to get to England, so was permitted to travel there, as the outlook in any case seemed bad.

This case was, of course, not one of simple catarrhal jaundice, but was probably one of organic disease (probably malignant) of the liver. I have no evidence that the condition of heart-block did not exist before the jaundice, except that the patient assured us that he was very fit and quite up to all his work as an officer at the front. The fact that the block was partially relieved by gentle exercise and completely and, as far as we were able to watch the case, permanently, by a single dose of atropine, would all point to the probability of the delay in transmission of stimuli through the auriculo-ventricular bundle being due rather to vagal origin than to any organic disease there. The patient had not been taking digitalis for days before the tracings were taken.

REFERENCE.

¹BRITISH MEDICAL JOURNAL, January 22nd, 1916, p. 123.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

BRADYCARDIA ASSOCIATED WITH JAUNDICE.

DR. WINDLE's paper on the heart's action in jaundice, in the JOURNAL of January 22nd, 1916, prompts me to put on record the following case:

A male, aged 17, came to see me on September 7th, 1914, suffering from well-developed jaundice. This may have been of the epidemic type, as another younger member of the same family developed jaundice some weeks later. He had not been under observation at the onset of the illness, but the initial symptoms appear to have been slight, and he had not kept his bed. The pulse was slow (40 to 44) and irregular. Later in the day he was seen at home and a pulse curve taken. Unfortunately it was not possible to get a tracing of the jugular pulse, so simultaneous curves of the apex beat and radial pulse were taken. The rate was just under 39, and the cardiogram demonstrates that

there were no premature beats; the pulse and ventricular rates were the same. This was confirmed by auscultation of the heart.

In the absence of a venous curve the presence of heart-block cannot be absolutely excluded, but it is improbable, for the following reasons: (1) In a later portion of the curve the rate is rather higher and there is marked (sinus) arrhythmia; (2) as the jaundice passed off the heart-rate

Fig. 1.—Simultaneous curves of the apex beat and radial pulse.

rose gradually. At visits to see me the rate was 43 on September 9th, 45 on September 11th, 55 on September 14th, and 56 on September 19th. It may be concluded, therefore, that, associated with the jaundice in this patient, there was a total bradycardia of high grade with a variable degree of sinus arrhythmia. His normal pulse-rate appears to be about 60. He belongs to a family several members of which have a rather slow heart-rate, his mother's being sometimes as low as 30.

Dr. Windle suggests that the arrhythmic bradycardia in these cases is not caused by the jaundice, but is the same as that which is commonly met with in young people after febrile illnesses. This may be so, and there is merely the apparent relation of the heart-rate to the degree of jaundice in my patient to suggest a causal connexion between the two. It is difficult to prove or disprove, but that the pulse-rate should not decline till the appearance of the jaundice is only what one would expect, since the latter is the outward sign of the entrance of bile into the blood circulation. Even if the jaundice appeared before the fever had subsided and the pulse had fallen, one might not be justified in concluding that bile salts do not lower the heart-rate. The fever, whatever its cause, is an opposing factor which may more than neutralize the supposed retarding influence of bile salts, just as it is difficult or impossible materially to alter the heart-rate in fever by means of digitalis.

Bradycardia is believed to be almost exclusively associated with the catarrhal type of jaundice. This is possibly due to the fact that this disease is much more common in young people, in whom apparently the retarding mechanism of the heart is very sensitive. The frequent occurrence of slow sinus arrhythmia in such patients after even slight febrile illnesses is evidence of this.

HULL.

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AMOEBAE IN URINE IN A CASE OF INFECTIOUS JAUNDICE.

WITH respect to the memorandum on the occurrence of amoebae in the urine of a case of infectious jaundice, recorded by Lieutenant-Colonel Ward, Dr. Coles, and Captain Friel in your issue of April 8th, on page 526, may I point out that an organism called *Amoeba urogenitalis* was described by Baelz in 1883? The species was found in masses in the sanguineous urine as well as in the vagina of a patient in Japan. The amoebae when quiescent had a diameter of about 50 μ , and exhibited granular cytoplasm and a vesicular nucleus.

Similar cases were also reported between 1892 and 1895 by Jürgens, Kartulis, Posner, and Wijnhoff. Jürgens found small mucous cysts, containing amoeboid bodies, in the bladder of an old woman suffering from chronic cystitis; they were also found in the vagina. Kartulis observed similar organisms in the sanguineous urine of a woman suffering from a tumour of the bladder; the organisms measured 12 μ to 20 μ , and exhibited slow pseudopodial movements, and a nucleus and vacuoles were seen after staining. Posner's case, a man, also passed blood-stained urine, in which amoeboid granular bodies, about 50 μ by 28 μ , were present. The amoebae exhibited change of shape, and contained one or more nuclei as well as red blood corpuscles. The patient was under observation for over a year, during which the attacks recurred, and Posner concluded that the amoebae had penetrated into the pelvis of the kidney. Wijnhoff observed four cases of amoeburia in Utrecht, and Jeffries (1904) found similar