Venomotor Tone in Hemorrhage and Shock

By ROBERT S. ALEXANDER, PH.D.

Employing the injection technic for venous distensibility determinations in intestinal loops of anesthetized dogs, an index to venomotor tone has been developed by measuring the volumes of blood required to produce specified changes in venous pressure. Application of this index in hemorrhage and shock experiments demonstrates a marked venomotor compensation to hemorrhage, and oscillations in venomotor tone correlating with the vasomotor waves in arterial blood pressure encountered in hemorrhagic hypotension. In the hypotension of shock, a serious deficiency in venomotor compensation develops, which should result in pooling of blood in the venous system and contribute significantly to the circulatory failure.

INTENSIVE searches for the primary hemodynamic defect in irreversible shock have failed to demonstrate the cause of the progressive fall in arterial blood pressure and reduction in venous return which characterize this condition. Considerable attention has been centered on possible dilator agents acting on the arteriolar and precapillary elements of the vascular bed. Hemodynamic studies, however, offer no evidence that reduction in total peripheral resistance, or decreases in resistance to blood flow of specific organs, are a dominant feature of shock. The idea that capillary leakage leads to dissipation of the circulating blood volume has also been discredited as a necessary phenomenon in the shock picture. The possibility of a defect on the venous side was championed years ago by Yandell Henderson, but adequate methods for demonstrating such a disturbance have not been available. Accumulation of evidence of pooling of blood in peripheral vessels in shock, however, has stimulated renewed interest in possible venous factors in the shock picture. As summarized by Wiggers: "A basic restudy of the dynamics of venous return is imperative, for our current fragmentary knowledge does not suffice to estimate the extent to which default of venopressor mechanisms can contribute to the circulatory failure of shock".

A new approach to this problem has become possible with the development of the distensibility technique for assessing the degree of constriction in the venous bed of the intestine. This report presents a method of quantitating these venous distensibility measurements so as to derive a "venomotor index", and describes the alterations of this venomotor index that have been observed during simple hemorrhagic hypotension and subsequent phases of shock.

EXPERIMENTAL METHODS

Dogs were sedated with morphine (5 mg. per Kg.), anesthetized with sodium barbital (180 mg. per Kg.) or chloralose (150 mg. per Kg.), and atropinized and vagotomized to minimize intestinal motility. A loop of ileum about 30 cm. in length was carefully isolated with preservation of its nerve supply, and arranged so that blood flow could be suddenly interrupted and the venous system of the loop injected with blood by a motor-driven syringe, according to the technique previously described. Measurement of venous pressure during the injection by means of an optical manometer gave a pressure-time tracing which, since the injection was performed at a uniform and known rate, was equivalent to a pressure-volume diagram of the venous system of the loop. These pressure-volume diagrams were recorded at ten minute intervals throughout the experiment; the remainder of the time the loop was perfused by the animal at its existing arterial blood pressure.

An optical manometer recorded the arterial pressure perfusing the loop, and the rate of venous outflow from the loop was recorded at intervals by means of the strain-gauge flow-meter. These data, together with the recorded venous pressure, were used to calculate resistance to blood flow through the intestinal loop in peripheral resistance units: A-V pressure gradient/flow in cc. per minute. Another manometer was connected to a femoral artery to provide for continuous registration of the arterial blood pressure of the dog.
Shock was induced by a minor modification of the procedure developed by Werle, Cosby, and Wiggers. Following completion of the surgical procedures and arrangement of the intestinal loop for venous distensibility measurements, control determinations were made and then the dogs were bled 0.5 per cent of body weight immediately after each distensibility determination (i.e., at ten minute intervals) until the mean arterial blood pressure had been reduced to approximately 50 mm. Hg. The dogs were held at this level for one hour, withdrawing additional blood as needed to maintain this blood pressure. The pressure was then reduced to approximately 30 mm. Hg by additional bleeding and kept at this level until it proved necessary to return a volume of blood equal to 0.5 per cent of body weight to prevent the blood pressure falling further, or until the dog showed signs of respiratory failure. When either of these two criteria was satisfied, all withdrawn blood was reinfused. The duration of the 30 mm. period, as determined by these criteria, averaged 40 minutes in the 12 dogs studied. This average conforms closely to the standard 45 minute period used by Werle and coworkers, though the 30 mm. period varied from 20 to 70 minutes in individual dogs. After reinfusion, the dogs were followed with venous distensibility determinations and flow measurements until the end of the experiment.

Method of Quantitating Venomotor Tone

It has been shown previously that the pattern of the distensibility diagram obtained with injection, and the width of the “loop” obtained in an injection-withdrawal cycle, are both significantly altered by contraction of the venous musculature. Using distensibility diagrams obtained in previous studies, we made a number of different types of measurements to determine how to quantitate the state of venomotor tone with the greatest sensitivity and reproducibility. The following discussion will be confined to the method which, on the basis of both theoretical considerations and empirical tests, appeared most useful.

As usually recorded, the initial pressure on the distensibility diagram is from 5 to 8 cm. of saline, this pressure having been selected to eliminate collapse phenomena from the distensibility determinations. When the injection is started, the pressure typically climbs fairly rapidly, then less rapidly, and then more rapidly again as high pressures are reached. The extent of this sigmoid characteristic is a function of the degree of venoconstriction, as has been documented previously and as is illustrated by the original recordings shown in figure 1. In constricted veins, the shoulder of the first inflection of the curve usually occurs at a pressure of about 20 cm. of saline. Between 10 and 20 cm. of saline, therefore, there will be a relatively small change in volume in a constricted vein. Beyond this first inflection, constricted veins show a large volume uptake in the 20 to 30 cm. range, while the dilated veins exhibit a reduced volume uptake. Taking advantage of this relationship, the venomotor index is defined as:

\[
\text{volume required to raise pressure from 20 to 30 cm.} \div \text{volume required to raise pressure from 10 to 20 cm.}
\]

The higher pressure interval is placed in the numerator so that venoconstriction will be indicated by an increase in the index. Its sensitivity obviously depends upon the fact that venoconstriction acts inversely on the numerator and the denominator. Since this index is dimensionless, measurements of the record do not need to be converted to actual volumes. With a constant injection rate and a constant speed of the recording kymograph, the horizontal distance between the respective pressure points may be measured in any convenient units. At our usual injection rates (see below) this index yields values which are convenient numerically, being a little greater than 1.0 for a normal animal, falling to a low of about 0.4 in maximal venodila-
Factors Influencing the Index Other Than Venomotor Tone

To determine the influence of injection rate on the calculated venomotor index, a series of injections were made at different speeds in random sequence in a normal unanesthetized dog, yielding calculated indices as shown in figure 2. Dependency of the venomotor index on injection rate was to be expected on the basis of earlier studies. Venous distensibility is characterized by significant delayed compliance. This phenomenon of slow distension is pressure dependent, manifesting itself to a much greater degree at the higher pressures. As a consequence, injection at rapid rates permits little time for delayed compliance and produces a steep distensibility slope over the 20-30 cm range. Slow injection rates permit considerable delayed compliance to take place over this pressure range, with a consequent increase in the corresponding distensibility volume. As a result, the calculated venomotor index varies inversely with injection rate.

This emphasizes the necessity of employing constant rates of injection if venomotor indices are to be compared in the same animal. In transferring data from one experiment to another, moreover, a problem arises from the fact that the effective rate of distension of the vessels depends upon the size of the particular vascular bed being injected, as well as upon the volume injected in unit time. In intestinal loops weighing from 200 to 300 grams, the best diagrams for analysis are obtained with injection rates which produce a pressure rise of 30 cm. saline in 5 to 8 seconds; a rate of distension which is satisfied by injection rates of from 25 to 50 cc. per minute. Control of the rate problem within this range permits a useful qualitative comparison of values obtained in different animals, in spite of the quantitative reservations in such comparisons which figure 2 dictates.

Figure 3 demonstrates the influence of varying the time lapse between injections. In confirmation of previous studies, an initial stretch alters venous elasticity so that an immediate repetition of the stretch reveals very little manifestation of venomotor tone in the distensibility diagram, producing a low venomotor index. With greater time intervals between injections, the distensibility pattern is restored so that essentially constant values for the venomotor index are observed after 30 minutes. In three experiments, where intervals between 30 and 60 minutes were investigated, there was no additional recovery which could be distinguished from the inability to maintain complete stability of the preparations over the periods of several hours which were required of such studies. In all cases, however, continued recovery has been clearly evident for 15 to 20 minutes.

This imposes a definite limitation in the applicability of this measure of venomotor tone. Not only does it dictate the necessity of rigorous control of the time between successive injections, but it also restricts the amount of information which can be obtained in the time span of an experiment. The ten minute interval which we have selected for standardizing our procedure represents a compromise, permitting enough recovery from the previous injection to provide adequate assessment of venomotor tone, at intervals that are short enough to yield adequate information during the course of an experiment. It has become our standard routine to submit the preparation to two successive cycles of injection and withdrawal of blood before performing the first “control” determination, with subsequent injections being made every fifteen minutes thereafter.

The studies described above have also provided an important control for the shock experiments pre-
Many preparations have been maintained for periods of five or six hours while making repeated distensibility measurements, without any significant alteration in blood pressure, demonstrating that preparation of the intestinal loop and the injection technique are not in themselves shock-inducing procedures. Evidence of the reproducibility of the venomotor index under stable circulatory conditions was also obtained. During the course of the experiment from which the data of figure 3 were obtained, for example, determinations of distensibility following a ten minute recovery were repeated six times over a period of 2.5 hours, yielding calculated indices whose maximum variation was from 1.08 to 1.20.

Two other technical problems arise in some experiments which interfere with the calculation of the venomotor index. Resistance to flow of the injected blood always results in a small upward displacement of the distensibility diagram which we subtract from the recorded pressures in identifying the points on the pressure tracing to employ in determining the venomotor index. In occasional experiments, this flow resistance is greatly augmented by spasm of the mesenteric vein just distal to the point of cannulation, which distorts the pressure tracing to such an extent that reliable distensibility values cannot be measured. In addition, alterations in the hydrostatic level of the intestinal loop produced by respiratory movements can interfere with accurate pressure measurement. Supporting the loop on a rigidly mounted platform usually eliminates this source of error. If the animal executes violent respiratory movements, however, traction on the narrow isthmus of mesentery which carries the nerve supply to the loop can seriously distort the recorded pressures. In a few cases, isolated records obtained during the hypotensive phases of the hemorrhage experiments had to be discarded because respiratory distortions prevented accurate calculation of the venomotor index.

**Experimental Results**

Of a total of 12 dogs submitted to the standard hemorrhage and shock procedure, one failed to show signs of shock when it was sacrificed two and one-half hours after reinfusion, another exhibited no recovery of blood pressure with reinfusion, and the latter portion of a third experiment had to be discarded because of technical difficulties. The remaining 9 animals all showed reasonable response to reinfusion of blood at the end of the hypotensive period and died in shock in from 30 to 270 minutes after reinfusion, the average survival time being 157 minutes. Six of these animals were maintained with barbital anesthesia, while three were under chloralose anesthesia. In spite of considerable variation in the exact course of the experiment from animal to animal, a survey of general trends revealed a striking similarity in pattern, particularly when the post-reinfusion periods were adjusted to equivalent time scales. This held true in spite of the different anesthetic agents employed; in this limited series there was no indication that chloralose altered the response to hemorrhage or the course of shock as compared to light barbital anesthesia.

It therefore appeared justifiable to summarize the results by averaging the data obtained from all 9 shock animals, as shown in figure 4. In calculating these averages, the following points were selected: the control values obtained before bleeding, the values obtained after each of the first four bleedings, values for the first half and the second half of the 50 mm. period, values for the first half and the second half of the 30 mm. period, values obtained immediately after reinfusion, and values for each quarter of the post-reinfusion period of normovolemic circulatory shock. In periods representing a number of individual determinations for each dog, the values for each animal were averaged separately before combining the data to obtain the over-all average, so that each animal contributed an equal weight to the data plotted in figure 4 regardless of variations in survival time. For comparison with these averages, the original data are shown for two representatives of the group in figures 5 and 6.
FIG. 5. Plot of data from an animal which exhibited moderate recovery with reinfusion and a reasonably long survival, but no evidence of venoconstrictor compensation to normovolemic shock. Venomotor index plotted in solid circles; peripheral resistance units in open circles.

FIG. 6. Plot of data from an animal which exhibited vasomotor waves following hemorrhage (arrows) associated with oscillations in venomotor tone, and early venomotor compensation to the hypotension of normovolemic shock, followed by venomotor failure in later phases.
**Hemorrhagic Hypotension**

Accompanying the initial bleedings, there was a steep rise in the venomotor index, demonstrating the expected venous constriction accompanying a fall in blood pressure. As the 50 mm. level of blood pressure was reached, this rise in the index was usually interrupted by either a fall (fig. 5) or a very erratic behavior (fig. 6).

This erratic behavior was of particular interest, since it was found to correlate with the appearance of vasomotor waves in arterial blood pressure (Traube-Hering waves) which are frequently observed at this stage. It was not possible to follow changes in venomotor tone throughout any single vasomotor wave, but inspection of the records, in which femoral arterial pressure was continuously recorded during the injection, revealed that high points of the venomotor index occurred during rising phases of blood pressure, while lower readings were obtained during falling phases of arterial pressure. This is indicated in figure 6, where arrows through the plot of mean arterial blood pressure indicate the direction of change in arterial pressure during the distensibility recording. The first peak of the plot of the venomotor index occurred during rising phases of blood pressure, while lower readings were obtained during falling phases of arterial pressure. This is indicated in figure 6, where arrows through the plot of mean arterial blood pressure indicate the direction of change in arterial pressure during the distensibility recording. The first peak of the plot of the venomotor index is followed by a sharp drop in the index, at which time arterial pressure was falling (first arrow). During the next determination, arterial pressure showed no significant change. The following determination exhibited a relative reduction in the index associated again with a falling phase of blood pressure. Ten minutes later the distensibility determination caught the vasomotor waves in a rising phase, and gave the highest index recorded in this experiment. Vasomotor waves continued for the next 20 minutes in spite of some rise in mean blood pressure; on the next determination the index was relatively lower when arterial pressure was falling, and then somewhat higher again during a rising phase of blood pressure.

In the average plot shown in figure 4, combining experiments in which the venomotor index fell during the 50 mm. period with those in which the venomotor index remained generally high but erratic, resulted in some reduction in the average venomotor index during the latter half of this period.

By the beginning of the 30 mm. period, the peak of the venomotor response had definitely given way to a progressive drop in the index in all but two of the twelve animals studied, although in only three had it fallen below the control values. As the 30 mm. period progressed, there occurred, usually rather abruptly, a fall of the venomotor index to values significantly below the control level. This evidence of venodilation coincided with a spontaneous fall in arterial blood pressure in the absence of further bleeding, which, at this critical level of hypotension, required small reinfusions to prevent a sudden exitus. This combination of venodilation and a spontaneous fall in blood pressure was seen in 11 of the 12 animals studied. The exception was an animal which showed no evidence of venomotor collapse and which maintained the 30 mm. level without the need of any small reinfusions for a period of 60 minutes. It is of interest that this is the animal which maintained an excellent arterial blood pressure following reinfusion and was sacrificed two and one-half hours later with no evidence of shock.

The flow data obtained during the bleeding and hypotensive phases are not reported here in detail since in general they conformed with the changes in peripheral resistance in the mesenteric vascular bed observed in this type of experiment by Selkurt and co-workers. Peripheral resistance units are included in figures 5 and 6, however, and illustrate the typical increase in resistance during hemorrhage and a generally high resistance through the 50 mm. period. Attempts to calculate resistance during the 30 mm. period were abandoned because of uncertainty as to how the calculated resistance should be evaluated at levels of extreme hypotension. Flow remained extremely low throughout the 30 mm. period, however, and neither our data, nor the data reported previously by Selkurt, reveal any evidence of significant dilation on the arterial side when, as described above, dilation is observed in the veins.

**Normovolemic Shock**

For a few minutes after reinfusion, the venomotor index remained low, often lower than just prior to reinfusion. Unfortunately the
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precise course of events could not be followed accurately, since distensibility determinations had to be kept to the ten minute schedule and the critical course of the experiment at this stage prevented any accurate timing of the reinfusion of blood in reference to this schedule. Thus in the experiment shown in figure 5, the first post-reinfusion determination of the venomotor index was made one minute after completing the reinfusion, while in the case illustrated in figure 6, seven minutes elapsed between the reinfusion of the withdrawn blood and the next distensibility determination, accounting for the recovery of the venomotor index in the latter instance.

Within 10 minutes after reinfusion, there was a recovery of the venomotor index and a relative stabilization of arterial blood pressure. The subsequent progress of normovolemic shock varied in different animals between two extremes. Animals exhibiting a fulminant type of shock demonstrated an incomplete recovery of the venomotor index, which then dropped off sharply to levels of extreme venodilation as the animal went rapidly into circulatory collapse. At the other extreme, animals with the longest survivals exhibited recovery of the venomotor index, which then dropped off sharply to levels of extreme venodilation as the animal went rapidly into circulatory collapse. At the other extreme, animals with the longest survivals exhibited recovery of the venomotor index, which then dropped off sharply to levels of extreme venodilation as the animal went rapidly into circulatory collapse.

Flow studies during the post-reinfusion period of normovolemic shock yielded results which were again quite similar to those reported by Selkurt. In cases of fulminant failure with rapid collapse of venomotor tone, intestinal blood flows were relatively high and resistances remained low. In animals showing longer survival, however, the calculated resistances recovered shortly after reinfusion to values that were generally in excess of those observed at similar pressures during hemorrhagic hypotension, and this elevated resistance was maintained throughout most of the shock period (figs. 5 and 6). There was no evidence on the arterial side of the circulation of a deficiency in constrictor tone, as judged by peripheral resistance, comparable to the observed deficiency in venomotor tone of the shock animal.

**Discussion**

Although the precise method of calculating the venomotor index has been derived as an empirical expedient, previous studies have
established a reasonably sound theoretical basis for this type of analysis of venous distensibility diagrams in terms of venomotor activity. The data accumulated in this study reinforce these theoretical arguments by their internal evidence of the validity of the measurement. The most convincing example of this was the correlation of vasomotor waves in the arterial blood pressure with corresponding alterations in the venomotor index (fig. 6). This demonstrates that the oscillatory behavior of the vasomotor centers following hemorrhage produces oscillations in venomotor tone as well as fluctuations in arteriolar tone. While it must be recognized that our injection technique is a very artificial departure from normal function of the veins in the intestinal loop, and that conclusions in regards to venomotor tone in the circulatory system must be extrapolated from this single segment of the vascular bed, these results have given us considerable confidence in the validity of this method for assessing venomotor tone.

On the basis of our general knowledge of the function of autonomic effector systems and our specific knowledge of the hemodynamic problems confronting the circulatory system, it would be logical to expect constrictor activity in the venomotor system to parallel constrictor activity on the arterial side of the circulation. This, indeed, has been found to be the case in a survey of pressure reflexes reported previously. Parallel function is here exemplified by the venoconstriction accompanying hemorrhage, and by the venomotor oscillations associated with vasomotor waves discussed above. The magnitude of the venoconstriction evoked by hemorrhage, moreover, indicates that this is contributing a significant role in the compensatory adjustments of the circulatory system. A normal animal, possessing a large potentiality for venoconstriction, can initially withstand an appreciable loss of blood with only a moderate impairment in venous return and arterial pressure. In contrast, it is well known that animals become much more sensitive to bleeding as arterial pressure is reduced to the level of 50 mm., where the capacity for further compensatory venoconstriction appears to be exhausted.

As a period of hypotension is maintained, however, there appears the first clear evidence in our experience of a dissociation between arterial vasomotor activity, and venomotor tone. While resistance to blood flow is maintained, venomotor tone tends to dissipate, most markedly toward the end of the 30 mm. period of critical hypotension. Venous dilation under these conditions was also observed in microscopic studies of the omental circulation by Zweifach and his associates. Study of the figures published by these authors reveals that the first evidence of failure in the peripheral vascular bed was dilation of muscular venules, usually preceding by a significant time interval any evidence of failure of other peripheral components. The functional importance of this venodilation is attested to by the necessity to infuse blood in order to counteract the fall in arterial blood pressure that is observed at this time. It is also significant that it is during this same period that the "irreversibility" of circulatory shock appears to be engendered.

In the post-reinfusion period of normovolemic shock, parallel collapse of both arterial and venous constrictor elements is evident only in cases of fulminant shock with rapid collapse and short survival. Venous dilation is associated with arteriolar dilation in these cases, not only in the intestine, but also in the general systemic circulation. Fulminant shock is also usually associated with profound disturbances in respiration, suggesting generalized failure of the controlling centers in the central nervous system.

In the more characteristic shock picture of a slowly but progressively declining arterial blood pressure, failure of venoconstrictor mechanisms is not associated with evidence of parallel failure on the arterial side of the circulation. Unless one is prepared to completely discount the ability to assess arterial vasomotor tone in shock by measurements of peripheral resistance, it must be concluded that, until terminal levels of blood pressure are approached, arterial constrictor tone is well maintained in the intestine, the spleen, the liver, the kidneys, and the legs. Studies of total peripheral resistance, though yielding somewhat variable results, exhibit little evi-
idence of arteriolar dilation in this type of shock. This places upon the venomotor system a major responsibility for the hemodynamic failure of the circulation in shock. It must be recalled, however, that in the long survival animals, the earliest portion of the decline in blood pressure is accompanied by some venoconstriction. Careful analysis of the data obtained from these animals made it clear that the deficiency in venomotor compensation to the declining blood pressure comes subsequent to, rather than at the time of, the first signs of hemodynamic failure. This forces us to one of two conclusions: either other areas of the venomotor system fail before the intestinal veins, or the primary initiating agent responsible for the start of the progressive circulatory failure resides in some other as yet unidentified hemodynamic factor.

Deficiency in the venoconstrictor response to a low blood pressure in the shock state offers an explanation for the fact that animals that have been submitted to shock-inducing procedures exhibit a significant deficiency in bleeding volume. This venomotor failure also throws new light on the process of blood pooling in shock. In the specific instance of the intestine, it has been shown in the dog that a disproportionate increase in hepatic resistance leads to the development of portal hypertension immediately after reinfusion and to some degree during the latter phases of normovolemic shock. On the basis of this portal hypertension, it has been argued that significant pooling of blood must occur in the mesenteric venous system at these times. It is noteworthy that these "critical phases" for pooling correlate not only with portal hypertension, but also with periods in which there is a serious deficiency in venomotor tone. Both factors should act synergistically to produce pooling of blood under these conditions.

Direct technics for measuring the volume of blood pooled in the venous system of the intestinal loop have not been devised. In accordance with the previous analysis of vascular distensibility, however, it is assumed that at high pressures the vessels have been stretched to the point that the inert fibrous tissue is limiting distension. It should therefore be possible to calculate back from the peaks of the recorded distensibility curves (fig. 1) an estimate of the volume change in the mesenteric venous system between hemorrhagic hypotension and the hypotension of normovolemic shock, using values for portal pressure under these conditions as reported in the literature. Selecting the periods when arterial pressure had fallen to the 80 mm. level for comparison, it was calculated in this fashion that the volume of venous blood in the loop would have averaged 2.6 cc. greater in the 9 animals in normovolemic shock than at the same pressure during hemorrhagic hypotension. In view of an average loop weight of 248 grams, this represents pooling of about 1 per cent of loop weight in normovolemic shock, compared with a bleeding volume of 1.5 per cent of body weight required to achieve this same pressure during the initial bleeding. Our assumption that the constricted vein would contain as great a volume as the dilated vein at the peak of the distensibility curve has recently been questioned, and, in any case, figs. 2 and 3 indicate that calculations based on injections made at the rate and time interval which we have employed would significantly underestimate the volume of blood that would be pooled in the intestinal veins in the intact circulation. Correcting for this would clearly bring the calculated venous pooling to the correct order of magnitude to explain the hypotension of shock. Transferring such calculations directly to the entire circulatory system, of course, ignores the unique hemodynamic features of the portal system. Nevertheless, these calculations do not detract from the hypothesis that venous pooling must be a significant factor in the hemodynamics of shock.

**SUMMARY**

An index to venomotor tone has been calculated from distensibility diagrams obtained by injecting the venous bed of an intestinal loop at accurately controlled rates and intervals. Application of this measurement to hemorrhagic hypotension and normovolemic shock indicates: 1. Venomotor tone rises sharply with hemorrhage. 2. Vasomotor waves in blood pressure, frequently produced by
hemorrhage, are associated with oscillations in venomotor tone. 3. During prolonged hemorrhagic hypotension, venomotor tone is not well maintained and tends to give way to venodilation, especially at critical levels of hypotension which are known to be shock-inducing. 4. When all withdrawn blood is reinfused, venomotor tone, after a brief period of persisting venodilation, returns towards normal. 5. In fulminant shock, characterized by a dilated arterial bed and a rapid deterioration of blood pressure, recovery of venomotor tone is incomplete and shortly gives way to venodilation. 6. In the more typical shock without evidence of significant dilation on the arterial side, venomotor tone recovers completely for a period and, in response to the earliest signs of a falling blood pressure, shows some compensatory increase. As shock progresses, however, a serious deficiency in venomotor compensation becomes evident.

It is concluded that failure of venomotor mechanisms is an important hemodynamic factor in shock, leading to pooling of blood in the venous system.

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