THE "BORROWING-LENDING" HEMODYNAMIC PHENOMENON (HEMOMETAKINESIA) AND ITS THERAPEUTIC APPLICATION IN PERIPHERAL VASCULAR DISTURBANCES*

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IT IS NOW A WELL ESTABLISHED FACT that the volume of organs undergoes spontaneous, and even rhythmic, variations, primarily attributable to changes in the volume of the blood within the particular part. When more refined technics of plethysmography were developed, it became practical to carry out accurate quantitative studies of the fluctuations in volume in such peripheral parts as the pinnae, the fingers and the toes, and thus to secure much valuable information under normal resting conditions as well as in diseased states.

There are still numerous gaps in our knowledge of the precise nature of, and the numerous factors involved in, this mechanism, but certain facts have been established which not only have a fundamental physiologic significance but which also appear to have a definite bearing upon certain clinical conditions. It is the purpose of this communication to consider these facts as the basis of a concept which we have chosen to call the "borrowing-lending" phenomenon and for which we are suggesting the term hemometakinesia. We believe that this concept will provide a more rational approach than now exists toward the management of disturbances in the peripheral vascular circulation.

PLETHYSMOGRAPHY

The plethysmogram, which is the completed record of the plethysmograph, is essentially an ordinary type of Cartesian coordinate, with volume represented on the ordinate and time represented on the abscissa. It is thus a record of the changes in volume, in relation to time, of the particular part enclosed in the extremity cup. These recorded volume changes are the algebraic summation of many volume changes occurring in various degrees and directions in many different portions of the enclosed part.[1] Essentially, however, the changes in volume of the part represent changes in the volume of the vascular bed of the part.

Systematic studies with the plethysmograph have established the fact that small blood vessels in such peripheral parts as the pinnae, the fingers and the toes undergo spontaneous variation in volume. The resting individual, in a comfortable environment, presents at least five types of rhythmic changes in

Aided by grants from the Life Insurance Medical Research Fund, the Helis Institute for Medical Research, and the Mrs. E. J. Caire Fund for Research in Heart Disease.

* Read before the American Surgical Association, The Homestead, Hot Springs, Virginia, March 27, 1947.

volume[1, 2] as follows: (1) pulse deflections, (2) respiratory deflections, (3) alpha deflections, (4) beta deflections, and (5) gamma deflections (Figs. 1–4). These changes which are conveniently (and arbitrarily) expressed as per 5 cc. of part, range in volume from less than 0.1 to 350 or more cu. mm.

Pulse Deflections.—Pulse deflections are obviously produced by the heart beat. They represent, in the main, the changes in volume of the part brought about by the blood delivered into the part with each heart beat (Fig. 1). The volume of the pulse deflections varies considerably, but in the normal resting individual the mean values have been found to be 6.9 cu. mm. in the tips of the fingers, 4.0 cu. mm. in the tips of the toes, and 4.1 cu. mm. in the pinnae.[2] The frequency of pulse deflection varies with the heart rate.

It is generally accepted that these changes in volume occur primarily within the arteries and arterioles, but it is believed that relatively large volume changes probably occur within the capillaries also, because of their number and total volume. It also seems likely that the veins and venules contribute to the pulse volume deflections. It is even possible that the volume changes within the arteriovenous anastomoses, capillaries, veins and venules in combination are of greater importance than those within the arteries and arterioles.

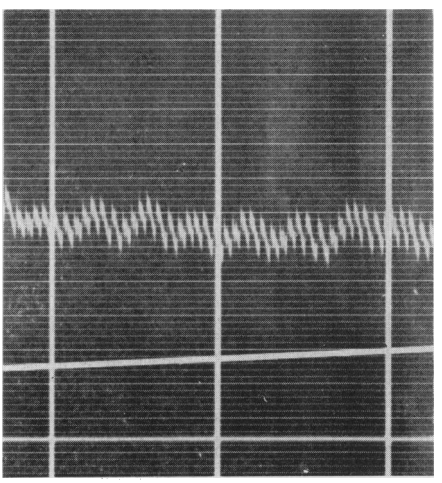

FIG. 1.—Plethysmogram of normal individual showing pulse deflections (reflecting the heart beats) and respiratory deflections which are represented by variations in the base line. Heavy white vertical lines indicate an elapsed time of 15 seconds.

Respiratory Deflections.—Respiratory deflections, which represent variations in volume occurring with the normal respiratory cycle, are most highly developed in the pinnae and least well developed in the toes (Fig. 1). Their volumes vary from less than 0.1 to 5 cu. mm. per 5 cc. of part.[2]

The respiratory deflection depends primarily upon the variations in the venous return to the heart brought about by respiration. Parallel with the increase in venous flow produced by inspiration there occurs a relatively rapid emptying of the venous blood from the part and a resultant decrease in its volume. With expiration, on the other hand, the relatively rapid rate of venous flow is retarded, and there is a resultant relative increase in the volume of the part.

Other factors possibly play a role in this mechanism, but too little is known about them at this time to warrant further comment. Of particular interest, however, is the special type of respiratory deflection which occurs in the fingers and toes immediately following a deep inspiration.[1, 2] There is first a spontaneous, sudden decrease in the volume of the part, varying from 5 to 105

cu. mm., and more prominent in the fingers than in the toes. At the same time the pulse deflections also show a decrease in volume. Shortly after these phenomena are observed, vasodilatation begins and continues until the previous pulsatile characteristics are resumed. Before vasodilatation is complete, however, a series of small alpha waves appear, varying from 2 to 8 cu. mm. After two or more successive deep inspirations, the extent of the response is progressively diminished until eventually a sort of tachyphylaxis occurs and vasoconstriction cease to appear. The volume change is lessened as the interval between inspirations is shortened. The volume change is not necessarily concordant; it may show either an increase or a decrease. Interestingly enough, it is not as definite, as predictable or as large in the pinnae as in the fingers and toes.

Alpha Deflections.—Alpha deflections, which occur less frequently than respiratory deflections, are usually smooth in contour, but are not uniform (Fig. 2). They vary in frequency and size, with a tendency toward an inverse relationship. The mean frequency has been found to be 7.9 per minute in the finger tips, 7.7 in the toes, and 8.6 in the pinnae,[2] and the mean volume for these respective parts has been found to be 14.5, 7.1 and 6.6 cu. mm.[2] Alpha deflections seem to vary from person to person, and even in the same person variations in the different peripheral parts are not necessarily concordant. Although they are independent of variations in arterial pressure,[3] these deflections are predominantly under the control of the sympathetic nervous system; they almost entirely disappear following interruption of sympathetic pathways.[2]

The volume changes represented by alpha deflections are due essentially to variations in volume of the blood within the part, but their frequency suggests the possibility that variations in lymph volume may be a contributing factor. The studies of Webb and Nicoll[4] on the bat's wing showed that contractions of the lymphatics are responsible for much of the flow of lymph, and McMaster's studies[5, 6] on other animals support these observations. Whether or not similar contractions occur in man is not known, but the frequency and rate of the lymphatic contractions observed in animals are of such a character as to permit them to be responsible for some part of the alpha deflections.

Alpha deflections indicate gross or total changes in the vascular bed slower than those produced by the pulse and respiratory deflections. Only a limited portion, and not necessarily all, of the vascular bed may undergo such volume changes at one time. Although the function of these shifts of blood volume is not now known, some significance seems apparent.[2]

Beta Deflections.—Beta deflections are larger waves upon which is superimposed the succession of smaller alpha deflections (Fig. 3). They vary in frequency from one-half to two per minute and their volume range is from 5 to 60 cu. mm.[2] They have been observed in all parts studied. Their frequency and volume are totally irregular, but they tend to vary concordantly in the fingers, toes and pinnae. Beta deflections are also considered to represent primarily volume changes in the vascular bed, but, as is also true of alpha

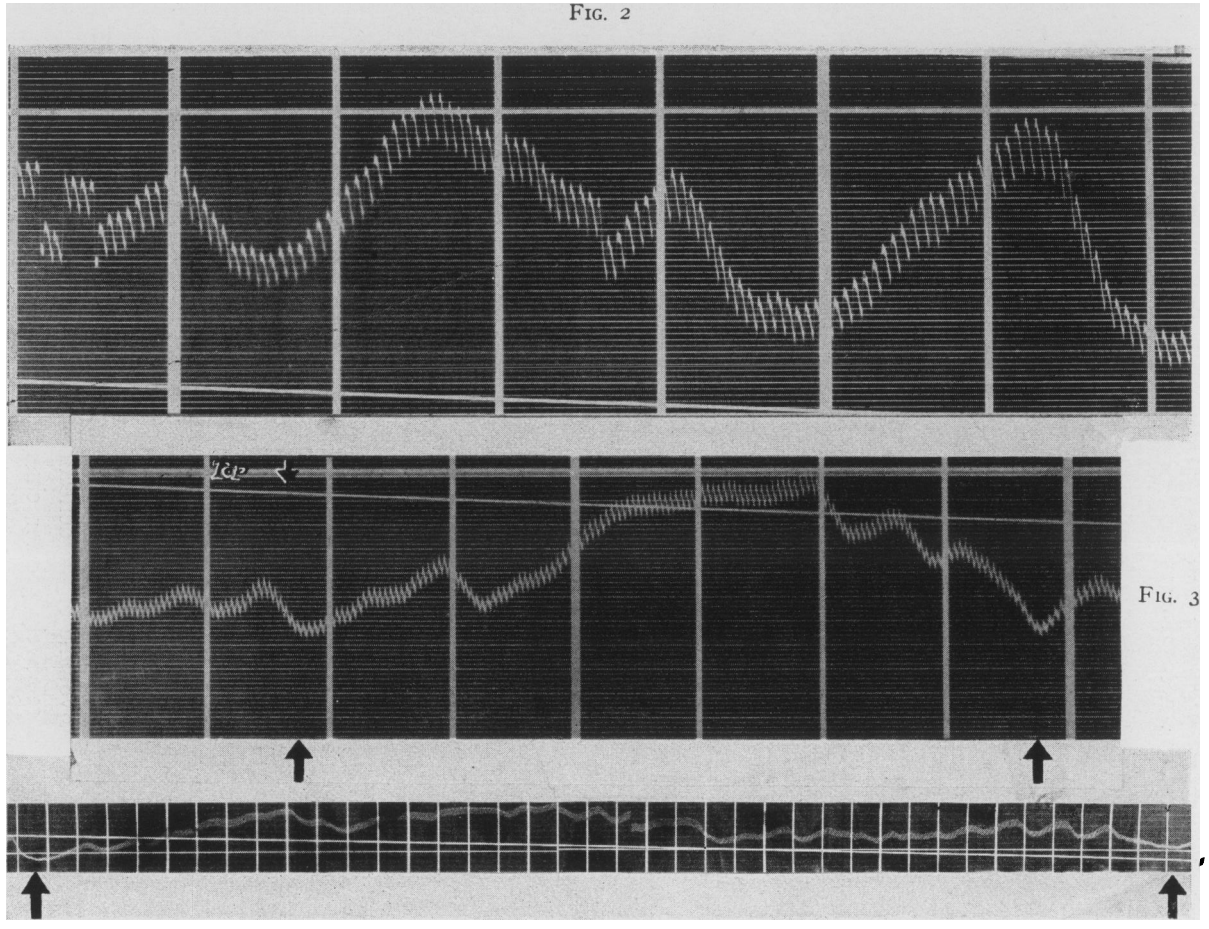

FIG. 2

FIG. 3

FIG. 4

FIG. 2.—Plethysmogram of normal individual showing alpha deflections represented by the marked variations in the base line.

FIG. 3.—Plethysmogram of normal individual showing a positive and a negative beta deflection extending between the arrows. Alpha deflections are superimposed upon the beta deflections.

FIG. 4.—Plethysmogram of normal individual showing a positive and negative gamma deflection extending between arrows. Alpha and beta deflections are superimposed upon the gamma deflections.

deflections, a significant part of these changes may be due to variations in lymph volume. It is possible, in addition, that changes in intercellular and intracellular volume contribute to them.

Gamma Deflections.—Gamma deflections (Fig. 4) develop more slowly than any of the other deflections. They are probably concerned with large, and usually with relatively slow, shifts in blood volume from one part of the body to another. They range in frequency from one to eight per hour and in volume from 50 to 350 cu. mm.[2] Although the relationship is not necessarily constant, they show a tendency to vary concordantly in the fingers, toes and pinnae. Like the other deflections, the gamma waves are predominantly the result of changes in volume of the vascular bed of the part, with variations in lymph volume probably contributing significantly to the volume change.

NORMAL VARIATIONS IN THE PLETHYSMOGRAM

The plethysomograms of normal individuals under certain circumstances present variations of considerable degree which may resemble the changes found in diseased states. For this reason it is desirable to review them briefly. Of particular importance among the factors which may produce such changes are (1) the psychic state of the individual, (2) the environmental temperature, (3) the relation of the part to the heart level, and (4) interruption of the sympathetic pathways to the part.

Psychic State.—Both the pulse and the alpha deflections may be greatly influenced by the psychic state. Fear, anxiety, or tenseness on the part of the subject, even as a reaction to the study, tend to produce a diminution in the pulse and alpha deflections and an increase in the rate of pulse deflections,[7, 8] apparently as the result of an increased sympathetic activity associated with the psychic disturbance which causes an increase in vasomotor tone. Any change in the mental state may affect the character of the spontaneous volume deflections. Flushing or blushing, for instance, is associated with an increase in the volume of the pulse deflections and a decrease in the alpha deflections, while if the individual is comfortable and relaxed, the volume of the pulse deflections is moderate and that of the alpha deflections is relatively large.

Environmental Temperature.—The spontaneous volume deflections are considerably influenced by the environmental temperature. Chilling the subject, either by local applications or in a cool room, produces vasoconstriction and a consequent reduction in the volume of the pulse and alpha deflections. In general, the degree of vasoconstriction is proportionate to the degree of chilling. After prolonged chilling, vasodilatation may supervene, the result being an increase in the volume of the deflections, in which the alpha deflections usually exhibit a relatively greater change than the pulse deflections. With prolonged chilling, however, a negative gamma deflection also occurs, indicating a slow but definite over-all decrease in the volume of the part. As the part warms, a positive gamma deflection appears, indicating restoration of local blood volume and an over-all increase in the volume of the part.

The application of heat, either locally or in a heated room, has the reverse

effect, that is, vasodilatation occurs, with a consequent increase in the volume of the pulse deflections and a decrease in the alpha deflections, while a positive gamma deflection appears, indicating an over-all increase in the volume of the part.

It has been suggested[1] that these reactions serve as good tests for organic occlusive arterial and arteriolar disease. In such conditions as thrombo-angiitis obliterans and obliterating arteriosclerotic endarteritis the vasodilating responses are impaired or absent. The effort to produce vasodilatation in the finger tips or the tips of the toes by the application of heat to another extremity tests the patency of the arteries and peripheral blood vessels to the part under investigation, as well as its neurovascular mechanism.

Relation of Part to Heart Level.—Spontaneous volume deflections are influenced by the position of the part in relation to the heart level. A decrease in volume of the pulse and alpha deflections may be produced by placing the part below heart level, the decrease being generally proportionate to the degree of lowering of the part. The pooling of blood and lymph in the dependent part, as a result of gravity, produces a positive gamma deflection.

The exact explanation of the decrease in volume of the pulse deflection has not been definitely established, but two factors, either alone or in combination, may account for it. The first is arteriolar constriction. The second, and more likely, is distention of the vessels, with a diminution in further distensibility of the vessel walls.[9]

Elevation of the part above heart level produces results just the opposite of those produced by dependency, that is, there is a pronounced increase in the volume of the pulse deflections, and, as a consequence of the draining out of the blood within the part, a negative gamma deflection. Obviously, for standard recordings, the part should be kept at or near heart level.

Interruption of Sympathetic Pathways.—The interruption of impulses over the sympathetic pathways produces profound changes in the spontaneous volume deflections. Within a few minutes after the regional sympathetic nerves or gauglia have been blocked by infiltration with 1 per cent procaine hydrochloride solution there is a marked increase in the volume of the pulse deflections and a virtual disappearance of the alpha deflections. The latter phenomenon would be expected, since these deflections are dependent upon intact sympathetic pathways.

It has been our observation that sympathetic block produces a maximum degree of vasodilatation in the part, which cannot be exceeded, and which is seldom equaled, by any other clinical procedure. During the early phases of sympathetic block the resulting engorgement of the vascular bed within the part produces a positive gamma inscription.

Sympathetic block furnishes another significant diagnostic test.[10, 11] By this means it is easy to determine the degree of the normal, or, in certain conditions, of the abnormal, vasoconstrictor tone and the extent of vasodilatation, or the general order of the amount of increase in vascularity of the part which can be achieved by interruption of the sympathetic pathways.

THE "BORROWING-LENDING" HEMODYNAMIC PHENOMENON (HEMOMETAKINESIA)

From what has been said, it is evident that spontaneous variations are constantly taking place in the vascular bed in different parts of the body, with resulting changes in the distribution of the blood volume. These changes may occur in widely separated parts of the body. They may be rhythmic, concordant, or discordant. They may be produced by intrinsic factors not yet well understood, or they may be influenced by various internal and external stimuli. Fundamentally, they indicate a continuous shifting back and forth of blood from one part of the body to another.[12]

It has been repeatedly demonstrated that there are spontaneous variations in the volume of certain internal organs, as well as in the volume of superficial structures. Experimental studies in animals have demonstrated intermittency or irregularity in the blood flow of the renal glomeruli,[13] tongue and skeletal muscles,[14] and ear.[15] Further experimental studies on the circulation in various organs by Zweifach[16] and Chambers and Zweifach[17] have confirmed the intermittency of blood flow in various parts of the body, a phenomenon which these workers term vasomotion. The spontaneous variations in splenic volume reported by several observers[18-22] are of a magnitude and frequency comparable to those of the beta and gamma deflections observed in plethysmographic studies of human subjects.

The alpha, beta and gamma deflections observed on plethysograms clearly indicate that spontaneous shifts in the blood flow are constantly taking place within the vascular system. There is evidently a continuous "borrowing and lending" of blood (hemometakinesia) to meet variations in local requirements.

The volume of the vascular system is quite variable within certain limits. In the average normal individual (a 70-kg. man) the system is filled with approximately 5,400 cc. of blood[23] at a pressure which varies from 120 to 80 mm. of mercury, with a mean pressure of about 93 mm. Great variations can take place in the vascular bed within a relatively short period of time, especially in isolated parts of the body, but the total blood volume, under ordinary conditions, remains relatively constant.

The vascular volume of a finger or toe can be observed to double in size within a matter of minutes. The filling of this augmented vascular bed with blood, however, obviously does not imply the doubling of the total blood volume of the body, nor is there reason to believe that there exists in the body a special reservoir of blood for such purposes. Yet the blood must come from some source. The obvious explanation is that is has come from the vascular bed of other parts of the body. It is achieved without any alteration at all in the total blood volume, by adjustment of the vascular bed (Fig. 5). An increase in the volume of the vascular bed in one part of the body and a decrease by the same amount in another part increases the volume of blood in the former at the expense of the latter without any variation in the total blood volume (Fig. 5).

With disease, local needs may increase and may remain elevated for long periods of time. Should an infection develop in a finger, for instance, blood is

shifted into the finger at the expense of other parts of the body with less urgent demands, and inflammatory hyperemia results. The shift is probably brought about by the local release, as a consequence of infection, of substances which produce arteriolar, capillary and venous dilatation. In other words, the "stop-cocks" are opened locally in the finger and more blood is borrowed temporarily from other parts of the body to aid in overcoming infection.

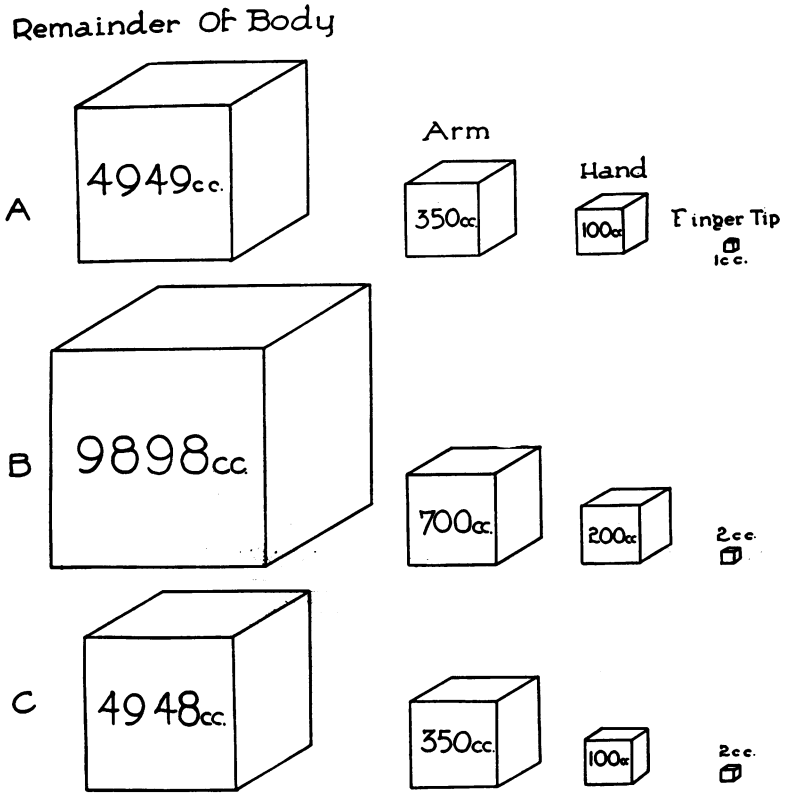

FIG. 5.—Schematic representation of the "borrowing-lending" hemodynamic phenomenon (hemometakinesia). Cubes are drawn to scale to represent volume of vascular bed in different parts of the body. A. Normal vascular bed, assuming a total blood volume of 5,400 cc. B. Vascular volume when component parts are all doubled. C. Vascular volume when blood volume of only one part, the finger tip, is doubled, with this increment of blood being derived or "lent" from the "remainder of body." It is apparent, when B and C are compared to A, the normal, that the same purpose is achieved in C as in B, the doubling of the blood volume of the finger tip, with no variation in the normal relationship in C but with obvious disturbance of it in B.

The amount of blood borrowed in this hypothetical case is relatively small, perhaps 1 cc., but it is sufficient to double, approximately, the normal local blood volume. An increase of this extent, however, in the blood supply of the finger does not disturb the general blood volume nor its hemodynamics. No effort is made by the body to double its general vascular volume in order to

double the volume in the finger. The body mechanisms do not produce for this purpose a generalized vasodilatation. This would not only be unnecessary. It would actually defeat the purpose. Were the vascular bed suddenly to double in volume, a great disproportion would develop, with equal suddenness, between the volume of the bed and the available supply of blood, and the subject, as a result, would collapse (Fig. 5).

With the total blood volume relatively fixed, it would be impossible to increase the circulating blood volume in any single part or tissue if there were a comparable increase in the volume of the entire vascular system. Furthermore, if the volume of the entire vascular bed were suddenly to double, and if the volume in one part, such as the hand, trebled, the drop in the intravascular pressure or blood pressure would be so great that the blood flow in the hand would be impaired rather than improved.

On the other hand, in the absence of generalized vasodilatation, even with maximum local vasodilatation the pressure head in the arterial system can be maintained, thus permitting in the local part an increase in volume of blood flow per unit of time. A response such as this is inevitable in order to obey fundamental principles of hemodynamics and at the same time to maintain normal circulation.

These spontaneous variations in blood volume in different parts of the body quite evidently are not mere chance occurrences. They seem, instead, to be part of a well regulated mechanism, with definite order and significance, concerned with certain vital functions such as proper thermal regulation, nutrition, repair, hemodynamics and other physiologic adjustments.

It is conceivable that at least a part of this significance lies in the attempt by the body to utilize its total blood volume in the most efficient possible manner. Obviously, the total blood volume in the body at any one time is too small to meet the maximum demand of all the tissues at the same time should a disease state arise to precipitate such a situation. On the other hand, it is more than sufficient to meet urgent local demands for large quantities of blood in isolated parts such as a finger, hand, foot, or even an entire extremity.

SPECIAL STUDIES

Numerous observations made upon normal individuals as well as on patients with various forms of peripheral vascular disease have invariably been consistent, regardless of the methods employed to determine and record variation in blood flow. Special studies have been made by thermometric and plethysographic methods.

These studies are done routinely, under controlled atmospheric conditions, in a room constructed to reduce psychic disturbances. Following rest in bed for a sufficient period of time to permit stabilization of the vascular system plethysmograms are obtained for the distal phalanges of the fingers and toes (usually the index finger and the second toe) as well as of the pinnae. Thermograms are obtained for these parts as well as for other areas; a total of 20 areas, distributed bilaterally and symmetrically, is usually observed. Plethys-

mographic and thermometric observations are usually made simultaneously. The response to such measures as interruption of sympathetic innervation, reactive hyperemia, drugs, environmental and local temperature changes, and psychic and neurogenic factors are observed as reflections in the plethysmograms and thermograms.

Observations made upon two patients, selected from the large number upon whom these studies were made, are presented to illustrate the "borrowing-lending" phenomenon (hemometakinesia):

First Patient.—G. Y., a white male 45 years of age, suffered with mild intermittent claudication due to early senile arteriosclerosis. Plethysmographic and thermometric studies were made after the patient had rested in bed in the observation room (room temperature 78° F., relative humidity 70 per cent) for 60 minutes. The skin temperature determinations were made bilaterally for the third toe, the dorsum of the foot, the mid pretibial area, the knee, the mid thigh, and the chest. The plethysmographic determinations were made for the distal phalanges of the right index finger and for the second toe on each side. After a state of stabilization had been reached, a left lumbar sympathetic block (first through fourth ganglia) was performed with 1 per cent procaine hydrochloride solution.

Within a few minutes after the performance of left lumbar sympathetic block the following events occurred simultaneously (Fig. 6):

1. A rapid elevation of the skin temperature of the left leg, with marked flushing.
2. A significant increase in the volume of pulse deflections of the left second toe.
3. An increase in the total volume of the tip of the left second toe.
4. A fall in the skin temperature of the right leg and the chest.
5. A decrease in the volume of the pulse deflections of the right second toe and right index finger.
6. A decrease in the total volume of the tip of the right second toe.

These changes indicated a marked vasodilatation in the left lower extremity after sympathetic block, with a considerable increase in the volume of blood and rate of blood flow. Blood was "borrowed" by the left leg at the expense of the remainder of the body. It was "lent" by the right leg and right index finger, as indicated by the cooling of the skin and the decrease in volume of pulse deflections and the total volume of these parts.

The expense of the shift, however, was not borne entirely by the right leg and right hand, since the vascular changes indicating a decrease in blood volume and blood flow were not as great as those reflecting the increase in the left leg. This might be expected. When relatively large quantities of blood are shifted to a part as large as the lower extremity, no single part of the body would be expected to suffer the entire loss, even if it were capable of "lending" it all. An index finger obviously would not be able to meet the whole demand, even if all its blood were shifted to the leg. Furthermore, if all the blood of a part such as the finger were shifted to the area of special need, the lending part would suffer serious damage from ischemia. Normal physiologic adjustments provide a mechanism whereby no organ is made to give up or "lend" its blood to an extent which would produce serious injury. Depending upon the quality and quantity of the shift, every organ that can afford to "lend" blood elsewhere does so. Under ordinary circumstances, however, each organ gives up only a small fraction of blood, and the changes reflecting a decrease in blood supply are relatively small in any one part under observation.

Second Patient.—J. H., a Negro male 34 years of age, who was suffering with a marked anxiety neurosis, was subjected to the same tests as the first patient and was also studied by certain additional procedures. A venipuncture was performed initially, with much deliberate manipulation, in order to produce pain and anxiety and to induce psychic

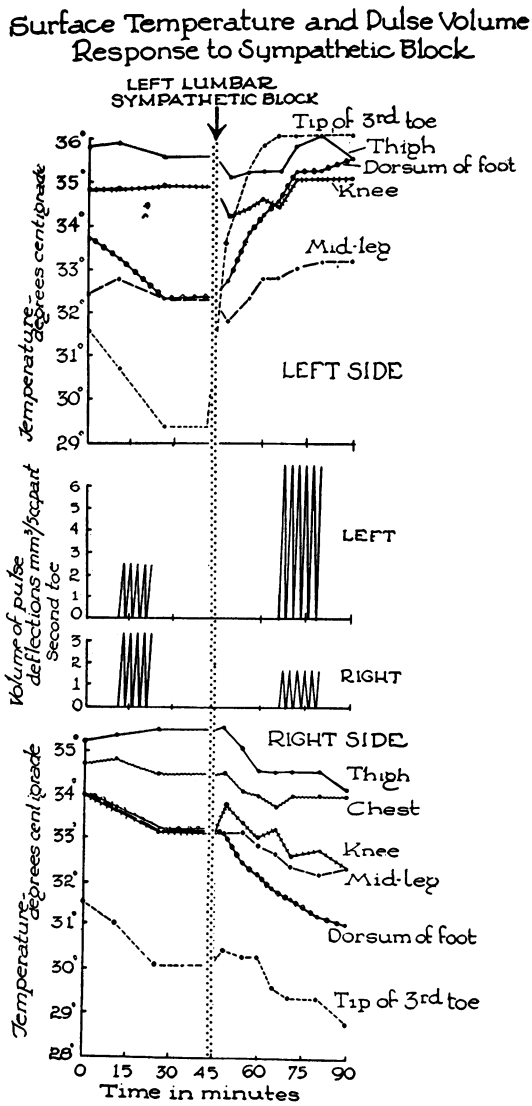

FIG. 6.—Surface temperature and pulse volume responses to left lumbar sympathetic block. It is obvious from the chart that following the block there was vasodilatation with increased blood flow in the left lower extremity, indicating the "borrowing," while on the right side there is evidence of diminished blood flow, indicating "lending" of blood.

tension and consequent diffuse vasoconstriction. After the vascular state had been stabilized at the level of vasoconstriction, 5 cc. (500 mg.) of tetraethylammonium chloride was injected intravenously. Then, after sufficient time had elapsed for the reaction to subside, a left posterior tibial nerve block was performed, using 1 per cent procaine hydrochloride solution. Two and a half hours later, a left lumbar sympathetic nerve block (first through fourth ganglia) was performed, using the same drug.

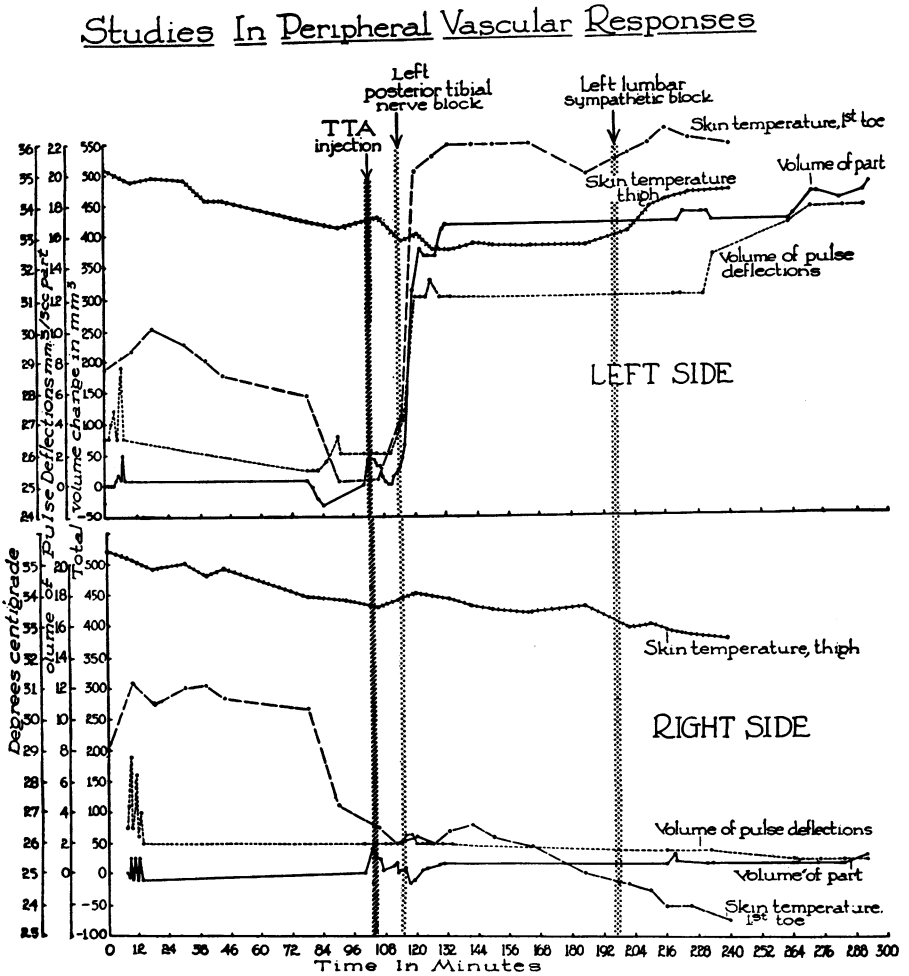

FIG. 7.—Graph showing thermometric and plethysmographic changes following injection of tetraethylammonium chloride (T. T. A.), left posterior tibial block and left lumbar sympathetic block.

The phenomena observed in the first patient are still further clarified by the results obtained in the second patient (Fig. 7):

The anxiety following the traumatic venipuncture resulted in considerable decreases in skin temperature, in the volume of pulse deflections, and in the total volume of the tips of the finger and toes studied. All these findings are evidence of diffuse vasoconstriction.

After the administration of tetraethylammonium chloride, which has been reported to produce vasodilatation,[24] there was little or no change in the skin temperature or in the

volume of the tips of the second toes, and only a slight increase in the volume of the pulse deflections of the finger and toes studied. Very little vasodilatation was to be expected. With generalized vasodilatation, each organ must "lend" and "borrow" at the same time, which is impossible unless the blood volume or cardiac output or both are increased to meet the demands occasioned by diffuse dilatation. Any drug which is a strong vasodilator would be expected at least to restore the state of the blood vessels of the superficial portions of the body to resting normal levels, which tetraethylammonium chloride did not do in this case. Furthermore, there is no evidence to show, nor any reason to expect, the sympatheticolytic effects to be greatest where the sympathetic tone is greatest. If the drug acts equally throughout the sympathetic ganglia, diffuse vasodilatation would be expected to result in little change in the blood supply to a particular part unless blood volume or cardiac output or both were increased.

Following left posterior nerve block the following changes were observed (Fig. 7):

1. An increase in the skin temperature of the left first toe.
2. An increase in the volume of pulse deflections of the second toe.
3. An increase in the total volume of the tip of the left second toe.

There were no changes on the right. The changes on the left, it should be noted, occurred in spite of the patient's state of anxiety and after the administration of tetraethylammonium bromide had failed to produce vasodilatation. Since the amount of blood that would be borrowed by a part as small as a foot to meet even maximum vasodilatation is relatively small, no measurable evidence of "lending" would be expected to be observed in other portions of the body, especially since each part might participate in supplying only a very small amount of blood.

At the end of two and a half hours there was relatively little change in the state of the circulation under observation. When left lumbar sympathetic block was done at this time, the following changes occurred:

1. A further increase in the temperature of the skin of the left leg and thigh.
2. A further increase in the volume of pulse deflections of the left second toe.
3. A further increase in the total volume of the tip of the left second toe.
4. A slight decrease in the volume of pulse deflections of the right second toe.
5. A slight but definite decrease in the temperature of the areas with intact sympathetic innervation.

After left lumbar sympathetic block, the volume of blood shifted to the whole left leg was evidently much greater than that following tibial nerve block, and, as in the first patient, evidence of a shift of blood from the right leg and right hand were obtained (Figs. 6 and 7).

DISCUSSION

The evidence now at hand seems to justify the following statements:

1. There is a continuous shifting back and forth of blood from one part of the body to another.

2. The "borrowing" and "lending" of blood (*hemometakinesia*) from tissue to tissue to meet variations in local requirements seem indicative of a well regulated mechanism which permits the body to utilize its limited total blood volume in the most efficient manner.

3. The essence of this mechanism seems to lie in the control and regulation of the vascular bed, which permits an increase in the volume of blood of one part of the body with a corresponding simultaneous decrease in the volume of blood in other parts.

4. The variation of the blood volume in different parts of the body does not involve any alteration in the total blood volume.

5. Variations in cardiac output, however, can produce changes in blood flow to a part independent of changes in blood volume.

The application of these principles of hemodynamics to peripheral vascular disease immediately suggests itself: Peripheral vascular disease is characterized by a disturbance of, or actual diminution in, the normal amount of circulating blood which reaches the part, and its effective therapy is based on improvement in the circulation or an increase in the blood supply of the part. Therapeutic measures designed to produce dilatation of the entire vascular bed do not seem rational for a disease state localized to a single peripheral part, but, in spite of the illogic of the attempt, there are numerous reports concerning the production of generalized vasodilatation as a form of therapy for peripheral vascular disease involving portions of the body. The results of these efforts are open to question; theoretically such measures are inefficient. Moreover, even if an effective agent, *i.e.*., with the ability to produce *maximum* vasodilatation, for this purpose did exist, its effects would be dangerous; the reaction would be shock-like, and the original purpose of increasing the blood supply to the local part would be completely defeated.

In view of the lack of rationale in peripheral vascular disease of the use of agents intended to produce generalized vasodilatation, quite aside from their ineffectiveness and theoretical dangers, the attention should be concentrated on measures which produce local vasodilatation. The best of these measures, in our experience, is sympathetic denervation of the affected part. It is rational, because it conforms with the principles of hemodynamics just laid down; it produces local vasodilatation insuring maximum improvement in the local circulation of the diseased part. It is safe, because it does not reduce the arterial blood pressure or produce serious systemic disturbances by the sudden creation of a disproportion between the total volume of the vascular bed and the total blood volume. Finally, it is effective. Theoretically, when this procedure is used, there should be a definite and significant increase in blood supply to the diseased part. Practically, there is such an increase, as is demonstrated by the two cases presented in this communication, which were selected from a large experience.

Indeed, we have yet to find a general vasodilator which could produce in a local part, such as the toes, fingers, foot, hand, or extremity, vasodilatation equal in degree or duration to that produced by sympathetic denervation of the part.

SUMMARY

1. Although gaps still exist in our knowledge of the hemodynamics of the peripheral circulation under normal resting conditions and in disease states, the evidence at hand justifies certain statements:

A. That there is a continuous shifting back and forth of blood from one part of the body to another; for this "borrowing-lending" mechanism the term *hemometakinesia* is proposed. B. That hemometakinesia seems to indicate the existence of a well regulated mechanism which permits the body to utilize its limited total blood volume in the most efficient manner to meet variations in

local requirements. C. That the essence of this mechanism seems to lie in the control and regulation of the vascular bed, which permits an increase in the volume of blood of one part of the body with a corresponding simultaneous decrease in the volume of blood in another part without any alteration in the total blood volume.

2. It is suggested that these principles of hemodynamics are applicable to the management of peripheral vascular disease, the effective therapy of which is based on improvement in the circulation or an increase in the blood supply of the part. Measures directed toward the improvement of the local circulation by production of dilatation of the entire vascular bed are of doubtful value, whereas measures directed toward local vasodilatation are in conformity with the natural "borrowing-lending" mechanism (hemometakinesia).

3. The most effective method of increasing the local blood supply is sympathetic denervation of the affected part. In addition, it is in complete conformity with the concept of hemometakinesia.

4. Two cases, selected from a large experience, are presented to illustrate the concept of hemometakinesia and to demonstrate the value of sympathetic denervation as a therapeutic measure in peripheral vascular disease.

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