Vasovagal Fainting: A Diphasic Response

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VASOVAGAL fainting, also called vasodepressor fainting, is a common and dramatic reaction frequently occurring as a response to an emotionally disturbing event. The pathophysiology of the faint itself has been recently reviewed by Edholm[7] and by Sharpey-Schafer.[19] The cardinal manifestations are low blood pressure and slow heart rate.

The cardiovascular events which precede the faint have received very little attention, however, and their importance has not been recognized. The few published protocols showing prefaint events are concerned with such special maneuvers as postural changes, hypoxia, or significant blood loss. Except for the Greenfield[11] and Engel-Romano[8a] reports, there are apparently no published records of data obtained at short intervals preceding the onset of emotionally induced fainting. Even the physiology of the faint seems not to be as familiar as would be expected from its frequency. Many physicians are unaware that asystole is common and that when it occurs convulsions may also develop. It is also not widely known that cardiac arrhythmias may occur as part of the faint, perhaps because few electrocardiograms made during a faint have been published.

The literature contains little discussion of an apparent paradox: Situations which provoke fainting seem in general to be those likely to arouse anxiety, yet the heart rate and blood pressure in anxiety are expected to be raised rather than lowered, a change exactly opposite to that seen in the faint. Wolf[21] alludes to this difficulty.

This report may resolve the paradox, by showing that vasovagal fainting is a *diphasic response*. From casual observation and from occasional newspaper accounts of faints, it appears that a faint occurs most commonly immediately after a threatening experience has ended. Thus it may be that, in response to a threat, an emotional state properly called anxiety occurs, of which rapid heart rate and rising blood pressure are a part; and with cessation of the threat, there is relief from anxiety, with the fall in heart rate and blood pressure which are the chief manifestations of vasovagal fainting.

The data which follow are evidence that such a diphasic response is, in fact, characteristic of vasovagal fainting.

Plan of the Experiment

Fainting was studied: (1) in persons giving blood for a hospital blood bank; (2) in persons having a simple venipuncture without significant blood loss; and (3) in patients undergoing pneumoencephalography. Particular care was taken to obtain measurements of cardiovascular variables

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at intervals not longer than about 3 min. for at least 10 min. preceding the onset of fainting.

The variables recorded in all three phases of the study were systolic and diastolic blood pressure and electrocardiogram changes. The blood pressures were obtained by means of a recording sphygmomanometer, which responds to sounds from the brachial artery by means of a microphone placed in the antecubital fossa. Blood pressure readings were obtained about every 2½–3 min. This determination sometimes took as long as 30 sec., so that during very rapid changes, such as those that occur at the beginning of a faint, it was not always possible to obtain both systolic and diastolic readings. In general, the electrocardiographic tracing was standard lead 2, in order to obtain most satisfactory evidence of changes in heart rhythm. This was usually recorded on a portable clinical electrocardiograph, although in a few instances of venipuncture fainting, the tracing was recorded on an eight-channel polygraph, which was also being used for recording of respiratory rate and skin temperature. If faint seemed imminent, the electrocardiograph was run continuously; otherwise, short records were obtained every 2½–3 min.

For the purposes of this investigation, a vasovagal faint was defined as a sudden drop in blood pressure and pulse rate, accompanied by a report by the donor of some disturbance of consciousness, expressed in such words as "dizzy," "lightheaded," and "woozy." Some of the subjects lost consciousness completely, but it is unrealistic to insist on complete loss of consciousness as a criterion of fainting, since it is clear, as will be shown below, that the vasovagal faint is not an all–or–nothing reaction but occurs in various degrees.

Blood-Donor Faints

Since the primary purpose was to study the course of fainting rather than to make comparisons between fainters and nonfainters, blood donors who seemed most likely to faint were selected. This procedure tends to minimize differences between fainters and nonfainters among the selected subjects; differences were nevertheless found. Various more or less intuitive criteria were used at first in the choice of subjects, but as the companion study of fainting prediction[10] progressed, improved criteria for selection were used. In general, those selected were young, with rapid heart rates and an appearance of anxiety. For the sake of greater homogeneity, only males were used. The study was continued until 10 faints had been recorded.

Procedure

Prospective donors came into the room where blood was drawn, and lay down on an examining table. Pulse rates and blood pressures were taken by the usual methods, after which the venipuncture and blood drawing proceeded. Differences from the usual routine introduced by this study for a selected donor consisted of attaching the sphygmomanometer cuff and microphone and the three electrocardiographic electrodes.

Thirty-two donors were studied. Of these, 10 fainted, according to the criteria given above. None of the donors who did not faint reported any significant symptoms. It, therefore, appears, for this and other reasons, that nearly all the so-called reactions occurring in blood donors are in fact vasovagal faints, although there may be admixtures of other phenomena, such as hyperventilation. A survey of the literature, as well as experience with other "reactors" who were not as intensively followed as those in the present group, supports this conclusion.

After the apparatus was in place, a control period of 6–12 min. elapsed before the actual venipuncture. The time was always long enough to permit a minimum of three control readings at intervals of about 3 min. before the donor was again touched

in any way. Longer periods were either the result of technical difficulties with the apparatus or necessitated by the demands of the routines of the blood-donor room. After the venipuncture had been performed and blood had begun to flow, records continued to be made at about 3-min. intervals (or more frequently if the donor appeared about to faint), until the donation had been completed.

The donor was allowed to sit up a few minutes after the end of the donation if there were no signs of a reaction. A final set of readings was taken after he had sat up, and if there were still no evidences of reaction, the apparatus was disconnected. If, on the other hand, a reaction developed at any point during this routine, readings were continued at frequent intervals until blood pressure and the electrocardiogram had stabilized and the donor was ready to leave. Since the times required for the completions of the donations varied between 6 and 15 min., the total number of readings varied considerably from donor to donor, even apart from the increase associated with the occurrence of a faint.

Results

Of the 32 donors studied, 10 fainted.

To facilitate handling of the data, the time of venipuncture was used as the standard reference point. The three sets of readings before and after that point form the basis for the accompanying tables and graphs. Because of various unavoidable irregularities in the mechanics of the blood donations, there was variation from donor to donor in the exact times at which measurements were made with respect to the venipuncture. Nevertheless, these were all at approximately the same intervals, and they were treated as though they had occurred exactly 8, 5, and 2 min. preceding the venipunctures, and at 2, 5, and 8 min. after it.

Figure 1 represents the course of the systolic and diastolic blood pressures and of the heart rate for those who fainted and those who did not. Table 1 gives the mean changes in systolic, diastolic, and mean arterial pressures and in the heart rate for subjects in both these groups. The mean blood pressure was estimated by the formula

$$\text{Diastolic} + \frac{\text{systolic} - \text{diastolic}}{3}$$

It is clear that there is a distinct difference between the two groups in the diastolic and mean blood pressure changes. The fainters showed a rapid rise in diastolic pressure in the 8 min. preceding the venipuncture; the mean rise for the group of 10 was 11.1 mm. Hg. This differs significantly ($P < .02$ by the t-test) from the rise of 3.0 mm. Hg shown by the nonfainters. They also, of course, showed a decrease in blood pressure following the venipuncture. This, however, is no more than to say that they had begun to faint at this point.

The correlation between pre- and post-venipuncture changes in diastolic pressure for the entire group of 32 subjects was −0.631 (31 d.f., $P < .01$). In other words, those whose diastolic pressure rose before venipuncture were also those whose pressure fell afterward.

The systolic blood pressures showed no such significant differences, although there was a slight average rise in the systolic pressure for the group of fainters.

In the companion study[10] of 414 blood donors (including the present group of 32), it was shown that donors with high resting pulse rates were more likely to faint than those with low, and that there was in fact a significant correlation between the pulse rate and the likelihood of fainting. Since all the subjects reported in the present paper were selected as being likely to faint, and since the resting pulse rate was one of the more or less intuitive criteria used in selection (the results of the companion study were not available at the time of the present work) fainters would not be expected to be significantly different from the nonfainters in this respect; and they were not. The mean blood pressure of the fainters rose more than that of the non-

fainters (8.4 versus 2.5 mm. Hg), but the difference was not statistically significant by the t-test.

In short, despite an effort to select only potential fainters for study—a procedure which is certain to reduce differences between fainters and nonfainters—significant differences in the behavior of blood pressure in the 2 groups were found.

There were differences between fainters and nonfainters in 2 obvious respects at the time of the initial cardiovascular measurements. The fainters were younger and had a lower diastolic pressure. It is of interest to know whether the subsequent occurrence of fainting was more closely related to either of these differences than it was to the diastolic pressure rise just discussed. In the companion study it was found that there was a significant inverse

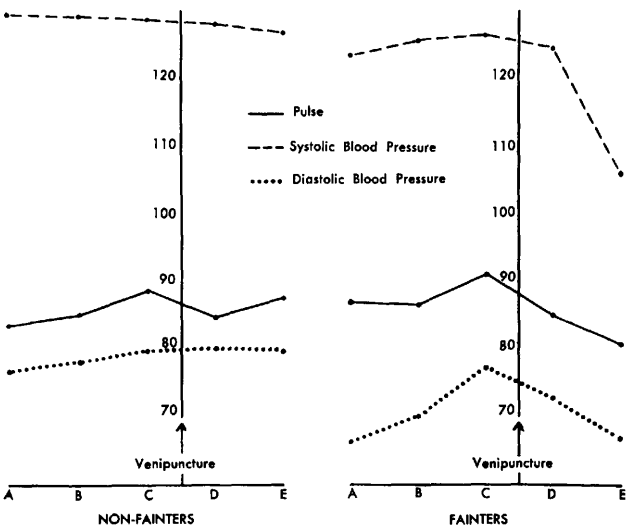

Fig. 1. Mean systolic and diastolic pressures and heart rate of the 22 nonfainters and the 10 fainters. Points *A*, *B*, and *C* are at approximately 8, 5, and 2 min., respectively, before venipuncture; points *D* and *E* at approximately 2 and 5 min. after venipuncture. "Pulse" in this figure represents heart rate.

TABLE 1. CARDIOVASCULAR CHANGES BEFORE VENIPUNCTURE IN BLOOD DONORS

Groups	No. in group	Age (average in yr.)	Mean changes between 1st and 3rd* recordings: Heart rate (beats/min.)	Systolic B.P. (mm. Hg)	Diastolic B.P. (mm. Hg)	Mean B.P. (mm. Hg)
Fainters	10	25.8	+3.9	+2.9	+11.1	+8.4
Matched nonfainters	10	26.9	+7.5	+1.2	+ 6.0	+4.3
Unmatched nonfainters	12	40.7	+3.8	−2.2	+ 0.4	−0.7
All nonfainters	12	34.4	+3.4	−0.5	+ 3.0	+2.5

*Immediately before venipuncture. Time between recordings was about 6 min.

correlation between age and rate of fainting (the younger the donor, the likelier he was to faint), but there was no correlation between diastolic pressure and subsequent fainting.

The nonfainting subjects of the present study were accordingly divided into 2 groups. The first of these consisted of the 10 nonfainters who matched the fainters most closely in age (mean of fainters, 25.8 years; of matched nonfainters. 26.9), and in initial diastolic pressure (mean of fainters, 66.3 mm. Hg; of matched nonfainters, 68.0). The second group consisted of the 12 remaining nonfainters, who had a mean age of 40.9 years and a mean initial diastolic pressure of 84.2 mm. Hg.

The results of this analysis are shown in Fig. 2 and in Table 1. It is clear that the matched nonfainters resemble the fainters much more closely than do the unmatched nonfainters, not only in their prevenipuncture but also in their postvenipuncture behavior. In other words, although they did not actually meet the criteria for a faint, many of them developed a cardiovascular tendency in that direction. It is also clear that, even with matching for initial level and for age, the diastolic rise was greater in fainters than in nonfainters.

Figure 3 is a graph of 1 blood donor faint, interesting because of the drop in systolic pressure which accompanied the rise in diastolic.

Comment

The blood donor fainters showed the expected diphasic pattern of cardiovascular response. They had high initial heart rates, which rose slightly immediately prior to the venipuncture. They showed a rise in diastolic pressure in the 8 min. before venipuncture, but the systolic pressure changed very little. The rise in blood pressure was

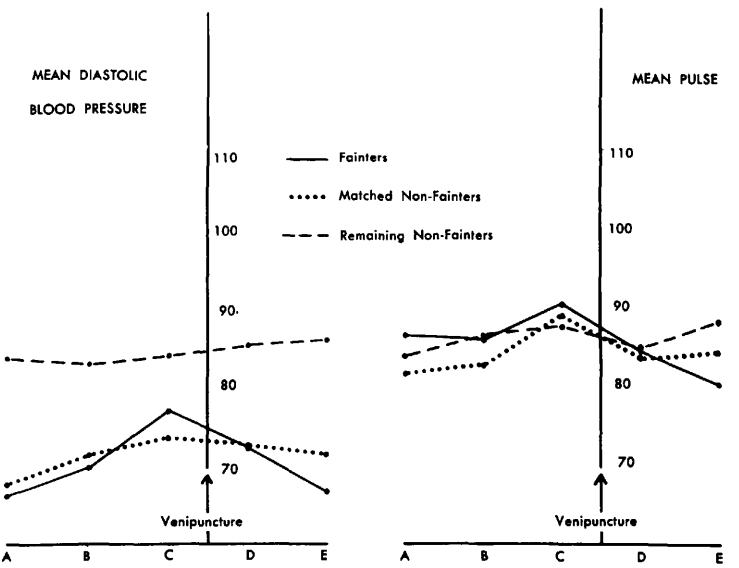

Fig. 2. Mean diastolic blood pressure and mean pulse (heart rate) of the 10 fainters, the 10 nonfainters matched with fainters for age and initial diastolic pressure, and the 12 unmatched nonfainters.

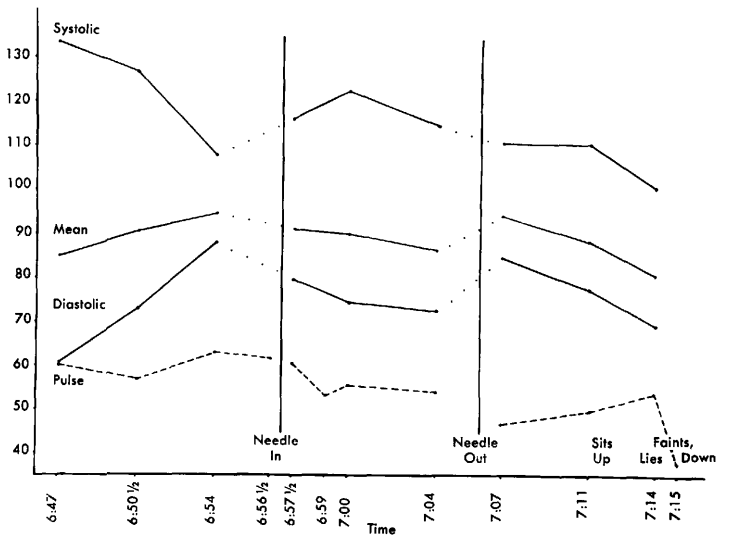

Fig. 3. Systolic, diastolic, and mean B.P. (estimated by formula given in text) and pulse (heart rate) of a donor who fainted only after sitting up at the end of the procedure.

significantly greater than that in nonfainters. In the entire group of 32 fainters and nonfainters, there was a significant correlation between prevenipuncture rise and postvenipuncture fall in diastolic pressure. A companion study has shown that prevenipuncture pulse rates of fainters are significantly higher than those of nonfainters. The similarity between the cardiovascular patterns of the fainters and of the matched nonfainters does not mean that the prevenipuncture rise in blood pressure and heart rate is a matter of age instead of being a precursor of fainting; it is rather a reflection of the greater tendency to faint of young than old persons, so that the young will, as a group, show the "fainting pattern."[10] Similarly, the rise in diastolic pressure of the matched nonfainters, indicates, not that the low initial level instead of a subsequent rise is important, but rather that rises are more likely to occur when the initial level is low (although the lack of correlation in the entire 414 donors[10] between initial diastolic level and fainting suggests that this factor is not a very strong one).

Fainting in blood donors is not primarily a function of the amount of blood lost (although blood loss may affect the incidence of fainting in donors,[17] and the faint under these circumstances is largely an emotional response. In the first place, 520 ml. of blood is probably not enough to produce any significant hemodynamic alteration. The present study suggests the following additional direct evidence: (1) The clear relation between the change in heart rate and blood pressure before the donor has lost any blood and the likelihood of his subsequently fainting makes it obvious that his reaction is to the entire situation; (2) the cardiovascular signs of fainting begin within 2 min. after the venipuncture, on the average, this being clearly much too soon to be the result of blood loss; and (3)

as the companion study showed, donors who acknowledged "nervousness" before the blood drawing were about 2½ times as likely to faint as those who denied it.[10]

Simple Venipuncture Faints

It remained to be seen whether fainting occurring with a simple venipuncture and without loss of more than a few milliliters of blood followed substantially the same pattern as that seen in blood donors. Young males, who had a history of fainting in connection with minor procedures, such as venipuncture, were selected and venipuncture was performed on them in the laboratory.

Procedure

The details of the procedure were not standardized in terms of the time elapsed between the subject's entrance into the laboratory and the venipuncture, the kind of warning he was given that a venipuncture was coming, the time between the warning and the venipuncture, and so forth. In particular, all subjects were not given the same reason for being asked to come to the laboratory. This contrasts with the situation with the blood donors, where all of them knew what was in store, and where the manipulations necessary to the present investigation were a side issue, rather than being the focus of interest. In further contrast, all the blood donors received identical treatment in the 10 min. preceding venipuncture. (Some attempts were made to study the effect of various time intervals and methods of warning on the rate of fainting with simple venipuncture. These were not fruitful, chiefly because of the difficulty in obtaining faint-prone subjects and the unpredictability of faints even in these.)

In 16 attempts with 16 subjects, pulse was counted at the wrist and blood pressure taken by the usual clinical auscultatory method. There were 13 attempts with 11 subjects in which electrocardiograms and blood pressure readings were obtained. These were gathered in the same way as with the blood donor fainters, except that the venipuncture subject was seated in a chair rather than supine. Again, readings were taken at approximately 3-min. intervals, beginning about 8 min. before the venipuncture.

Results

Seven faints in association with the venipuncture were observed in 6 subjects, all of them in the group of 11 having instrumental recording. In 4 of these, records of respiratory rate as well as of the cardiovascular variables were obtained. One subject fainted as the blood-pressure cuff was being applied to his arm, so that a complete set of data about the prefaint state could not be obtained.

Of the 6 venipuncture faints which occurred after preliminary cardiovascular records had been made, 5 showed unequivocally the diphasic pattern. One subject was counted as having fainted only because he replied to a direct question that he "felt weak," and because his heart rate fell at one point to 47, with nodal rhythm. His lowest blood pressure was 103/57 at about the same time. His prevenipuncture diastolic pressure, rose over-all from 75 to a maximum of 87, although it fell to 77 at the time just before the venipuncture.

In the other subjects there were rises in diastolic pressure and in heart rate, or else a constantly high heart rate, in the period just before the venipuncture. In 1 instance, mentioned above, the subject fainted when the blood pressure cuff was applied; in 2 others, the fall in blood pressure began immediately after the subject's arm was touched with an alcohol swab in preparation for venipuncture. The others began to faint after the venipuncture needle was inserted.

Figure 4 shows the course of a faint induced by venipuncture together with puncture of finger for blood count.

Comment

Because of difficulties in standardizing the stimulus situation, comparisons be-

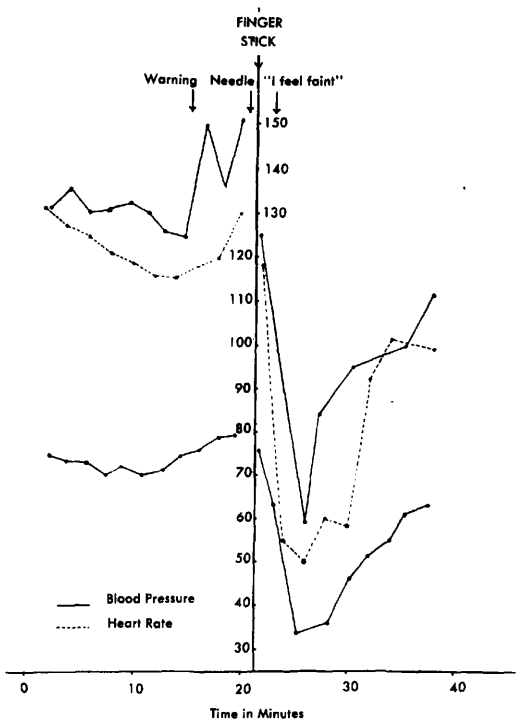

Fig. 4. Systolic and diastolic pressures and heart rate in a male subject before, in, and during recovery from a faint induced by venipuncture with puncture of finger for blood count.

tween fainters and nonfainters are harder to make than with the blood donors. In the latter situation, the subject's attention was not focused on the manipulations incidental to the procedure; with investigative venipuncture, the manipulations necessary for physiological measurement assume crucial importance, and the entire course becomes less predictable.

Systematic comparisons of fainters with nonfainters in the simple venipuncture situation have, therefore, not been made. A number of those subjects who did not faint showed a drop in diastolic pressure and pulse or heart rate after venipuncture, but not one of sufficient degree to justify calling the response a vasovagal faint, and more important in terms of the definition of fainting, they did not have any generalized symptoms. The occurrence of these mild changes demonstrated again that definition of fainting must be somewhat arbitrary.

Pneumoencephalography Faints

Disturbance of consciousness is a common occurrence with diagnostic pneumoencephalography. To determine if this was vasovagal fainting, and if so, whether a diphasic cardiovascular pattern was present, blood pressure records and electrocardiograms were obtained in 15 patients during this procedure. Six of these, including 2

women, had reactions that included some disturbance of consciousness, bradycardia, and lowering of the blood pressure, and therefore satisfied the criteria for vasovagal fainting.

Procedure

Even more than with the simple venipuncture faints, standardizing this investigation was almost impossible. Pneumoencephalography, of course, is a complicated procedure and there are at least three critical points from the patient's point of view: (1) when procaine is injected into the skin prior to lumbar puncture; (2) when the lumbar puncture is performed; and (3) when the injection of air is completed. Times for the various stages were extremely variable. The requirements of the clinical procedure sometimes interfered with obtaining readings at the best times for the purposes of the study. There were also factors in the manipulation of the patient that contributed to the difficulty of standardization.

Results

All of the faints showed the same diphasic pattern seen in the other 2 situations. Figure 5 shows the readings obtained from a 31-year-old white man, who showed the most striking response of any of the group, partly because it was delayed long enough to permit good prefaint records. There were 2 slight drops in diastolic pressure, after the completion of the spinal puncture and after the injection of air, respectively. However, the faint occurred only after he had been told that the procedure was over.

Comment

Although it is impossible to exclude the presence of air in the cerebrospinal fluid space as an important contributing factor

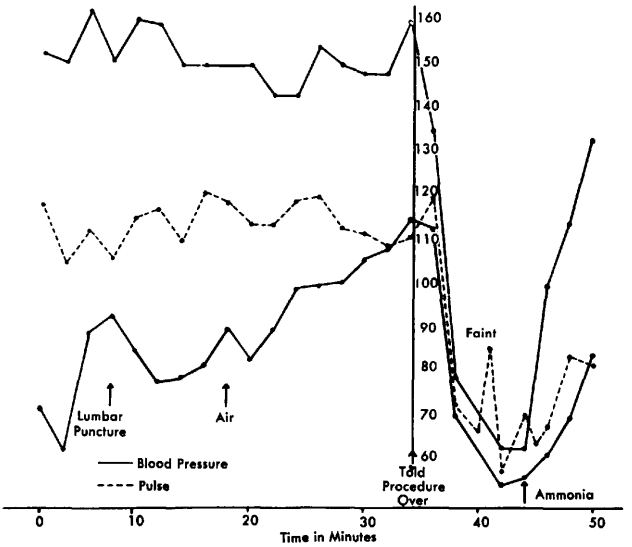

Fig. 5. Cardiovascular variables during a faint occurring with pneumoencephalography. "Pulse" in this figure represents heart rate.

in the reaction, nevertheless the data suggest that the reactions of the pneumoencephalography patients were vasovagal faints and that the same emotional factors were operating as in the other 2 situations.

It is not critical that the reaction in this situation be accepted as emotional; the major point is that it is vasovagal and that it is the second limb of a diphasic response. However, it is true that the pre-lumbar puncture cardiovascular changes observed were like those seen prior to venipuncture in the other situations. Thus, the same reasons for believing that anxiety is involved apply here also.

Special Characteristics of Faints

In many instances, the slowing of the heart rate represented more than simple sinus bradycardia.

Five of the 10 blood-donor fainters showed nodal rhythm, interference dissociation, or both. Three of the fainters with simple venipunctures had the same disturbances, as did 4 of the pneumoencephalography fainters.

More striking was the occurrence of asystole for periods ranging from 4½ to 18 sec. Three instances of asystole were seen with simple venipuncture, none with pneumoencephalography, and 2 with blood donations. In 4 of these instances, generalized convulsive movements occurred; convulsions were never seen without asystole. Restoration of heart beat occurred during the convulsion on each occasion. Figure 6 shows 18 sec. of asystole in a blood donor; Fig. 7, interference dissociation and nodal beats in another blood donor, and Fig. 8, asystole in a simple-venipuncture fainter.

Respirations were counted in 4 of the venipuncture faints by means of a strain gauge attached to an elastic belt around the chest. This is not very satisfactory, since changes in the mechanics of breathing, and irregular respiratory and other skeletal muscle movements, such as occur in anxious subjects, may make interpretation of the record difficult. However, in 2 of the venipuncture fainters, there were periods of 10 and 12 sec. respectively, coinciding roughly with the periods of cardiac arrest, during which there were no definite respiratory excursions. It, therefore, appears that apnea may be part of a severe faint.

It was not necessary for the subject to be sitting or standing to faint. The most severe faint seen occurred in a supine subject.

Discussion

Human reactions are such that irregularities in either the rising or falling phase of a diphasic reaction may make it difficult

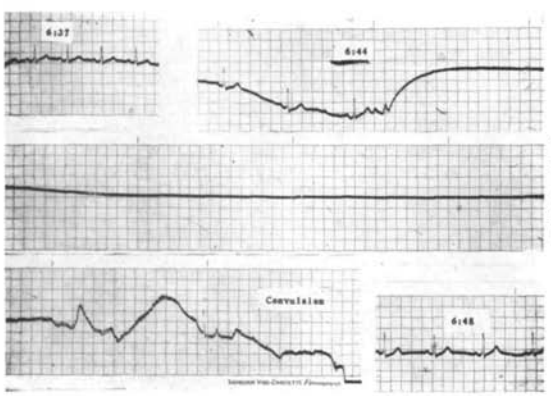

Fig. 6. Electrocardiogram showing 18 sec. of asystole in a faint during blood donation. The faint and asystole were terminated by convulsive movements. Subject remained supine throughout the procedure.

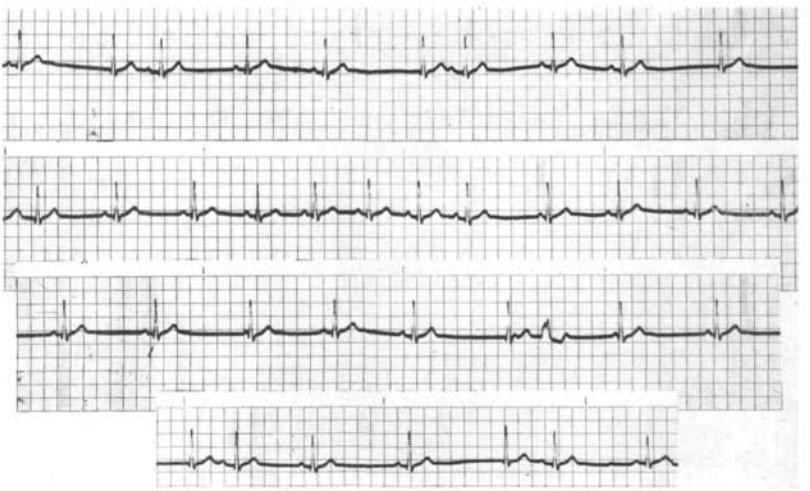

Fig. 7. Electrocardiogram showing interference dissociation and A. V. nodal beats in a faint during blood donation.

to determine whether one is observing anything more than random variations. There are always fluctuations in the graphs of the variables for any subject, so that, by arbitrarily selecting the segment of the graph dealt with, it is possible to "produce" a diphasic effect. The problem can be solved by finding: (1) an obvious "turn-around point," at which the change from the rising to the falling phase occurs (the falling phase being essentially uninterrupted as it proceeds into the faint) or (2) a point that can be designated for all subjects as the critical point. The latter was the case in our recordings from blood donors and, usually, in those from simple venipunctures.

The hypothesis guiding this investigation states not only that the fainting response is diphasic, but also that the turn-around point occurs when the threat disappears; in other words, at the time the subjects's reaction changes from anxiety to relief from anxiety. In venipuncture or blood donation, it is natural to assume that, in general, either the time at which the needle enters the vein or the time at which it is removed is critical. Since the former seemed to be more commonly the case, it was taken as the reference point for the blood donors. This procedure understates the significance of the results, since it does not allow, for instance, for the donor who continues to show the first-phase pattern throughout the period of blood drawing.

In several instances, the onset of the second, or fainting, phase began at the moment when the subject's arm was first touched in preparation for the venipuncture. It is possible to consider such instances as evidence that the proposed interpretation of the diphasic response is wrong and that the second phase should be looked on simply as anxiety which has passed some critical point of intensity. This alternative interpretation would fit some of the faints seen in this study and some seen in daily life. A frequently cited observation is that of the man waiting in line for an injection who faints when watching

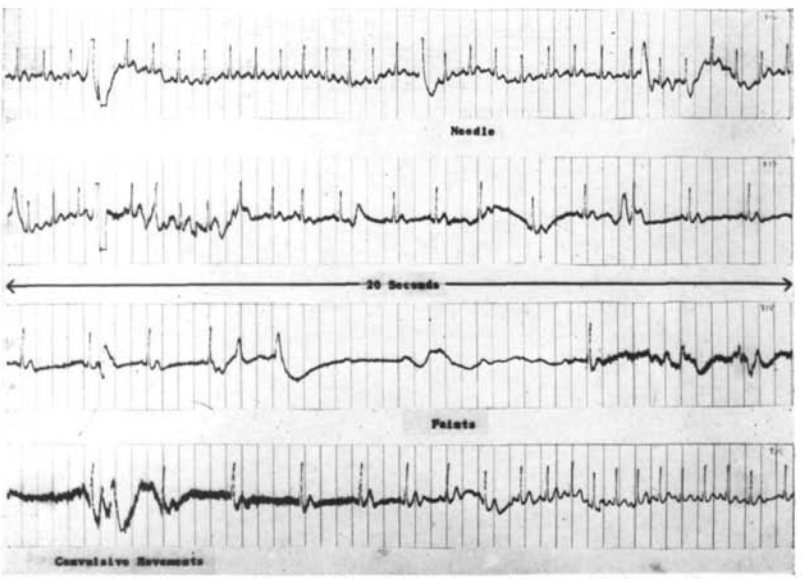

Fig. 8. Electrocardiogram showing asystole in a faint during venipuncture. Obvious muscle tremor was present before the faint. Asystole and faint were terminated by convulsive movements.

someone ahead of him receive one. It is sometimes felt that such an occurrence can only be understood as the consequence of mounting anxiety, since there has been no opportunity for relief of anxiety to occur. However, it may be that the fainter identifies with the other person, so that the carrying out of the threat to the latter is reacted to as if the event were happening to himself. There are many observations of this kind of reaction, e.g., the fainting of medical students at surgical procedures where there is no real threat to the fainter at all.

One of the venipuncture subjects fainted when the blood pressure cuff was applied and before there had been a threat of venipuncture. In this more deviant instance, one can suppose that the application of the cuff represented to him the carrying out of a threat.

Faints, such as that of the pneumoencephalography patient who fainted only after he was told the procedure was over, as well as those of the blood donation or venipuncture subjects who had the first evidence of fainting when the procedure ended, are hardly compatible with any view except that offered by our hypothesis. One could, therefore, invoke two different explanations, one for the apparently aberrant cases, and another for the majority. On the grounds of parsimony, the use of one explanation for all is preferable.

The biological interpretation of vasovagal fainting offered by Engel,[8] who studied it intensively, has aroused much interest. He proposed that such fainting is to be understood as a result of preparation for running when, in fact, running is not carried out. This interpretation is based in part on the demonstration by Barcroft *et al.*,[2] that the mechanism of the drop in blood pressure is vasodilatation in muscle,

mediated by sympathetic vasomotor nerves; thus, it appeared logical that such vasodilatation would be important in increasing the blood flow to the muscles used in running.

There are several objections to Engel's interpretation in its simplest form. (1) It takes no account of the bradycardia, a reaction that cannot be regarded as desirable for running; (2) the occurrence of fainting *after* the threat has either been carried out, or can be seen to be safely passed, is hard to call preparation for running (Even if all vasovagal fainting is not a postthreat occurrence, there are too many cases in which it is clearly such for the preparation theory to be generally valid.); (3) blood flow is not necessarily increased in the tense muscles of an organism preparing for, but not carrying out action. (It is decreased if contraction is sustained at 20 per cent or more of maximal contraction.)[2] Nevertheless, if the entire response of two phases is considered the inhibited running theory may be applicable to the first phase rather than the second. Engel does comment that "sometimes" a diphasic response may be seen, but his interpretation of it is different from ours We suggest that the diphasic response is always present.

The diphasic nature of the process must be recognized if meaningful interpretations are to be made. It resolves the dilemma mentioned by Wolf[21] of relating fainting to anxiety and simplifies the physiological questions raised by Edholm[7] in classifying the symptoms and signs of fainting.

It is not clear whether some special psychological or psychodynamic interpretation should be given to the faint itself. Clearly, the first phase deserves the name "anxiety," and is a response to a threat. It is possible to look on the faint as simply the physiological expression of the sudden cessation of anxiety. An alternative is that the stimulus which induces the second phase (the faint) means more than simply the end of a threat. It might, for instance, be a symbol that all is lost, so that the subject is, in effect, saying to himself, "I'm dead." Although this seems plausible where the threat is actually carried out, it is not so applicable to faints occurring when a threat is suddenly removed without being carried out. Many everyday observations support this concept of "fainting with relief."

We suggest that the faint can be understood, on the physiological level, as the consequence of the sudden cessation of those physiological processes which support the hyperdynamic first phase. These almost certainly call forth antagonistic reflexes which act to prevent the pulse rate and blood pressure from rising without control. Then, if the hyperdynamic processes cease abruptly, the opposing reflex mechanisms will be suddenly unopposed, and fainting will occur. In the case of heart rate, vagal effects which have previously only modified the tachycardia will now, without sympathetic opposition, produce bradycardia or even asystole. Except for the reports of Dermksian and Lamb,[5, 6] little attention has been paid to disturbances of heart rhythm in fainting. They suggest that respiratory stretch reflexes are important in mediating the arrhythmias but neither their work nor ours indicates that these reflexes are always of primary importance. Relevant reflex cardiovascular responses have been discussed by other authors.[1, 4, 12]

It is striking that asystole, fall in blood pressure (not purely a consequence of the asystole), and apnea present a picture indistinguishable from death. There is reason to think that vasovagal fainting may sometimes be fatal.[8, 20] The many sudden catastrophes developing in connection with anxiety–inducing medical procedures may be examples of this phenomenon. Death with surgical anesthesia, where there has been a stormy induction, probably fits this diphasic pattern. In this case the "relief" may be provided by pharmacologic suppression of the activity of the centers mediating the hyperdynamic phase.

The observations of Richter[18] on sudden vagal death in wild rats swimming in cold water or held in the hand appear to fit

very well with the ideas here presented, although not enough details of the protocol are given to be certain. It is possible, however, that his animals were first frightened by hair-clipping or other procedures and that the subsequent immersion in cold water may have been a stimulus situation for them tantamount to insertion of a venipuncture needle for a man.

There are features of interest in the physiology of the first phase, the phase we have considered to be part of anxiety. The pattern of rising diastolic pressure, rapid heart rate, and steady or even falling systolic pressure appears to be what is referred to by Laurell[15] as orthostatic arterial anemia, and more recently discussed by Hickler *et al.*[13] Although these authors describe this picture as induced only by standing, it appears not to differ from that seen in some of our subjects while they were lying-down, and in whom it appears to have been frequently a prelude to vasovagal fainting. Protocols of faints involving significant blood loss and those induced by hypoxia,[2] indicate that these also fit the diphasic pattern, and this may be true of all vasovagal fainting, however induced.

Barcroft *et al.*[2] have presented data indicating that the cardiac output does not fall during a faint, although it may progressively drop before a faint. This drop corresponds to what Laurell[15] reports, and it may be peculiar to situations in which the subject is upright. However, it is obvious that in severe faints with asystole the cardiac output must fall to zero. The findings of Barcroft *et al.* may be due to difficulty in measuring cardiac output at such short intervals. Inspection of their graph suggests that they did not determine the output until after the recovery from the faint had begun.

The data presented in this paper indicate that the vasovagal fainting response consists in its entirety of two successive phases, of opposite direction. It has been suggested that a pattern of this kind is involved in the reactions leading to migraine,[9] asthma,[14] and acne vulgaris.[16] It may be that the diphasic pattern will be found in many other responses and diseases.

Summary and Conclusions

1. Vasovagal (vasodepressor) fainting was studied in three different situations: in persons donating blood, in persons having a simple venipuncture, and in patients undergoing pneumoencephalography. Ten faints were observed in the first situation, 7 in the second and 6 in the third.

2. In all instances, the faint itself, characterized by low blood pressure and bradycardia, was the second phase of a diphasic response; the first phase was characterized by rapid or rising heart rate, and by rising blood pressure, especially diastolic.

3. The data are compatible with the view that the first (hyperdynamic) phase is a reflection of anxiety, while the second phase begins with the sudden cessation of anxiety. It is suggested that physiologically the faint reflects the action of reflex mechanisms activated by the first phase, and then left suddenly unopposed. The other psychobiological interpretations exhibit inconsistencies because they fail to recognize the diphasic nature of this response.

4. Fainting and dying resemble each other closely, and there is reason to think that some vasovagal faints are fatal. Cardiac asystole is not rare in faints and is often associated with convulsions. Apnea was observed in 2 of our subjects.

5. Disturbances of cardiac rhythm, including A. V. nodal rhythm, interference dissociation, and asystole (in 1 instance for 18 sec.) were observed. Generalized convulsions were seen on four occasions, always in association with asystole.

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