The predicrotic wave is all but lost, whilst respiratory irregularity and hyperdicrotism appear. Such a fall of tension in an

Fig. 1

old patient with moderately high arterial pressure indicate serious cardiac inadequacy.

CASE II.

A woman, aged 63, suffering from heart failure, with oedema of the ankles, a mitral systolic murmur and a grossly irregular pulse. On exertion the irregularity becomes extreme, but the pressure varies with each arterial beat; the pulse is markedly

Fig. 2.

affected by respiration, and is apparently very empty during diastole. There is a complete absence of the predicrotic wave, and some of the pulsations are almost monocrotic.

In these two cases the almost complete disappearance of the predicrotic wave is presumptive evidence of a diminished systolic pulse pressure.

CASE III.

The man of 72 years whose pulse tracing this represents was not a patient, but was so breathless on exertion that some degree of cardiac inefficiency seemed certain. The tracing shows the condition of the circulation where urgent dyspnoea is a prominent symptom. The systolic pulse pressure was

Fig. 3.

150 mm. in the sitting posture, but apart from some hypertrophy of the heart there were no other evident signs of cardiac disease. The respiratory irregularity, the sudden and abrupt nature of the systole, and the empty condition of the artery during diastole, deserve attention. Note the gradual return to a higher tension one minute afterwards.

CASE IV.

A male patient of 56 years, suffering from cardiac dilatation, with a mitral systolic murmur, oedema of the ankles, engorgement of the liver, and well-marked dyspnoea. On exertion feeble contractions, with a flat plateau at the summit, such as

Fig. 4

we associate with heart failure, develop. There are also intervals during which the pulse fails to affect the tracing. The evidence here points to a much diminished systolic pulse pressure.

CASE V.

A female patient of 67 years, suffering from chronic bronchitis, with cardiac dilatation, and a mitral systolic murmur. While at rest, the pulse of this patient was irregular in rate and rhythm, with about five "intermissions" during the minute, not shown in the tracing; but after exertion there

Fig. 5.

is great irregularity of the pulse. At first one "extra-systole" occurs after each full contraction, but towards the end of the tracing these increase in number till they finally leave the descending line, and appear as small, rounded, independent contractions along the base line.

CASE VI.

This continuous tracing shows the effect the running of half a mile at a quick pace had on the pulse of a healthy

boy of 18 years. Before the start the sphygmograph was applied to the wrist, but some adjustment was called for at the end of the first quarter-mile. The attention necessary at the end of the half-mile was very slight, and after inserting the smoked paper an additional 100 yards were run to keep up the action. The result was chronicled almost immediately. The prominent features are the very

Fig. 6.

low diastolic pressure, the marked respiratory irregularity, the hyperdicrotism, and the variations in force and fullness of the artery with each pulse wave. The effect is not unlike that produced by the inhalation of amyl nitrite, and certainly suggests a pulse of low tension, though the systolic pulse pressure is undoubtedly powerful. After a few seconds the arterial tension begins to improve.

Cleghorn⁶ took pulse tracings of runners two minutes after a Marathon race, and he and Williams⁷ found that there was a great reduction in blood pressure after such severe exertion.

CONCLUSIONS.

1. Exertion causes a rise in systolic pulse pressure, though a general vaso-dilatation may nullify this effect.
2. There is always a lowering of diastolic pressure.
3. In heart disease with failing compensation the fall in diastolic pressure is most pronounced; and there is often an almost complete absence of the predicrotic wave, pointing to a diminished systolic pulse pressure.

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RAPIDITY OF THE PULSE DEPENDENT UPON PERSISTENT DISTURBANCE OF THE VASOMOTOR MECHANISM.

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CAPTAIN R.A.M.C.

THE following remarks are based upon observation of a series of twenty cases of rapid pulse in my charge. The subjects were all Indians drawn from units of the Indian Expeditionary Force in Flanders, who, after undergoing treatment at a casualty clearing station and base hospital for various injuries and ailments, had eventually been transferred to a convalescent camp. In this camp it was noticed that a certain number of the men did not improve as they should; they always appeared languid, tired, and generally unfit. On being questioned no particular complaint would be made, the man probably saying that he felt weak and unable to do anything; his general aspect coincided with this view of things.

The brief physical examination possible under the circumstances revealed nothing much except the rapidity of the pulse, and the patient was placed among the group requiring prolonged convalescence. Improvement was either very slow or did not take place at all, and it soon became apparent that there was a permanent residue of "unfits" in the camp—a residue which was slowly but surely increasing.

I was privileged to investigate and report upon a certain number of these "unfits," and the following remarks are based upon observation of a series of twenty during a period of six weeks.

The experience gained by medical officers during the South African war on the subject of cordite and its effect on the heart is probably well known, and in order, as far as possible, to eliminate errors of this nature, the patients were placed in a special ward, and were not allowed to leave it for any purpose, a guard being mounted to enforce this.

Every article of clothing and kit was taken away from them except water bottles. Food and water were brought

to them, and special latrine arrangements made. No smoking was allowed, and no drug of any kind administered. The patients were kept in bed until after I had made the morning visit; subsequently they were allowed to get up and walk about the ward.

Among the twenty cases there was no common factor which could be taken directly as having a causative influence. They were of all races and castes—Mohammedans who smoke and do not take alcohol; Sikhs who take alcohol and do not smoke; Hindus and Gurkhas who usually do both. Most of them had been in France nine or ten months, and for the greater part of that time had, according to their own statement, been occupied in digging trenches. Two had been gassed and four had been sent down with mumps. None had recently suffered from any acute debilitating disease, and none had undergone a period of prolonged rest in bed. Only three had been wounded; a good many of them had never taken part in an engagement.

When first seen by me they all presented the same general characters—a tired, listless expression, fatigue on walking a few yards or after a few simple exercises, and in one or two cases undue shortness of breath after any such slight exertion. In no case was any complaint made of pain, palpitation, or other subjective symptom.

Physical examination of the patients while lying in bed gave the following results:

One patient had marked dullness to the right of the sternum. An x-ray photograph showed a normal shadow, and at a subsequent examination two days later the dullness was no longer present. One other patient had dullness to the extent of a fingerbreadth to the right of the sternum, but there was no upward increase, and the position of the apex beat was normal. In this same patient was also observed slight pulsation in the jugular veins. The heart sounds were normal and there was no oedema.

With this possible exception there was no evidence of dilatation of the heart in any of them. None presented signs of hypertrophy. In every patient the position of the apex beat was within the nipple line. The cardiac impulse was not forcible or heaving, in fact, in some cases it was so weak as to be difficult of localization. The heart sounds were normal in character and correctly spaced. Accentuation of the pulmonary second sound was absent, and no reduplication was observed. No form of irregularity was present.

With regard to the character of the pulse, in every patient except one the rate was normal while lying down. The volume was good, and no irregularity in rate or force could be detected by the finger. The tone of the vessel wall appeared good, but as a certain number of them showed thickening of the vessel wall (in five of them it was marked, although no cardiac hypertrophy was present) it is difficult to generalize about this. Blood pressure in the radial measured by a Riva-Rocci manometer varied from 120 mm. to 140 mm. Hg. In no case was there any diastolic.

On slowly assuming the erect position, in six patients only did the pulse keep within what was considered normal limits. In the remaining fourteen the rate was enormously increased, in some cases going up to 130 or even 140—in one case to 156. In this latter case the increase amounted to nearly double the rate, and in nearly all it represented an increase of at least 30 beats a minute over the rate while recumbent. On walking the ward twice slowly, the rate might be further increased by six to eight beats a minute, but in some cases it hardly suffered any increase at all. Thus in every one of these 14 cases the great increase in rate took place merely as a result of standing up, done in every case slowly and without effort. The character of the pulse underwent a corresponding change. The volume became poor and the radial vessel appeared only half filled.

Blood pressure readings taken again showed in most cases a drop of 5 to 10 mm. Hg. In a few cases the drop was greater than this, reaching 15 to 20 mm. Hg. In one case the reading fell from 135 mm. to 110 mm. Hg. There were no missed beats and no diastolic could be observed.

After the patient had been standing a couple of minutes the rate would probably fall by 6 to 8 beats per minute, after which there would be no further alteration. On lying down again the pulse would continue to beat at its

altered rate for 4, 6, or 8 beats, and then drop suddenly to normal. In a few cases this reduction would take place gradually, and the normal rate would not be gained for 30 seconds.

I next tried the effect of raising the patient passively from the bed into a sitting posture. The increase in this case was usually not as marked as on standing up, but was nevertheless very striking. I append figures illustrative of this, as below:

Lying.	Raised to Sitting Position.	Standing.	After Standing Three Minutes.	Lying Down Again.
Pulse ... 80 B.P. ... 128	112	120 122	120	Sudden drop to 96 after 6 beats.
Pulse ... 80 B.P. ... 130	104	120 126		Sudden drop to 80 after 4 beats.
Pulse ... 84 B.P. ... 140	116	120 120		Sudden drop to 94 after 7 beats.
Pulse ... 64 B.P. ... 134	84	100 116	100	Gradual drop to 80 at end of 30 secs.

These figures will serve to show what took place in a typical case. The extreme cases I have purposely omitted.

Referring to the six cases to which exception was made in the first place, the fluctuations were not so marked. In these six, on standing up the rate was increased by 15 to 25 beats a minute, and after walking the ward twice slowly the rate did not exceed 100; but if an increase in rate of about 10 beats a minute on resuming the erect position be considered normal, even these six would show abnormality.

Assuming that these facts could best be explained on the hypothesis of some vasomotor disturbance, the urine of every patient was examined for intermittent albuminuria, controls being done at the same time on a dozen healthy Indian soldiers who had never been to the firing line.

Immediately on waking, samples of urine were taken, and after the patients had been walking about the ward samples were again taken four hours later. On one occasion six, and on another occasion seven, of the patients showed faint traces of albumin in the second specimen.

The first urine passed was free from albumin on both the above occasions, with one exception—a patient subsequently invalidated with nephritis. Of the twelve controls made on healthy men, there was a trace of albumin in one specimen only.

Other evidence of vasomotor instability was to be found in various circulatory disturbances exhibited in some cases. Cold hands and feet, even when warmly wrapped up in bed, were commonly noted, and in two cases flushing of the face was so marked as to be spontaneously remarked upon by an independent observer. A "tache" could be obtained in two cases.

Such, then, briefly stated, were the facts brought to light during this period of observation, and it but remains to appraise them at their true value.

I think it will be admitted that everything points to a central lesion of the controlling nervous mechanism. The patients neither showed signs of hypertrophy nor of dilatation supervening on hypertrophy. There was nothing in the condition analogous to the staleness of the over-trained athlete, neither was there evidence of complete breakdown due to cardiac overstress from hard work. Blood examination made on two unselected cases showed red count and colour index within the normal limits.

Toxic action, whether that of a toxin of endogenous or of exogenous nature, must also be excluded as a possible cause, since any such toxin must be supposed to act independently of bodily position.

That somewhat vague expression, "irritable heart," has been applied, according to Allbutt, to two states which are radically different—the one a functional condition, a "sort of restless distress on muscular exertion," occurring chiefly in growing boys, and eminently curable; the other comprising the group also known as "soldier's heart," a condition involving more or less permanent myocardial damage, and too often incurable. But in the cases of my series there is no evidence of any such damage, and in this Sir

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.

BY

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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 $\frac{1}{2}$ against $\frac{2}{3}$ 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 $\frac{2}{3}$ sec., whereas the normal mechanism is maintained in Fig. 2 with an interval of $\frac{1}{2}$ 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.