

John Rose Bradford, who also saw the cases, agreed; consequently they cannot fall into this category.

With regard to the former ailment—the irritable heart of adolescents—the case is different. Under this heading Da Costa and others described cases in which the heart action was feeble for long periods, and attempts to sit up were followed by fainting attacks and vanishing pulse (Allbutt). A psychical element was often prominent, although not a necessary concomitant.

As to the mechanism of central nervous control, we know that reflex acceleration of the heart may be brought about by (1) accumulation of blood in the great venous reservoirs; (2) diminution of the total volume of the blood as after a haemorrhage, and (3) peripheral expansion; the principle involved being the same in each case—namely, stimulation of the cardiac centre brought about either by inferiority in the quality or in the quantity of the blood supplying it.

The third of these factors corresponds to a loss of vasomotor control, and is represented by a general diminution of tone all over the body.

It is also known that loss of tone occurs as a result of

general exhaustion, although, judging by recorded cases, it would not appear to be of long duration. Instances are mentioned by Allbutt of rapidity of the pulse supervening on muscular exertion in which the vasomotor system was obviously at fault, but they recovered in a week or so, or at any rate showed marked improvement, whereas, of the cases mentioned in this report, all of which were of at least two months' duration when first seen by me, the majority at the end of six weeks showed no improvement at all. Further, in my cases there is no history of great muscular effort. The history is rather one of laborious work of more or less vexatious nature, spread over a considerable time in a strange climate, and frequently under conditions of great personal danger—conditions which with more reason might have been expected to produce the signs of strain as exhibited in "soldier's heart."

The causation is probably to be found in a great complexity of factors, into which the psychology of the Indian, the exposure, shock, and fatigue of the campaign in a climate to which he is unaccustomed, all enter.

ESCAPE OF THE VENTRICLE, IN ASSOCIATION WITH CEREBRO-SPINAL FEVER.

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By escape of the ventricle is understood interruption of the normal mechanism of the heart by the occasional manifestation of the idio-ventricular rhythm. It is an unusual mechanism, and I do not know whether it has been noted before in course of acute disease. In the present case illustrating this arrhythmia (Figs. 1 and 2), the diagnosis of

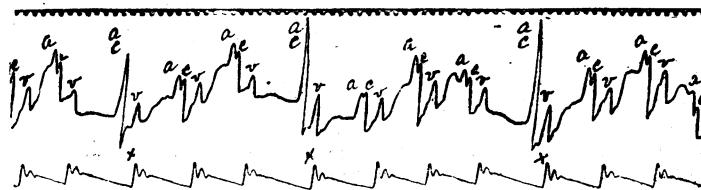


Fig. 1.

cerebro-spinal fever was established by bacteriological examination of the spinal puncture fluid.

The patient, a factory worker, aged 15 years, left work on April 16th, 1915, because of headache and biliousness. He came to my surgery on April 20th, looking pale and tired, and complaining of sickness. He said he had been subject to sick headache on and off for some months past. I could find nothing definitely wrong, and told him to let me know how he was getting on in a day or two. I did not see him again until the evening of April 30th; he said that since his last visit the headache had been very bad, he had been sick occasionally, and had had diarrhoea. However, he had kept about, and felt so much better on April 20th that he went to work for half the day. The temperature was 99° F., the pulse quite regular at 86 to 90 per minute. He looked very poorly, and I sent him home to bed. I visited him at his home next morning (May 1st). He complained of headache, was drowsy, and had been sick. The temperature was 100° F., pulse regular at about 80 per minute, no physical signs in chest or abdomen, the pupils were equal and reacted normally. Kernig's sign was present; his mental condition was clear.

On May 2nd the general condition was much the same, except that he was more drowsy and the pulse was irregular at about 60 per minute; tracings of the pulses and breathing showed sinus irregularity, the shortest diastoles falling with inspiration.

On the morning of May 3rd the general condition was much the same; on examining the heart, however, occasional beats were noted to be unusually forcible, and with these beats the sounds of the heart were modified, and there was a large single pulse in the veins of the neck. These events suggested that the auricles and ventricles

were contracting at the same time, as commonly happens in extra-systole of the ventricle and with some of the beats in heart-block.

Evidence of simultaneous chamber contraction is furnished by the records (Figs. 1 and 2) taken on this day; but analysis indicates that the mechanism by which it is brought about is due to "escape of the ventricle," the stimulus probably originating at the *a-v* node. In Fig. 1 the normal mechanism of the heart is disturbed in three places, by simultaneous contraction of auricles and ventricles, as evidenced by the large waves *a*, corresponding to the radial pulses marked *x*. These beats are due to idio-ventricular contractions, the inherent rhythm of the ventricle becoming operative, because the auricular rate is slowed to, or a trifle below the idio-ventricular rate. The inter-auricular intervals next after the escaped beats are nearly but not quite so long, measuring $\frac{2}{3}$ against $\frac{3}{4}$ sec. This slight difference, however, allows time for the sinus impulse to reach and provoke the ventricle before the idio-ventricular stimulus can become operative.

Fig. 2 was recorded some hours later on the same day as Fig. 1; here ventricular escape is shown after inter-auricular intervals of $\frac{3}{4}$ sec., whereas the normal mechanism is maintained in Fig. 2 with an interval of $\frac{2}{3}$ sec. The relationship of the escape of the ventricle to the respiratory movements is notable; it occurs at the lowest part of the venous curve, that is to say, at the end of expiration. Expiratory inhibition of the auricle is a common phenomenon in meningitis, but it rarely shows the heart below a rate of 45 to 50. The extreme susceptibility of

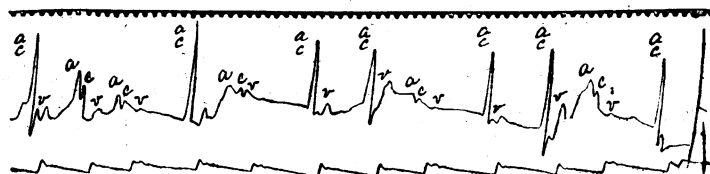


Fig. 2.

the inhibitory mechanism to respiratory influence in the present case suggests that the vagus centres were involved in the lesion.

The patient died suddenly in the night (May 4th), about twelve hours after Fig. 2 was recorded. When I left him his general condition seemed satisfactory; there was no symptom other than the heart rhythm to arouse immediate anxiety; presumably death was occasioned by vagus inhibition.

At the annual meeting of the American Gynaecological Society in Washington next May a discussion will take place on the relations of syphilis to obstetrics and gynaecology. Dr. George Gellhorn, of 713, Metropolitan Buildings, St. Louis, Missouri, who has been delegated to arrange details, asks for the assistance of those who are doing scientific work on the subject.