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EVIDENCE FOR CRITICAL CLOSURE OF DIGITAL RESISTANCE VESSELS WITH REDUCED TRANSMURAL PRESSURE AND PASSIVE DILATATION WITH INCREASED VENOUS PRESSURE

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Burton (1951) predicted from Laplace's law on the equilibrium of cylindrical tubes that if the pressure across the wall of a small blood vessel fell below a certain critical value, the vessel would close. Evidence supporting this theory of a 'critical closing pressure' was obtained in animal experiments by Nichol, Girling, Jerrard, Claxton & Burton (1951) and in the human digit by venous occlusion plethysmography (Gaskell & Burton, 1953; Yamada, 1954). Using the same technique Gaskell & Burton (1953) found that a small increase in digital venous pressure caused a large decrease in digital blood flow. They considered that this was caused by arteriolar constriction in response to the rise in venous pressure. Since the same result was obtained in a patient who had had a lumbar sympathectomy, they concluded that the constriction was mediated through a local nerve network and named the phenomenon the veni-vasomotor reflex.

The interpretation of plethysmographic records obtained under conditions which alter transmural pressure or raise venous pressure is sometimes difficult, and this is especially so in the digits owing to the small capacity of the digital vessels, the artifact produced by application of the collecting cuff and the effect of this cuff on the venous and arterial pressures. Gaskell (1955) and Allwood (1955) have shown that part of the evidence on which the veni-vasomotor reflex was based was due to a cuff artifact.

In the present experiments the effects on the digital vessels of reduced transmural pressure and increased venous pressure have been studied using heat elimination from the finger as a measure of the blood flow. This has the advantage that measurements may be made without interference with the circulation. Our results strongly support Burton's concept of a critical closing pressure but do not support the suggestion that arteriolar constriction occurs in response to a rise in venous pressure.

METHODS

The experiments were carried out on five normal subjects in a room at 21–23°C. Digital blood flow was estimated with heat flow disks (Hatfield, 1950; Catchpole & Jepson, 1954), the disks being stuck to the pulps of the terminal phalanges of the index fingers with 'Nobecutane' plastic dressing (Evans). With the subject comfortably seated the index fingers were inserted into digital plethysmographs (Greenfield & Shepherd, 1950) just above heart level. The water in the plethysmographs was maintained at 29°C and vigorous stirring was continued throughout the experiment. Precooling of the arterial blood arriving at the digits was minimized by wrapping the arms in electrically heated woollen cloth. As only the two terminal phalanges were in the plethysmographs, there was little opportunity for the blood to be precooled by the water in the plethysmograph before reaching the heat flow disks.

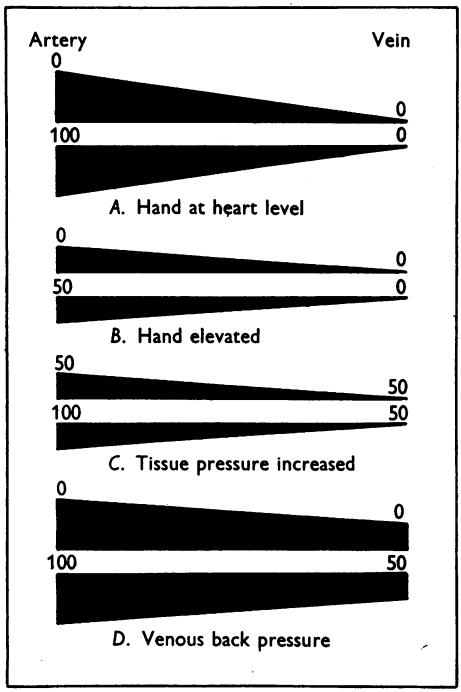

Fig. 1. A diagrammatic representation of the effects on transmural pressure of lowering the digital perfusion pressure by 50 mm Hg with hand elevation (*B*), positive tissue pressure (*C*), venous congestion (*D*). The transmural pressure is represented by the thickness of the vessel walls.

Fig. 1 shows diagrammatically three ways in which the perfusion pressure through the digit can be lowered. (1) When the hand is raised (Fig. 1 *B*) the arterial pressure in the digit falls by a value equal to that of the hydrostatic column of blood above the heart. The venous pressure does not fall since a negative pressure cannot be transmitted along a vein owing to collapse of the vein. Thus there is a reduction in both perfusion and transmural pressures. In some of the present experiments one of the plethysmographs was raised on bricks to various heights above the heart level. (2) When the tissue pressure is increased (Fig. 1 *C*) the venous pressure rises until it just exceeds the tissue pressure. This results therefore in a reduction in perfusion and transmural pressures similar to that seen when the arm is raised. To achieve this the inner jacket of one

plethysmograph was connected to a water manometer. The level of water in the manometer tube could be raised or lowered to exert any required hydrostatic pressure on the digit. Since the finger has such a large surface area in relation to its volume it is reasonable to suppose that changes in pressure brought about in this way were transmitted without decrement to all the soft tissues of the finger tip. (3) When the veins are congested (Fig. 1 *D*) the venous pressure rises until it is just sufficient to overcome the congesting pressure so that despite the decrease in perfusion pressure the transmural pressure in the veins and capillaries is increased. In the present experiments venous pressure was increased by inflating a pneumatic cuff round the base of the digit. A comparison was made of changes in heat elimination brought about in these three ways using the corresponding finger of the opposite hand as a control. The control finger was kept at heart level throughout.

Vasomotor tone was altered in one of two ways. In some experiments the ulnar nerve was blocked at the elbow with 3% (w/v) solution ethocaine hydrochloride with adrenaline (1:50,000) to cause release of sympathetic vasomotor tone in the little finger. In others body heating was carried out by immersing the feet and calves in water at 44° C and wrapping the subject in blankets (Lewis & Pickering, 1931: Gibbon & Landis, 1932).

Presentation of results. After a change in perfusion pressure had been made time was allowed for equilibrium to be established under the new conditions. This took from 5 to 10 min. Heat flow from the experimental and the control finger was then measured at ½ min intervals over the following 5 min period. The heat flow from the experimental finger was then expressed as a percentage of the heat flow from the control finger tip. Thus the results are independent of random fluctuations in vasomotor tone, which are assumed to be symmetrical on the two sides.

The arterial pressure was taken as the lowest pneumatic cuff pressure which gave the same heat flow from the finger tip as a cuff inflated to 220 mm Hg. Since the fingers were above heart level the venous pressure was assumed to be zero except during periods of venous congestion and increased tissue pressure when the congesting cuff pressure and the hydrostatic pressure of the water column respectively were assumed to equal the venous pressure.

The percentage heat flow was plotted against the perfusion pressure.

RESULTS

Fig. 2 shows the results of four experiments on two subjects. Each subject attended twice. On the first occasion the effects of elevation of the limb were compared with those of raised venous pressure. The heat flow from both digits was first measured to determine the degree of symmetry on the two sides. The left hand was then elevated and the measurements repeated. The hand was then lowered and the cuff around the base of the digit inflated to produce the same decrease in perfusion pressure and measurements again made. This sequence of events was repeated at each perfusion pressure. On the second occasion the effects of increasing the tissue pressure were compared with raising the venous pressure and the procedure was similar.

Increased resistance to blood flow with decreased transmural pressure

If the digital blood vessels behaved as rigid tubes and there were no anomalous blood-viscosity effects the blood flow would fall linearly with decreasing perfusion pressure irrespective of the method used to reduce the perfusion pressure (dotted lines, Fig. 2), since there would be no change in resistance to flow. With small reductions in transmural pressure (hand elevation or

increased tissue pressure) the fall in heat flow was slight. Further reduction in transmural pressure caused an abrupt and complete cessation of heat flow at a point where the perfusion pressure was still considerable, i.e. 35–60 mm Hg. It was thought possible that as the blood flow decreased the internal temperature of the finger might fall towards the temperature of the surrounding water, i.e. 29° C, and that vasoconstriction due to local cooling might explain the abrupt fall in heat flow. However, when the fingers were kept in water

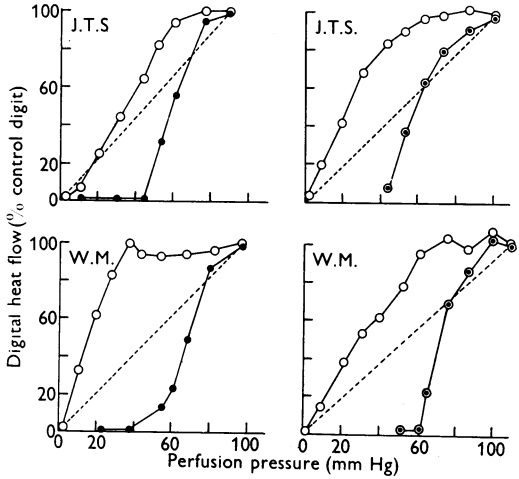

Fig. 2. A comparison of the effects of venous congestion and decreased transmural pressure on the heat flow from the finger tips in two subjects. Heat flow is expressed as a percentage of the heat flow from the corresponding finger of the opposite hand kept under control conditions at heart level. The dotted lines represent the expected fall in heat flow if the peripheral resistance remains unchanged. ○, venous back pressure; ●, positive tissue pressure; ⊙, hand elevation.

at 42° C and the rate of heat uptake by the finger was used as an index of blood flow, the flow decreased in a similar manner to that seen when the finger was immersed in water at 29° C. In water at 42° C diminution of blood flow would cause the internal temperature of the finger to rise. These findings were therefore consistent with the thesis that the vessels had closed when the transmural pressure fell below a critical value.

Decreased resistance to blood flow with venous congestion

When the perfusion pressure was lowered by venous congestion the digital heat flow was always greater than that following a comparable reduction in perfusion pressure brought about by an increase in tissue pressure or hand elevation. Heat flow ceased only when the perfusion pressure was reduced to zero. Application of a second congesting cuff at the wrist at the same pressure as the cuff at the base of the digit did not alter the heat flow so it was concluded that the digital cuff was effectively congesting the vein to the required

pressure. Since the decrease in heat flow was much less than would be predicted if the vessels behaved as rigid tubes it was concluded that resistance to flow had decreased in the digital vessels. Mendlowitz (1941) has shown that approximately 90% of the available heat is given off to water about 31°C during its unobstructed passage through the finger tip. Hence if the venous congestion did facilitate heat loss from the finger tip it could increase it at most by about 11%, and even if this correction is made, the same conclusion holds. When vasomotor tone was lowered by body heating the decrease in resistance to flow was greater than when vasomotor tone was high.

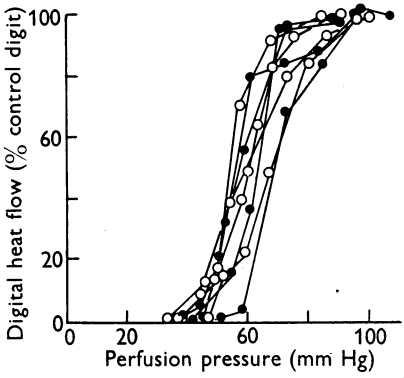

Fig. 3

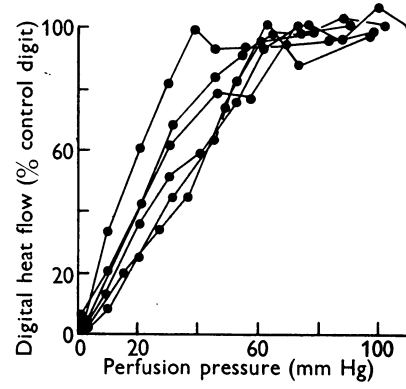

Fig. 4

Fig. 3. Critical closure of digital blood vessels during reduction of transmural pressure in four subjects. Heat flow is expressed as a percentage of the flow from the corresponding finger of the opposite hand kept under control conditions at heart level. ○, transmural pressure reduced by hand elevation; ●, transmural pressure reduced by positive tissue pressure.

Fig. 4. The effect of venous congestion on the digital heat flow in three subjects. Heat flow is expressed as a percentage of the flow from the corresponding finger of the opposite hand kept under control conditions at heart level.

The results of all the experiments on the four subjects are summarized in Figs. 3 and 4. The response to a decrease in transmural pressure was similar whether this was produced by raising the limb or applying a positive tissue pressure.

Critical opening pressure

Fig. 5 shows the result of an experiment where the transmural pressure was lowered until closure occurred and then raised again. It shows that the blood vessels re-opened at a pressure just greater than that at which they closed, and that the pressure flow loop is almost devoid of hysteresis.

Arteriolar tone and critical closure. In one experiment sympathetic vasoconstrictor tone was released in the blood vessels of the little finger by blocking the ulnar nerve at the elbow. The heat flow from that finger was compared with that from the intact index finger of the same hand as the transmural

pressure of the finger vessels was reduced by increasing the finger tissue pressures (Fig. 6). Critical closure was observed in the digit with lower vasomotor tone, but a greater reduction in transmural pressure was necessary to effect it. In another experiment where the transmural pressure was lowered until closure had just occurred, body heating caused the vessels to open again. These observations are consistent with the thesis that the critical closing pressure varies with alterations in vasomotor tone (Burton, 1951).

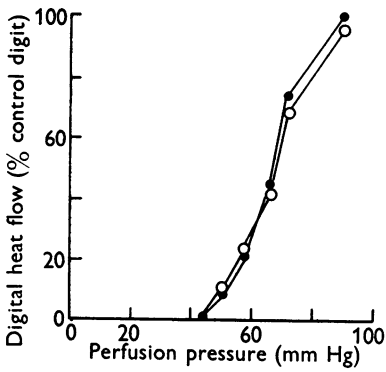

Fig. 5

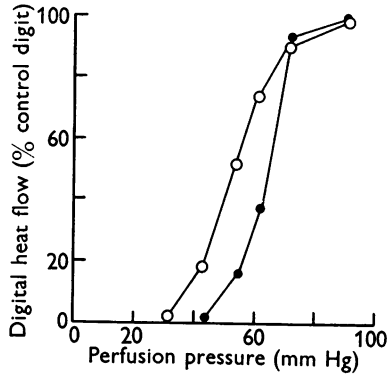

Fig. 6

Fig. 5. The effect of lowering the digital transmural pressure below critical closure level and then raising it again under comparable conditions of vasomotor tone. The transmural pressure was lowered (●) and raised (○) by increasing and decreasing the tissue pressure respectively. The heat flow is expressed as a percentage of the flow from the corresponding finger of the opposite hand kept under control conditions at heart level.

Fig. 6. A comparison of critical closure in the intact and the nerve-blocked digit during exposure to positive tissue pressure. The heat flow from the nerve-blocked digit (○) is expressed as a percentage of the flow from the nerve-blocked digit under control conditions at heart level. The heat flow from the intact digit (●) is expressed as a percentage of the flow from the corresponding finger of the opposite hand kept under control conditions at heart level.

The site of closure

When the transmural pressure was lowered, pulsations synchronous with the heart beat were observed in the water column with pressures at which flow through the digit had ceased. These pulsations persisted until the tissue pressure exceeded the arterial systolic pressure, but were abolished by occluding the digital arteries proximal to the plethysmograph, so it was evident that the pulsations were not transmitted from the proximal part of the finger. It was concluded that at a pressure where flow through the digit had ceased the arteries were still patent and that closure had occurred in the smaller vessels, presumably in the arterioles.

DISCUSSION

Critical closure of resistance vessels with decreased transmural pressure

The main difficulty in determining whether or not resistance vessels have a critical closing pressure lies in the inability to measure accurately the pressure in small blood vessels and to distinguish between vascular reactions and viscosity effects. The same difficulties have to be faced in studying the reactions of resistance vessels to increased venous pressure. In the present experiments these have been largely overcome by comparing heat flow in the same digit when the digital perfusion pressure had been lowered to the same level by methods which had different effects on transmural pressure.

With reduction in transmural pressure the heat elimination decreased abruptly. Although the estimation of perfusion pressure was indirect it was clear that blood flow ceased at a point when the pressure was still considerable since with venous congestion the pressure could be lowered some 30–60 mm Hg below this point before flow ceased. This difference is independent of any error that may have been made in the estimation of digital arterial pressure.

When the perfusion pressures and the viscous properties of the perfusion fluid are the same, differences in flow can only be initiated by differences in the calibre of the resistance vessels. The difference between the flows with the same reduction in perfusion pressure cannot be explained by viscosity effects alone and must therefore have been mainly due to changes in the diameter of the resistance vessels. The findings therefore strongly support Burton's concept of a critical closing pressure.

Dilatation of resistance vessels with increased venous pressure

The finding that the heat flow from the finger was unchanged during large reductions in perfusion pressure with venous congestion cannot be explained by greater facilities for heat exchange and must mean that the diameter of the resistance vessels was increased. This is in agreement with the findings of other workers in this department (Greenfield & Patterson, 1954; Shanks, 1955; Rosensweig, 1955; Roddie, 1955; Coles & Greenfield, 1956), and provides further evidence that venous congestion does not cause arteriolar constriction. The observations also confirm those of Shanks (1955) and Howell & Richards (1955) that the distensibility of the resistance vessels is greater when vasomotor tone is low.

There is evidence that when the transmural pressure of a blood vessel is increased, the tendency to dilatation is opposed by a myogenic constrictor reaction in the vessel wall and that, if the pressure is decreased, the tone of the smooth muscle of the resistance vessels is reduced (Bayliss, 1902; Folkow, 1949; Patterson & Shepherd, 1954; Coles & Greenfield, 1956). Since it is not possible to predict the dilatation that would occur if the vessels responded

passively to the increases in transmural pressure with venous congestion, these experiments provide no evidence for or against the concept of local myogenic reactivity in the blood vessel wall. With reductions in transmural pressure of the order of 5–20 mm Hg, the fall in heat flow was less than that predicted from the fall in perfusion pressure (Fig. 2) and this may have been due to decreased arteriolar tone.

SUMMARY

1. Digital blood flow was estimated by heat flow disks in five normal subjects during reduction of local vascular transmural pressure by either hand elevation or positive tissue pressure and during venous congestion.

2. When the transmural pressure was lowered the blood flow through the finger abruptly fell to zero when the perfusion pressure was still as much as 35–60 mm Hg. This finding was consistent with critical closure of digital resistance vessels, and as pulsations were still present in the arteries it must have been the finer vessels, presumably arterioles, that closed.

3. The critical closing pressure was the same as the critical opening pressure under comparable conditions of vasomotor tone, but both varied with changes in vasomotor tone.

4. When the perfusion pressure was lowered by venous congestion critical closure did not occur, but the resistance to flow through the digit decreased, probably owing to passive dilatation of resistance vessels in response to the increased intra-luminar pressure. This finding provides further evidence against the thesis of a veni-vasomotor reflex.

We wish to thank Dr W. H. Moffat for his collaboration in the earlier experiments.

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