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ON THE VASODILATATION IN HUMAN SKELETAL MUSCLE DURING POST-HAEMORRHAGIC FAINTING

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It is generally agreed that when a person faints the sudden fall of the arterial blood pressure causes anaemia of the higher centres. Foster's (1888) explanation of the blood-pressure fall was that the vagus excited such an intense bradycardia that cardiac output and blood pressure suffered a decrease. The emphasis was laid on the failure of the heart; it was a cardiac syncope. Lewis (1932), however, observed cases of fainting without much bradycardia. Moreover, when atropine was injected into a subject just after he had fainted, the heart rate increased to normal but the blood pressure did not. Lewis introduced the term 'vasovagal syndrome' to show that blood vessels and heart were both implicated. He thought that the peripheral vasodilatation was the primary cause of the fall of the arterial blood pressure. This view was supported by the experiments of Barcroft, Edholm, McMichael & Sharpey-Schafer (1944), who observed no fall in cardiac output in man during fainting induced by venesection, but vasodilatation in the forearm.

They considered that (1) the dilatation was probably in the forearm muscles, (2) it was of nervous origin, and (3) if a similar dilatation took place throughout the whole skeletal musculature it would certainly cause a drop in blood pressure, which would be perhaps great enough to explain the fall observed during fainting.

The object of this paper is to give a more detailed account of the changes in the peripheral circulation in fainting in so far as they can be inferred from experiments on the blood flow through the forearm and hand. Observations have been made on normal subjects, on sympathectomized subjects and on subjects with the nerve supply to the forearm blocked.

Methods

Healthy men aged 20–30 acted as subjects. Room temperature was about 20° C.

The subject stripped to the waist. The left forearm was shaved. The plethysmograph for measuring blood flow by Lewis & Grant's method (Barcroft & Edholm, 1943) was fitted as shown in Fig. 1. The subject lay down in a comfortable position on a couch with his back raised to an angle

of about 45°. It is easier to induce fainting in this position than with the subject supine. The collecting cuff and wrist cuff were put on. The arm in the plethysmograph was placed in a comfortable position in an electrically heated and stirred constant temperature water-bath, with the water level just above the elbow, and the water temperature about 34° C. (Barcroft & Edholm, 1943). The subject was covered with blankets. The arrangements for recording the flow were completed, and the apparatus calibrated. A sphygmomanometer cuff was put on the right arm. A 6½ in. sphygmomanometer cuff was put on each thigh as high up the thigh as possible.

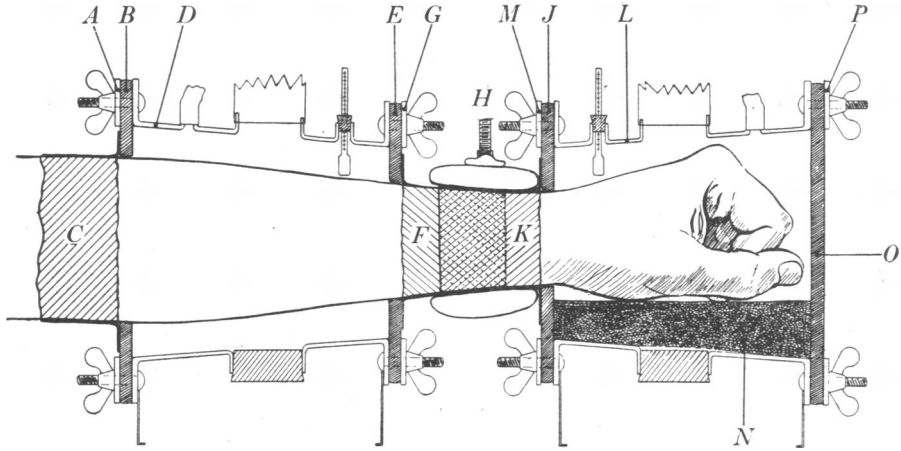

Fig. 1. To record the blood flow in the forearm the plethysmograph was fitted as follows. The brass ring *A* was slid up the arm, followed by the nicely fitting 'made to measure' rubber diaphragm *B* with its 2 in. long thin rubber cuff *C*. The cuff *C* was rubber cemented to the skin as high up the forearm as possible. Plate *A* and diaphragm *B* were then bolted to the plethysmograph *D*. Diaphragm *E* and attached cuff *F* were then slid into position, and the cuff *F* cemented to the skin. Diaphragm *E* was plated to the plethysmograph with two semicircular plates *G*.

To record the blood flow in the hand the plethysmograph *L* was fitted as follows. Cuff *H*, made of cycle inner tubing, was slipped on the forearm. Next came diaphragm *J* with attached cuff *K*. The position of the diaphragm was adjusted so that it was just over the wrist joint. Cuff *K* was cemented to the skin. The plethysmograph *L* was of the same pattern as *D*. The opening in its narrower end was plated to the diaphragm *J* by plates *M* so that the hand was palm downwards. The flexed fingers were supported on a perforated zinc platform *N*. The wide end of the plethysmograph *L* was closed by a diaphragm *O* plated on by ring *P*. Finally the wrist cuff *H* was slid distally over cuff *K* till quite close to the plethysmograph, where it was covered with insulating tape to prevent it ballooning outwards. To record the blood flow in the forearm and in the hand simultaneously the forearm plethysmograph and then the hand plethysmograph were fitted as above. With a little manipulation cuff *K* was cemented to the skin and to cuff *F* under the venous occlusion cuff *H*.

One observer was responsible for recording blood flow, blood pressure (B.P.) and pulse in the left arm; a second for B.P., pulse, and venesection in the right arm; a third for inflation of the thigh cuffs and for noting symptoms, etc.

Blood pressure, blood flow and pulse rate were recorded at 5 min. intervals in the first part of the experiment.

After three or four readings the cuffs on the thighs were inflated with air to 80 mm. Hg, and, during the next 20 min., the pressure was gradually raised to about 10 mm. below systolic pressure.

This procedure dams back blood in the veins of the legs and so produces the equivalent of a large haemorrhage (McMichael & Sharpey-Schafer, 1944; Ebert & Stead, 1940). 30 min. after inflating the thigh cuffs B.P. measurements on the right arm ceased and venesection began. No local anaesthetic was used. An arbitrary maximum of 5 c.c. blood per lb. body weight was fixed for the volume of blood to be drawn off, and it required 10–15 min. to remove the full quota. If the subject fainted before this quota had been withdrawn, the needle was removed and venesection ceased. After venesection, the thigh cuffs were kept inflated till (*a*) the subject fainted, (*b*) a gradual rise of the B.P. showed that he was unlikely to faint, or (*c*) the legs had been congested for 1 hr. (The above technique induced fainting in about 90 % of the subjects.)

Until the onset of fainting, the collecting pressure on the plethysmograph arm was adjusted in accordance with the B.P., and was kept at approximately diastolic pressure. During fainting, when the B.P. falls precipitously, it is essential to use a very low collecting pressure, i.e. 30 mm. Hg or less, otherwise the usual collecting pressure may be well over the level of the systolic pressure and no blood flow will be recorded. As soon as fainting began the collecting pressure was quickly lowered to about 30 mm. Hg. Blood flows were recorded at about 1 min. intervals. The wrist cuff was kept inflated between readings. B.P. readings were made at short intervals. In the fully developed faint the B.P. was sometimes unobtainable. The pulse was generally impalpable. The heart beat could often, but not always, be auscultated at the apex.

After two flow-records had been taken in the fully developed faint the pressure in the thigh cuffs was released, so that the blood dammed back in the lower limbs was returned to the general circulation. If the faint was severe the head was lowered. B.P. usually began to recover within 3 min. (In two cases in which it did not, an intramuscular injection of 20 mg. methedrine was given and the blood pressure rose rapidly. During the war, in this country alone, more than 25,000 blood donors have fainted and all have recovered.)

Recording was continued during recovery. If more than 500 c.c. of blood was withdrawn, the subject was transfused. Recording stopped ½–¾ hr. after the faint, or rather later if transfusion had been done. By the time the plethysmograph had been taken off, the subject felt perfectly fit to go on with his occupation. During the recovery period the subject was asked to recall any symptoms noticed before fainting. One 10-stone subject who lost 700 c.c. and was not transfused had a second delayed faint. Others felt a little tired; no other after-effect was ever noticed.

For measuring hand flow, Freeman's (1935) method was used. The hand plethysmograph was fitted as shown in Fig. 1.

In two experiments, forearm and hand flows were measured simultaneously in the same limb (see Fig. 1). In these experiments two schemes for taking the records were tried. In one, turning a single tap threw the collecting pressure into the cuffs on the upper arm and on the wrist simultaneously. In the other, the blood flow through the hand was recorded first, then, after an interval of 1 min. the circulation in the hand was arrested and the forearm flow taken. Owing to the greater initial distortion when flows were measured simultaneously, independent recordings were preferred.

The sympathectomized forearm. Preliminary tests were made to see if the sympathectomy had been complete. Two tests were used: Landis & Gibbon's (1933) body heating and finger temperature test; and the body heating and forearm blood flow test (Prinzmetal & Wilson, 1936; Wilkins & Eichna, 1941). Both tests are based on the fact that rise in the body temperature causes a vasodilatation in the arm, mediated by the sympathetic. A limb was regarded as completely sympathectomized when the normal rise in finger temperature and forearm blood flow were absent. These tests enabled us to exclude one subject who did not conform to these standards.

In this group of six subjects there were three females, and the age limits of 16–64 were wider than in the normal group. It is unlikely that these factors could have affected the significance of the results.

The nerve-block forearm. The musculo-spiral, median and ulnar nerves in the left forearm were each injected with 2 c.c. of 4 % procaine in 1 in 50,000 adrenaline (Barcroft, Bonnar, Edholm & Effron, 1943). After 15 min. the injections were repeated, and by this time, in almost all cases, the movements of the hand were rapidly becoming paralysed. In a few subjects, in whom the block

was unsatisfactory, the experiment was given up at this point. As soon as the injections had been repeated, the thigh cuffs were inflated. The arrangements for recording the forearm blood flow were completed. One or two estimations of pulse, B.P. and forearm flow were made. The hand was examined to check the paralysis. Half an hour after the thigh cuffs were inflated, venesection was begun.

RESULTS

Blood flow in the forearm during fainting. Results were recorded in nine faints. Typical data are shown in Fig. 2. The time-span in this and all subsequent diagrams is 50 min., 25 before and 25 after the deepest point of the

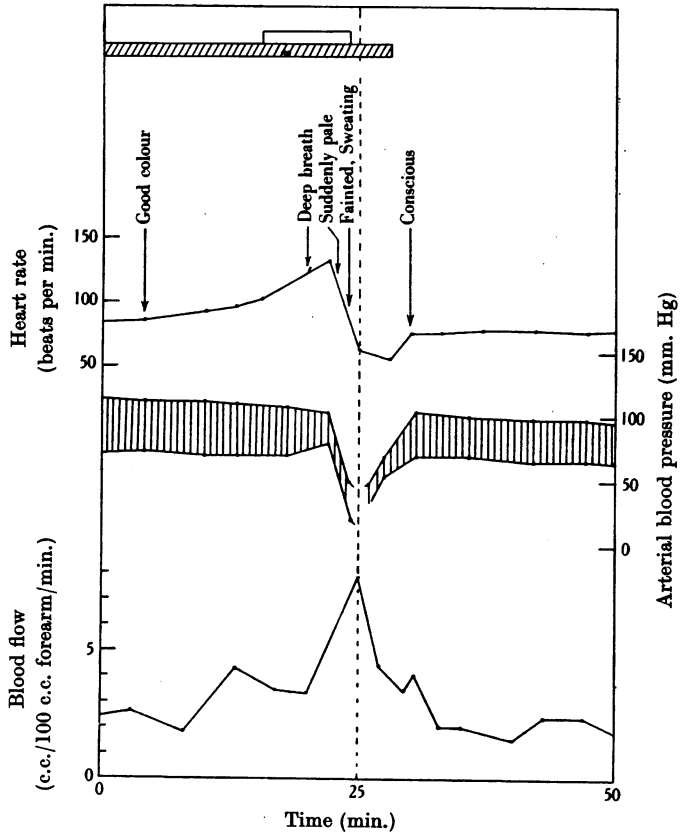

Fig. 2. Post-haemorrhagic fainting. Typical symptoms and typical changes in the heart rate, in the arterial blood pressure and in the blood flow in the *normal forearm*. Plain rectangle: venesection. Shaded rectangle: venous tourniquets on both thighs.

faint as judged from the B.P. and subject's condition. In all diagrams the 25th min. is marked by a vertical broken line; the shaded rectangle shows the duration of venous congestion of the legs, and the plain rectangle shows the duration of the venesection. The symptomatology of post-haemorrhagic fainting has been described by Wallace & Sharpey-Schafer (1941) and by

Brown & McCormack (1942). Our observations on the signs and symptoms will be described elsewhere. The top curve in Fig. 2 shows the typical bradycardia. The B.P. record shows the acute fall from about 105/90 to about 50/20 mm. Hg. In this experiment the subject fainted during the venesection which was stopped before the full quota, 770 c.c., had been withdrawn. Recovery began before the pressure in the thigh cuffs was released. The behaviour of the forearm blood flow is shown at the bottom of the diagram. During the faint, forearm blood flow rose from 3·3 to 6·8 c.c. per 100 c.c. forearm per min., in spite of the acute fall of B.P. There must therefore have been a very marked dilatation of the vessels in the forearm to bring about a doubling of the rate of the blood flow when the B.P. was falling so fast. Fig. 3 shows a typical tracing.

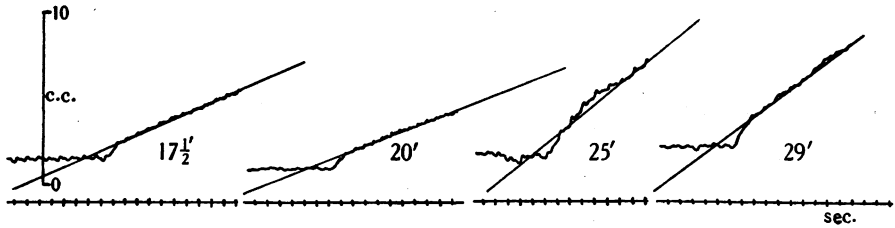

Fig. 3. Tracings of the blood flow in the *normal forearm* obtained by Lewis & Grant's plethysmographic method in the experiment shown in Fig. 4, No. 1. Taken at the times marked on the tracing. Fainting began soon after 20 min., and was fully developed at 25 min.

The B.P. and forearm blood flows of all nine experiments in this group are shown in Fig. 4. Every experiment except No. 5 shows increase in the forearm flow during the acute B.P. fall of the faint—proof of vasodilatation. The faint in No. 5 was severe, breathing was stertorous, the pulse slowed to 30 beats per min. and the B.P. was unobtainable for some minutes. Yet in spite of this profound drop in the B.P. the forearm flow did not decrease, so here too there must have been vasodilatation.

The averages of the B.P. and forearm flows of the group are shown in Fig. 5. There is thus no doubt that when a person faints from blood loss a peripheral vasodilatation in the forearm occurs.

Blood flow in the hand during fainting. Barcroft *et al.* (1944) had two reasons for believing that the vessels which dilated were in the forearm muscles. Firstly the death-like paleness of the skin of the face seemed incompatible with increase in the rate of the circulation through the skin. Secondly Weiss, Wilkins, & Haynes (1937) had found a decrease in the rate of the blood flow through the hand during fainting. Apparently, in fainting, hand and forearm blood flows alter in opposite directions. This could be explained by supposing that there is hyperaemia in muscle and ischaemia in skin and bone. The hand is about 55 % skin and bone (Abramson & Ferris, 1940) and has only 15 % muscle so

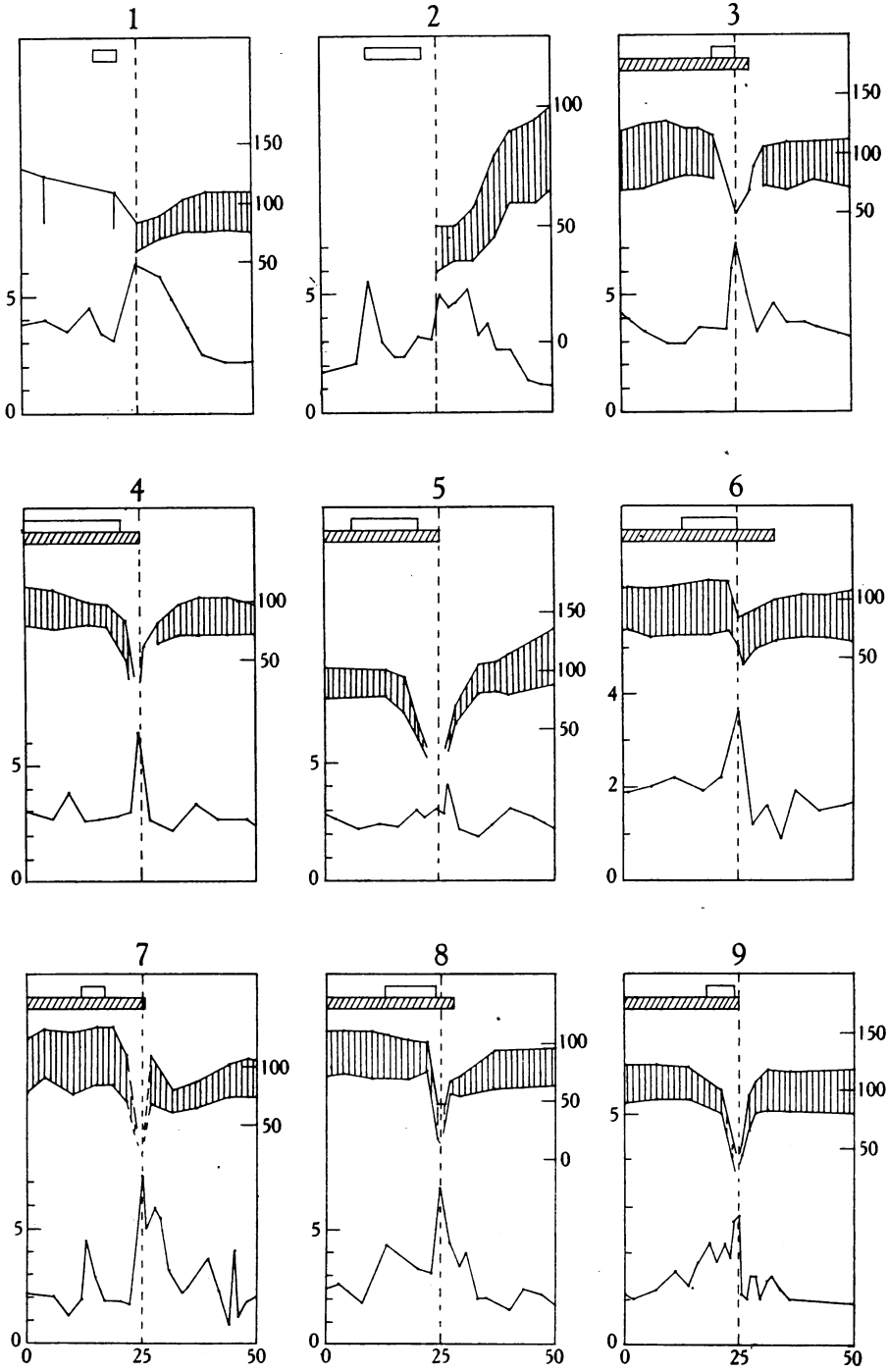

Fig. 4. Plain rectangle: venesection. Shaded rectangle: venous tourniquets on both thighs. Upper curve: arterial blood pressure, mm. Hg. Lower curve: blood flow in the *normal forearm*, c.c./100 c.c. forearm/min. Time in min. Broken vertical line: faint fully developed. In the experiment shown in No. 6, 20 mg. methedrine was given intramuscularly at 28½ min.

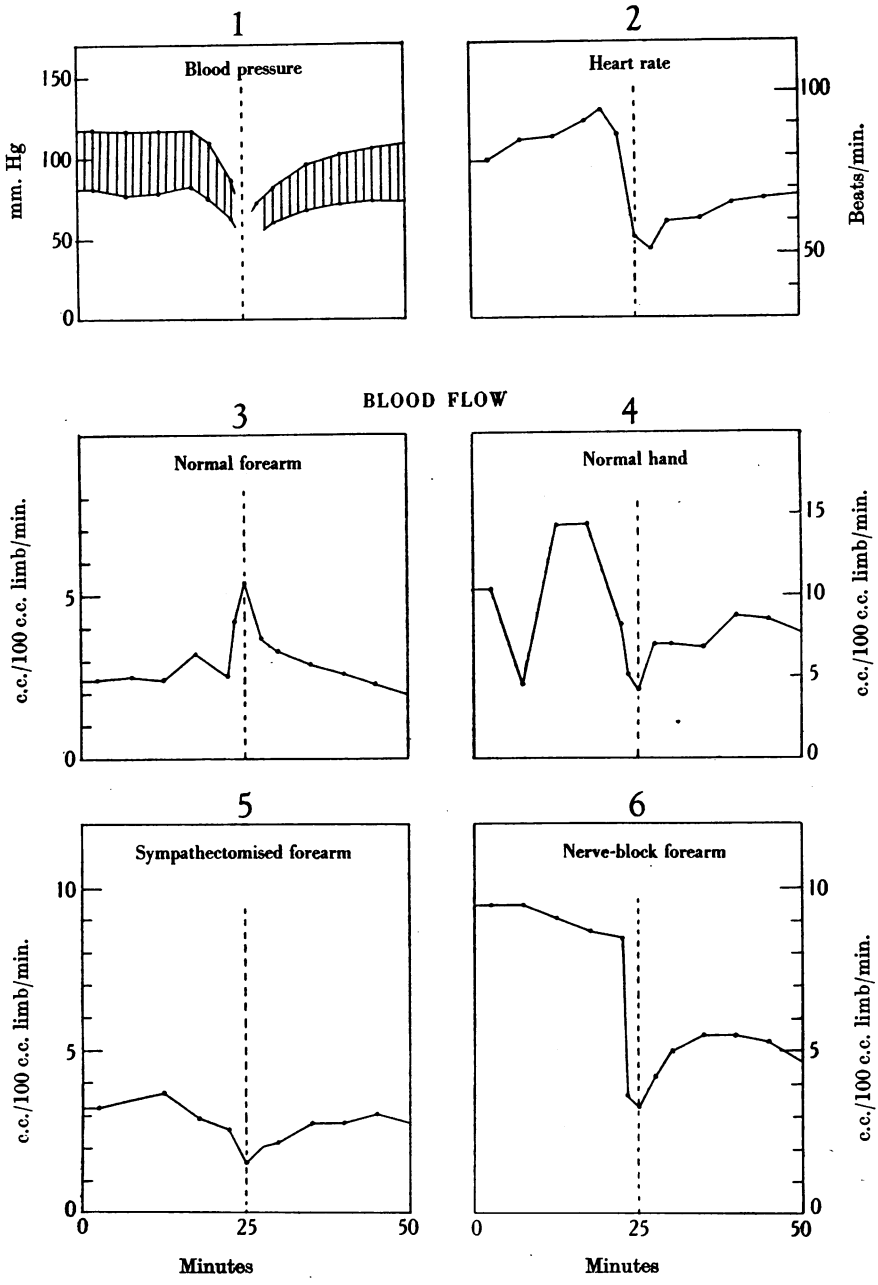

Fig. 5. Averages of results of experiments. Broken vertical line: faint fully developed.

the net effect would be ischaemia. On the other hand the forearm is 60 % muscle, with only 20 % skin and bone, so the net effect would be hyperaemia.

There was one important point about the validity of the results obtained by Weiss *et al.* The hand flows which they recorded during fainting were, to all intents and purposes, nil. This result would have been obtained if they used

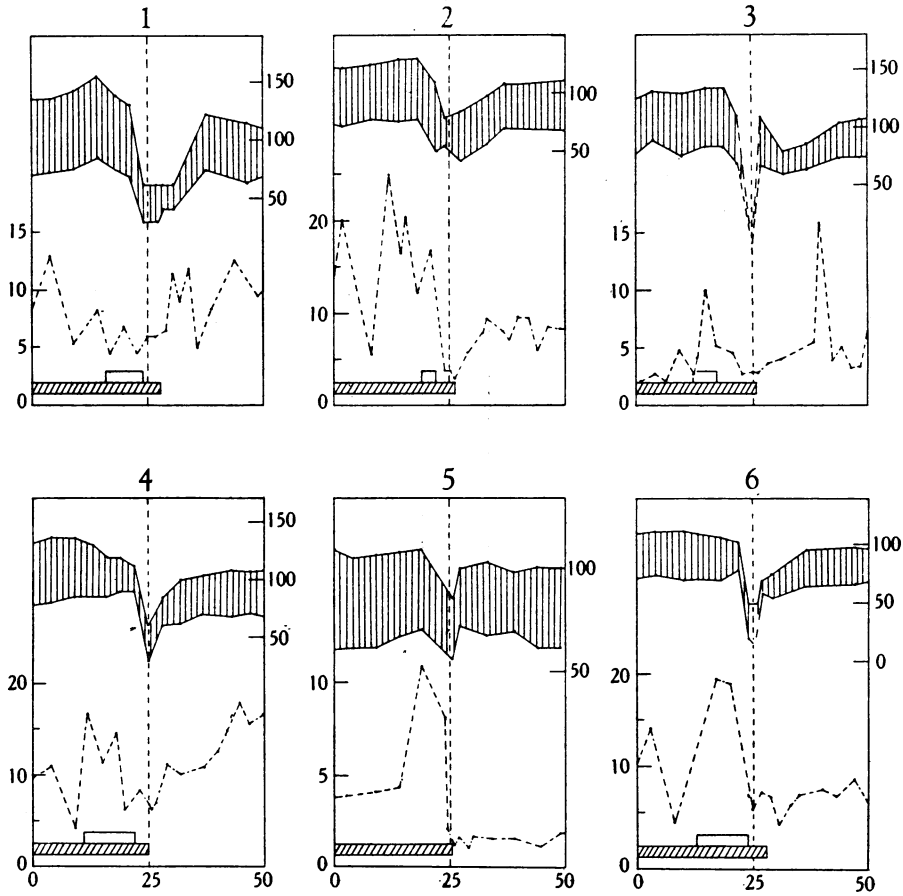

Fig. 6. Upper curve: arterial blood pressure mm. Hg. Lower curve: blood flow in the *normal hand*, c.c./100 c.c. hand/min. Plain rectangle: venesection. Shaded rectangle: venous tourniquets on both thighs. Time in min. Broken vertical line: faint fully developed.

the usual collecting pressure of about 60–70 mm. Hg and did not take the precaution to lower it during the faint; after the collapse of the blood pressure, 60–70 mm. Hg might have arrested the circulation in the hand. Also they induced fainting by administration of amyl nitrite, followed by tilting the subject to the upright position, so that possibly the mechanism of post-haemorrhagic fainting was different. These considerations led us to examine the hand flow in post-haemorrhagic fainting.

In the normal person, hand flows are much greater than, and fluctuate much more than, forearm flows (Abramson & Ferris, 1940). The B.P. and hand flows of six faints are shown in Fig. 6. The consistent thing about them is the low hand flow shown at the onset of the faint in every experiment. The flows in

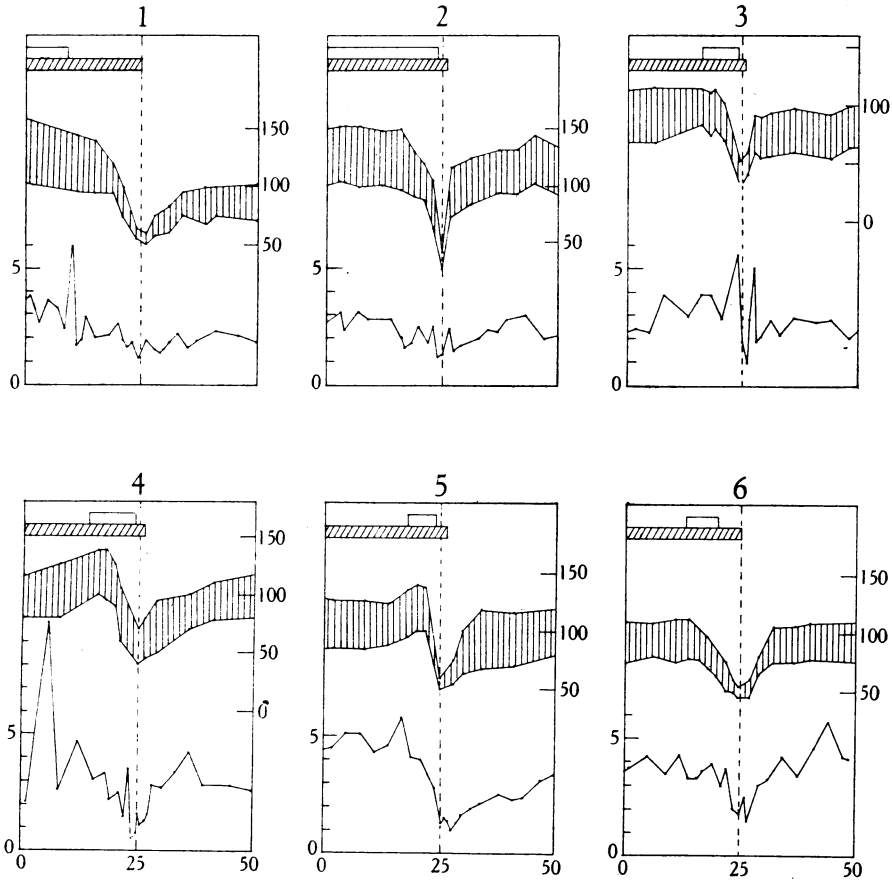

Fig. 7. Plain rectangle: venesection. Shaded rectangle: venous tourniquets on both thighs. Upper curve: arterial blood pressure in mm. Hg. Lower curve: blood flow in the *sympathectomized forearm* c.c./100 c.c. forearm/min. Time in min. Broken vertical line: faint fully developed.

Nos. 3 and 6 were recorded simultaneously with the forearm flows of Fig. 4, Nos. 7 and 8. In both experiments we see small hand and large forearm flows during fainting.

The hand flows for the group have been averaged and the results plotted in Fig. 5, No. 4. There is no doubt about the decrease in the blood flow through skin and bone in fainting. The increase in the forearm flow must have been in the muscle.

Blood flow in the sympathectomized forearm during fainting. To decide the mechanism of the vasodilatation in muscle during fainting, experiments were carried out on subjects with upper limb sympathectomies. If the dilatation were brought about by the vasomotor centre via the sympathetic, it should be absent after sympathectomy. If any other factor were responsible, the dilatation should still be obtained.

The B.P. and forearm flows of six sympathectomized subjects are shown in Fig. 7. The results are strikingly different from those on the normal forearm (Fig. 4), there is no suggestion of an increase in the flow during fainting. The flow is at a minimum at, or close to, the broken vertical line. At first sight No. 3 seems to gainsay the others. As the B.P. starts falling steeply at the beginning of fainting, forearm blood flow increases, just as in the normal forearm. Then it falls to a low level at the broken line, then rises to a peak again as recovery occurs. We think the explanation may be as follows. Sudden brief vasodilatations are seen in Fig. 7, no. 1 at about 10 min., in Fig. 7, no. 4 at about 5 min., in Fig. 4, no. 2 at about 10 min., and in Fig. 4, no. 7 at about 15 min. 'Spontaneous' vasodilatations like these are absent when the subject is mentally at rest. They are almost certainly emotional reactions, due to the stress of the experiment. Similar reactions have been described by Wilkins & Eichna (1941), who thought they were caused by adrenaline because they could be elicited in sympathectomized but not in adrenalectomized subjects. The faint in Fig. 7, no. 3, may coincide with a strong emotional dilatation, and so the forearm blood flow is atypical. For that reason this experiment was omitted from the averaged forearm flows of this group which are seen in Fig. 5, no. 5. The figure shows that sympathectomy abolishes the increase in the forearm blood flow which takes place in the normal subject in fainting.

Blood flow in the nerve-block forearm during fainting. The vasodilatation observed in the forearm muscles of the normal person during fainting might be due to release of vasoconstrictor tone or to active vasodilatation. This might be decided by comparing the average blood flow during fainting in the normal and sympathectomized forearm. If it were greater in the former, active vasodilatation would be involved. However, it is not advisable for this purpose to use the results obtained with the sympathectomized subjects described above, since within a few weeks of sympathectomy the immediate post-operative increase in blood flow has practically disappeared (Grant & Holling, 1938). But such sympathectomies can be carried out temporarily by means of a nerve block of the median, ulnar and radial nerves (Barcroft *et al.* 1943). There is no appreciable spontaneous recovery of tone in the short interval between the block and the induction of fainting (Barcroft & Edholm, 1944). Blocking the motor and sensory fibres is unavoidable, but Barcroft *et al.* (1943) have shown that this has no effect on the blood flow.

Fig. 8 shows data from six nerve-block subjects. Comparing Fig. 8 with Fig. 4 one sees that the initial level of flow in the nerve-block forearm is more than double that in the normal forearm (Barcroft *et al.* 1943). This is because the vasoconstrictor fibres were paralysed and tone in the muscle blood vessels

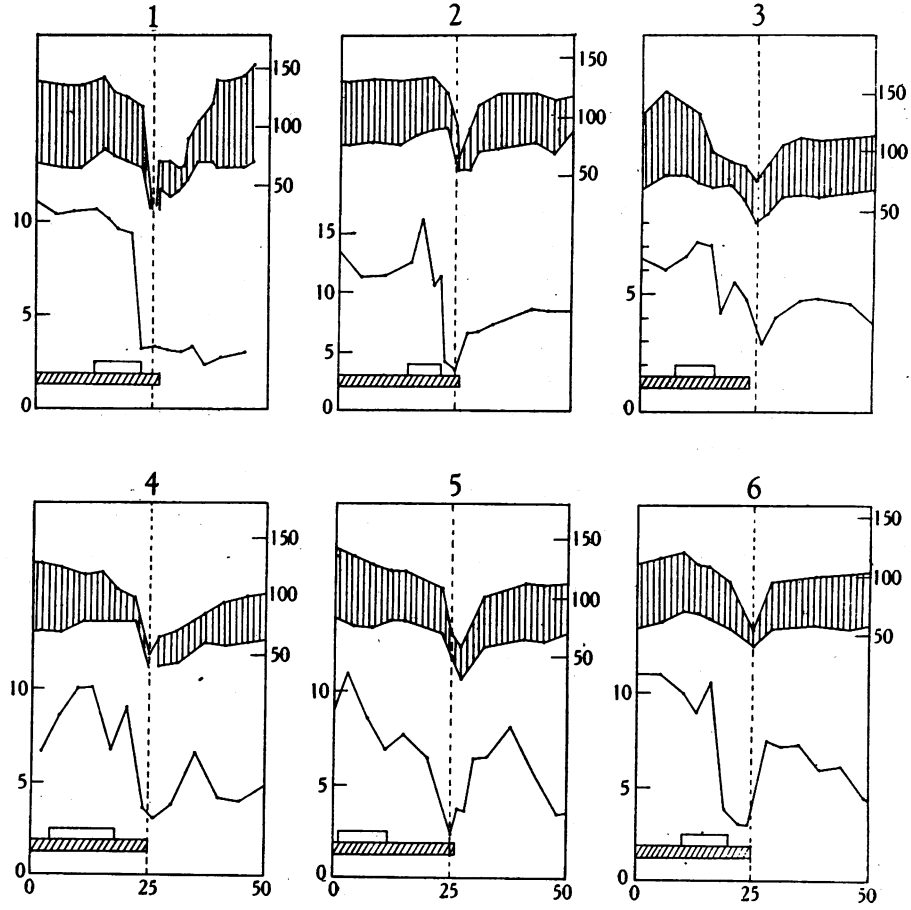

Fig. 8. Upper curve: arterial blood pressure in mm. Hg. Lower curve: blood flow in the *nerve-block forearm*, c.c./100 c.c. forearm/min. Plain rectangle: venesection. Shaded rectangle: venous tourniquets on both thighs. Time in min. Broken vertical line: fully developed faint. In the experiment shown in No. 1, 30 mg. methedrine was given intramuscularly at 28 min.

abolished. In Fig. 8, B.P. and blood flow fall together in every experiment. B.P. recovers as the faint passes off, so to a less extent does blood flow; recovery of the blood flow may be complicated by an injection of methedrine in one experiment, Fig. 8, no. 1, and by the effect of the local anaesthetic beginning to wear off in the other experiments. Fig. 5, no. 6, shows the average forearm flow for the group. The features are those just described. The average

blood flow through the acutely 'sympathectomized' forearm during fainting is seen to be 3·3 c.c. Normal forearm blood flow was much greater during fainting—average figure 5·3 c.c.—implying that the vasomotor centre stimulated active vasodilatation via the sympathetic.

Discussion

The vasodilatation in the forearm during post-haemorrhagic fainting, reported by Barcroft *et al.* (1944) has been confirmed. Their suggestion that it was in the muscle has been investigated and verified. In addition, it has now been shown that the dilatation is actively excited by the vasomotor centre via sympathetic fibres. Sympathetic vasodilator nerves supplying the blood vessels of skeletal muscle have not previously been described in man. Bülbring & Burn (1937) have demonstrated the presence of such fibres in dogs and in hares. The blood vessels of skeletal muscle are therefore supplied by both constrictor and dilator fibres. There is no definite evidence as to the cholinergic or adrenergic character of the fibres.

The absence of the dilatation in the sympathectomized subjects excluded each of the following possible causes:

(1) Adrenaline. There is good evidence that this may be secreted in animals in haemorrhage (Tournade & Chabrol, 1925; Saito, 1928; Saito, Kamei & Tachi, 1928; Brooks, 1935) and that it can cause vasodilatation in human muscle (Grant & Pearson, 1938).

(2) Vasodilator impulses via the posterior roots.

(3) Loss of muscle tone in fainting. It was conceivable that the blood flowed more quickly through the relaxed muscle.

Grant & Pearson (1938) and Wilkins & Eichna (1941) studied the blood flow in the forearm and in the calf. The responses produced by a number of procedures were substantially the same in both parts. Both are mainly muscle. Therefore the behaviour of the circulation in the forearm muscles is probably a reliable index of what is happening elsewhere in skeletal muscle. If this is conceded, it follows that, during fainting, vasodilatation takes place in all skeletal muscles.

Barcroft *et al.* (1944) considered what effect this dilatation would have on the blood pressure. Their observations indicated that the state of the circulation at each of three different stages of a typical experiment was probably approximately as seen in Table 1.

The circulation through the skeletal muscles was estimated from the forearm blood flow, that through the remaining tissues was obtained by difference between cardiac output and skeletal muscle flow.

Haemorrhage caused a small fall in the B.P. and a large decrease in cardiac output. The B.P. must have been maintained, to a considerable extent, by

TABLE 1

	Arterial blood pressure mm. Hg	Mean blood pressure mm. Hg	Cardiac output c.c./min.	Skeletal muscle blood flow c.c./min.	Blood flow through all other organs c.c./min.
Before haemorrhage	130/70	100	5000	800	4200
After haemorrhage before fainting	105/75	90	3000	900	2100
Fully developed faint	50/30	40	3000	2000	1000

vasoconstriction of the non-muscular tissues. Gesell (1920) demonstrated decrease in blood flow through the salivary glands of animals during haemorrhage, and Gregersen (1941) has shown that in man a haemorrhage of 500 c.c. will only cause a slight fall of B.P. and reduce the salivary secretion to one-twentieth of the previous level.

The sudden and profound fall in the B.P. in the faint was not accompanied by any further decrease in cardiac output and therefore was not directly due to the loss of blood. It must have been caused by peripheral vasodilatation. There was evidence of such dilatation in the skeletal muscles since muscle flow was approximately doubled in spite of the profound fall in the B.P. Outside of the skeletal muscles, in the faint, blood flow must have decreased from about 2 to about 1 l. per min. This remarkable diminution accompanied the sudden reduction of the mean B.P. to about half its value immediately preceding the faint. The fact that both B.P. and flow decreased to about the same extent suggests that the reduction in flow was mainly a passive change due to the fall in the B.P., for Wiggers & Werle (1942) consider that, in passive changes, B.P. and flow are almost directly proportional. It may be concluded that vasodilatation in skeletal muscle was the prime cause of the acute fall in the B.P. during fainting.

Fig. 5, nos. 1 and 3 show that the time relations of the changes in B.P. and forearm flow during fainting and recovery are in fair accord with the idea that B.P. and muscle flow are inversely related.

The view that, apart from skeletal muscle, the peripheral vascular system behaves passively in post-haemorrhagic fainting needs further investigation. The loss of consciousness is generally attributed to passive decrease in the cerebral flow. The paling of the skin as the faint develops is probably mainly a passive effect since sympathectomized skin pales like normal skin. This was noticed in the experiments on the sympathectomized subjects. The decrease in the blood flow through the hand may have been due to the fall in B.P. Furthermore the intestines look pale during fainting. This was observed by Bailie (1944) in abdominal operations under spinal anaesthesia.

After injury and haemorrhage, it is likely that the vaso-vagal syndrome supervenes and the condition is probably comparable with that known as primary shock. In our experiments, recovery occurred very rapidly even in

cases where the blood lost was not replaced. The pallor and slow pulse however persisted. The pulse rate was not maintained at the slowest recorded rate but did not return to the fast rate found immediately before fainting supervened. Clinically this may be of some importance in that a subject who had lost sufficient blood, and fainted, might only be seen after recovery from the faint, when the pulse rate would be slower than expected. This might lead to confusion.

Haemorrhage is only one of many conditions that may excite the vaso-vagal syndrome. Others are:

(1) Sudden strong emotions (Lewis, 1932).

(2) Severe pain (probably) (Lewis, 1932).

(3) The maintenance of the upright position: (*a*) with the weight off the feet (Mayerson & Burch, 1939); (*b*) after administration of sodium nitrite (Weiss *et al.* 1937); (*c*) in rarefied air (Mateeff & Schwarz, 1935); (*d*) after strenuous exercise (Mateeff, 1935).

(4) Anoxia (Bauer, 1926).

(5) Spinal anaesthesia (Bailie, 1944).

In each of the circumstances enumerated above, the subject is liable to a sudden fainting attack characterized by bradycardia, pallor, sweating, unconsciousness and low B.P. Valuable information could be obtained from examination of the peripheral circulation in these faints.

It is interesting to speculate about the possible function of this mechanism, excited as it is by such varying causes. Is the subject after recovery from fainting in the same condition as before he fainted? Is the vago-vagal syndrome rightly called a circulatory collapse, or is it part of a protective mechanism which leaves the subject's circulation more stable than before?

Summary

1. Post-haemorrhagic fainting was induced by venous tourniquets on the thighs combined with venesection. This procedure induced fainting in twenty-eight out of thirty-two subjects.

2. Blood flow was studied in the normal forearm and hand, in the sympathectomized, and in the nerve-block forearm.

During fainting:

1. Blood flow in the normal forearm increases as the arterial pressure falls. Therefore there is vasodilatation in this region.

2. Blood flow through the hand, which is mostly skin and bone, decreases. Therefore the vasodilatation in the forearm is in the skeletal muscles.

3. Sympathectomy abolishes the forearm vasodilatation. Therefore it is brought about by the vasomotor centre and sympathetic nervous system.

4. Blood flow is greater in the normal than in the nerve-block, i.e. acutely sympathectomized, forearm during fainting. Therefore the vasodilatation is

actively excited; and there must be sympathetic vasodilator fibres in the forearm muscles.

5. The vasomotor centre probably excites vasodilatation in the arterioles of all skeletal muscles. This may explain the sudden fall in the arterial blood pressure in fainting.

The first three experiments on post-haemorrhagic fainting were performed in conjunction with McMichael & Sharpey-Schafer.

We express our deep appreciation of the help given by the members of the Belfast Medical Students' Association, by certain Naval Ratings, and by the sympathectomized subjects.

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