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EARLY EFFECTS OF ABRUPT REDUCTION OF LOCAL PRESSURE ON THE FOREARM AND ITS CIRCULATION

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SUMMARY

1. The pressure at the surface of a segment of forearm enclosed in a plethysmograph was abruptly reduced from atmospheric to −20 to −120 mm Hg.

2. Forearm circumference (equivalent to the volume of a small segment of forearm (V_f)) was measured with a strain gauge. Pressure was measured in the plethysmograph (P_p), in veins exposed (P_{ve}) and not exposed (P_{vne}) to suction, in the brachial artery not exposed to suction (P_{bane}) and in forearm tissue (P_t).

3. Reduction of P_p caused increase of V_f. This was not due to gas evolution, since bubbles would not be liberated at the pressures employed. Nor was increase of V_f due to venous backflow since P_{ve} fell, but P_{vne} did not, even with upper arm circulation occluded or when P_{vne} was raised by venous occlusion prior to reduction of P_p.

4. Reduction of P_p temporarily arrested venous outflow since

$$P_{ve} < P_{vne} < P_{bane}$$

for 30 sec. With reduction of P_p 30 sec after occlusion of the upper-arm circulation, $P_{ve} < P_{vne}$ for > 1 min, indicating that arterial inflow was then minimal.

5. Increase of V_f, following reduction of P_p, was therefore due to inflow of arterial blood, of soft tissue or interstitial fluid. Interstitial fluid could flow from regions external to the plethysmograph, or enter as the result of filtration across capillaries. Occlusion of the upper arm circulation was not expected to interfere with motion of forearm soft tissue or the intra-tissue flow of interstitial fluid. It appears that capillary filtration is small compared with observed blood flow. Therefore subtraction of $V_{f_{occl}}$ measured at intervals after reduction of P_p (upper arm circulation occluded)

from V_f similarly obtained (but upper arm circulation free) appeared to give change of forearm volume due to inflow of arterial blood (ΔV_b). $\dot{V}_b$, the volume inflow rate of arterial blood during suction, was then obtained.

6. Resting forearm flow was 1·8 ml./min/100 ml. in seven normal subjects (average mean arterial blood pressure 86 mm Hg). With $P_p = -90$ mm Hg, $\dot{V}_b$ was 10·2 ml./min/100 ml. Suction therefore reduced vascular resistance, measured as $(P_{ba_{ne}} - P_{ve})/\dot{V}_b$.

INTRODUCTION

There have been a number of studies of the local circulatory changes that follow alteration of transmural pressure. Bayliss (1902) found the cat hind limb to vasodilate, after reduction of transmural pressure. Folkow (1949) showed that in normal, sympathectomized, and deafferented limbs in animals arterial occlusion was followed by vasodilatation. Folkow (1953) found that the venous outflow from a cat limb rose when the arterial perfusion pressure was increased, and fell when it was decreased, tending to return towards the previous value over the course of a minute. Greenfield & Patterson (1954) observed reduction of forearm flow, after exposure of a limb to subatmospheric pressure; and similar results were obtained on the human calf by Coles, Kidd & Patterson (1954).

There has however been little work on the circulatory changes that occur during, rather than after, this type of experiment in man. Blair, Glover, Greenfield & Roddie (1959) deduced, from measurements of venous blood oxygen content, that forearm blood flow was increased during the first 3 min of exposure to subatmospheric pressure. And by measuring venous pressure they inferred that increased transmural pressure had dilated resistance vessels.

But in none of these investigations in man were techniques used which would permit measurement of blood flow at the immediate time of application of subatmospheric pressure, when vasomotor responses might be minimal.

The present study is an attempt to analyse the early effects of local subatmospheric pressure on the forearm and its circulation in intact man. The findings suggest that it is possible to measure forearm blood flow, with a strain gauge, during the application of subatmospheric pressure, and further that subatmospheric pressure reduces resistance to blood flow.

METHODS

The subjects were nine healthy volunteers or patients without cardiovascular disease (Table 1). All but one were studied seated. Ambient temperature was approximately 22° C.

The subject's forearm was enclosed in an air-containing Perspex plethysmograph 15 cm in length, which was arranged at about right atrial level to minimize distension of forearm veins (Fig. 1). The forearm was supinated to prevent venous compression.

TABLE 1

Subject	Age	Sex	Diagnosis	Mean B.P.
F.G	26	M	Normal	88
M.S	27	M	Normal	83
T.F	32	M	Normal	73
C.C.	41	M	Normal	83
S.S.	21	F	Pleural effusion	97
V.S.	29	M	Pulmonary Tuberculosis	83
G.A.	33	M	Peptic Ulcer	93
A.F.	52	M	Peptic Ulcer	87
S.C.	65	M	Carcinoma of Bronchus	90

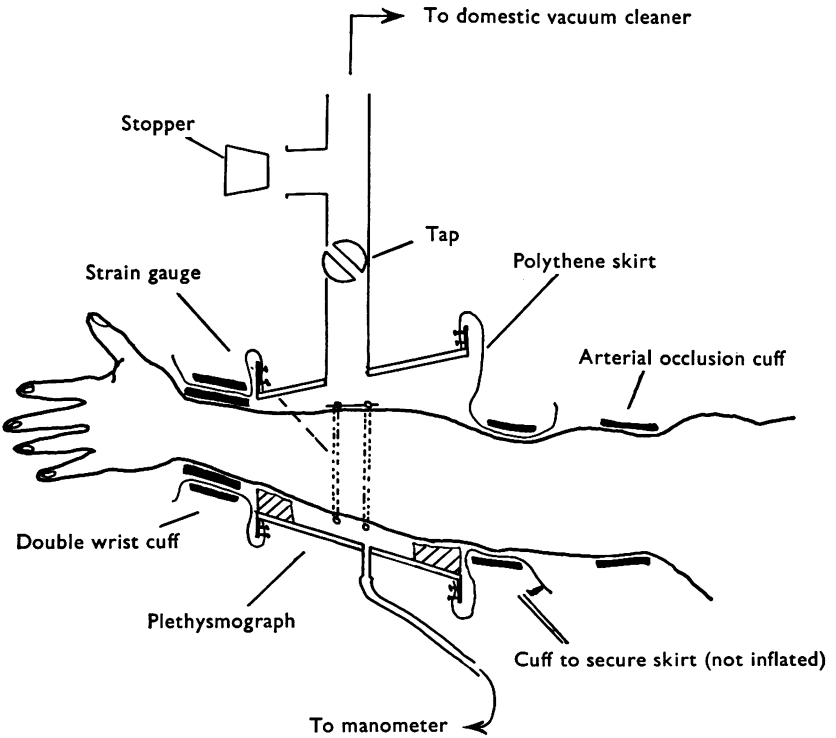

Fig. 1. Line-drawing of apparatus.

Seals were formed with polyethylene sleeves. Proximally, the sleeve was sealed to the skin of the forearm with short strips of adhesive tape, or passed under a firm uninflated blood pressure cuff, folded back on the cuff and sealed to its outer surface with adhesive tape. At

the wrist the sleeve was sealed to the outer surface of a blood-pressure cuff by means of a second cuff. Both were kept inflated to a pressure of 300 mm Hg and thus occluded the hand circulation. The inner cuff also increased effective cross-sectional area, making it comparable with that of the upper forearm. The forces (pressure × area) acting on the forearm at the two ends of the plethysmograph were thus equalized. As a result there was almost no longitudinal motion of the forearm with respect to the plethysmograph when pressure was reduced. An additional cuff was placed on the upper arm, and used to produce arterial or venous occlusion in some experiments.

Subatmospheric pressure was developed by two domestic vacuum cleaners arranged in series. An adjustable leak allowed pressure to be varied over the range 0 to −120 mm Hg. Suction was applied by suddenly closing the side arm of a T tube while the vacuum motors were running: 90 % of the final pressure was achieved in less than 1 sec. Plethysmograph pressure was measured with a C.E.C. (Devices Limited) strain-gauge pressure transducer.

Forearm circumference was monitored with a mercury-in-rubber strain gauge encircling a 3 cm wide section of the middle of the limb (Whitney, 1953). The original gauge had four strands of rubber (0·2 cm o.d.) in parallel. The tension at working length was 50–100 g and extensibility was 0·1 %/g. Resting tension was therefore higher and extensibility lower than is commonly used. A longer two-strand gauge, again 3 cm wide, was subsequently made, reducing resting tension to 20 g and doubling extensibility.

Suction on the forearm caused up to 3 % extension of the gauge (equivalent to a tension of 15 g). Taking account of forearm radius, the number of strands and the dimensions of the rubber, the pressure acting on the forearm was, with the newer gauge *ca.* 17 g/cm² in the resting state and 30 g/cm² at full extension. The stiffer gauge presumably gave lower absolute values for blood flow. However, the arguments advanced here are not invalidated, since they depend on changes, rather than on absolute levels of blood flow.

Static calibration was performed after removal of the gauge from the limb. The electrical output was linear, but only over a limited range, determined by the bridge circuit. When this range was exceeded during an experiment the bridge was either quickly rebalanced, or unbalanced before the experiment was repeated. These procedures did not alter static calibration. There was no output when the gauge, wrapped around a solid former, was exposed to the suction pressures used. Dynamic response was tested by subjecting the gauge to sinusoidal extension. The amplitude of current output was flat to 4 c/s.

Despite the rather large changes of forearm circumference, assuming forearm cross-section to have been circular, no serious error arose from directly equating circumference changes to changes in cross-sectional area. Changes of cross-sectional area were taken as proportional to changes of volume of the 3 cm section of forearm studied. Blood flow was expressed as ml./min/100 ml. tissue, at resting forearm volume. Correction for increase of forearm volume was unjustified, since blood flow increased by as much as 500 % while limb volume increased at most 20 %.

Brachial artery pressure was measured with a sphygmomanometer; mean pressure was calculated as diastolic plus one-third of pulse pressure. Venous pressure was measured in four subjects and brachial artery pressure in three subjects, using fine catheters attached to C.E.C. (Devices Ltd.) strain-gauge pressure transducers. Pressure was measured within forearm tissue in two subjects. A saline-filled needle was inserted into muscle and connected to a pressure transducer and flushing syringe.

The manometers and needle or catheter systems had natural frequencies in the region of 60 c/s. All manometers responded linearly to negative pressures up to −150 mm Hg. Records were made with 300 c/s galvanometers in an ultraviolet-light recorder (S.E. Laboratories S.E. 2000).

RESULTS

Reduction of plethysmograph pressure (P_p) to as low as -120 mm Hg for 30–60 sec caused negligible discomfort and no petechiae were seen.

Forearm tissue pressure (P_t) was measured at depths of 1·3 and 1·7 cm respectively in two subjects. In the relaxed forearm, P_t promptly followed changes of P_p, and $P_t/P_p \approx 1$ for static pressures up to -117 mm Hg, as

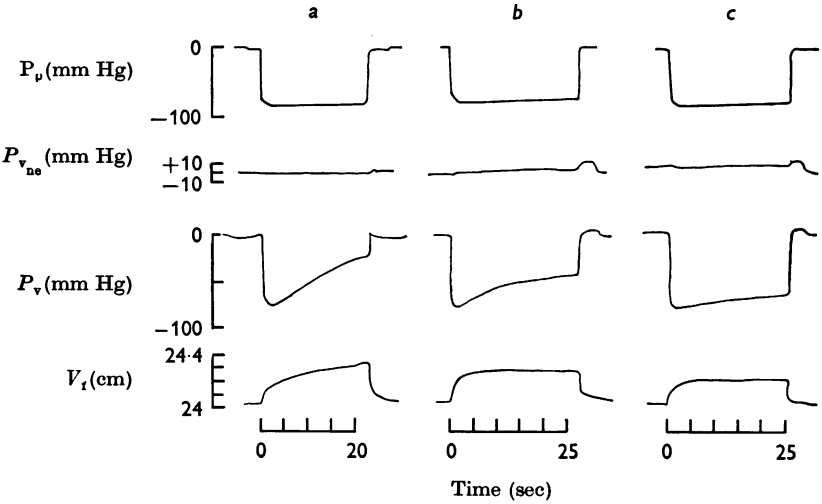

Fig. 2. Effect of reducing plethysmograph pressure (P_p) on pressure in forearm veins proximal to ($P_{v_{ne}}$) and within (P_{v_e}) the plethysmograph and on the volume of the forearm segment within the plethysmograph (V_f).

also found by Coles (1956). P_t could not be satisfactorily measured with forearm muscles tensed. P_t then responded sluggishly to changes of P_p, suggesting that needles were obstructed.

In Fig. 2 (subject V.S.) pressure was measured in a superficial forearm vein exposed to suction (P_{ve}) in a vein on the upper arm not exposed to suction (P_{vne}), and in the plethysmograph. A strain-gauge encircling the middle of the forearm recorded forearm volume (V_f). In Fig. 2*a*, P_p was rapidly reduced to -90 mm Hg, with the blood pressure cuff on the upper arm proximal to the site of measurement of P_{vne} uninflated. P_{vne} did not fall. P_{ve} fell rapidly to -90 mm Hg and then gradually returned to its original level over the course of 30 sec. The strain gauge recorded an initial rapid, followed by a more gradual increase of V_f. During the first 5–10 sec of this later period the rate of change of volume was approximately constant.

In Fig. 2*b* the upper arm cuff had been rapidly inflated to 300 mm Hg,

approximately 3 sec before reduction of P_p, P_{ve} again fell by an amount equal to P_p and then rose, but more slowly than in the experiment shown in Fig. 2*a*. P_{vne} was again unaffected by change of P_p. V_f showed an abrupt initial increase, but the rate of subsequent increase was considerably reduced (cf. Fig. 2*a*).

This experiment was repeated (Fig. 2*c*) but reduction of P_p was delayed for 30 sec after inflation of the upper arm cuff. P_{ve} again fell, but now rose extremely slowly (cf. Fig. 2*a* and *b*). P_{vne} was again unaffected. The slope of the later rise of V_f was not detectably different from that in Fig. 2*b*.

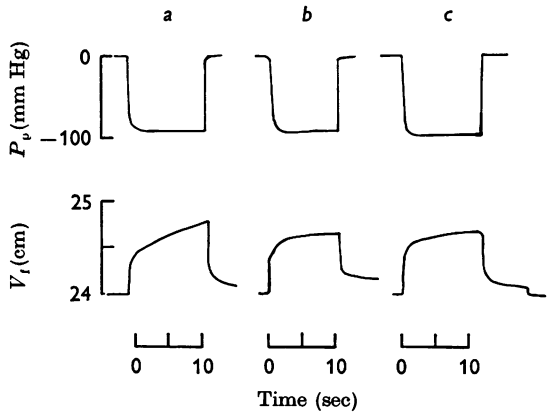

Fig. 3. Effect of reducing P_p on forearm volume (V_f). *a*, No arterial occlusion; *b*, arterial occlusion by cuff on upper arm at 300 mm Hg; *c*, brachial artery occluded by digital compression.

In the same subject (data not illustrated), P_p was rapidly reduced to −40 mm Hg. V_f increased abruptly and then more gradually. During the period of gradual increase of V_f the cuff on the upper arm was inflated to 60 mm Hg, i.e. 20 mm Hg in excess of suction pressure. This was without effect on the rate of increase of V_f, or of P_{ve}.

In Fig. 3*a* (subject M.S.), P_p was abruptly reduced to −80 mm Hg. In Fig. 3*b* the experiment was repeated after the upper arm cuff had been inflated to 300 mm Hg to occlude arterial and venous flow. In Fig. 3*c* the upper arm cuff was uninflated but the brachial artery was occluded by pressing it against the humerus. Care was taken not to exert pressure elsewhere on the arm, so as to ensure that blood could flow freely in the cephalic vein. The changes of V_f, following reduction of P_p, are almost identical in the latter two experiments.

In subject S.S. (data not shown) brachial artery pressure (P_{bane}) was measured with a catheter (0·1 cm o.d.). The open tip of the catheter (facing the direction of flow) was approximately 10 cm proximal to the elbow and thus in a region not exposed to suction. Rapid reduction of P_p

(upper arm cuff uninflated) caused no detectable change of mean P_{bane}. A short time after this experiment a strain gauge was applied to the subject's forearm. Reduction of P_p caused the usual changes of V_f, sub-

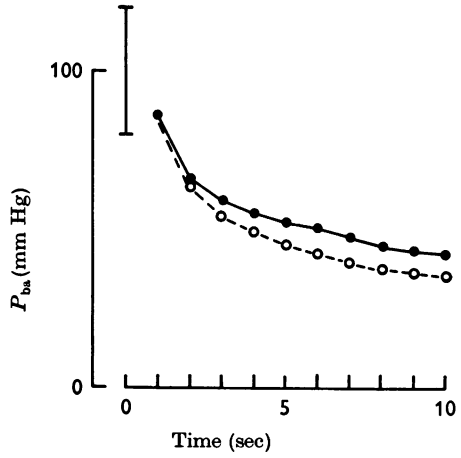

Fig. 4. Effect of reducing P_p on decay of brachial artery pressure following arterial occlusion. The vertical bar indicates pulse pressure. ●—●, P_p = 0 mm Hg; ○- -○, P_p = − 80 mm Hg.

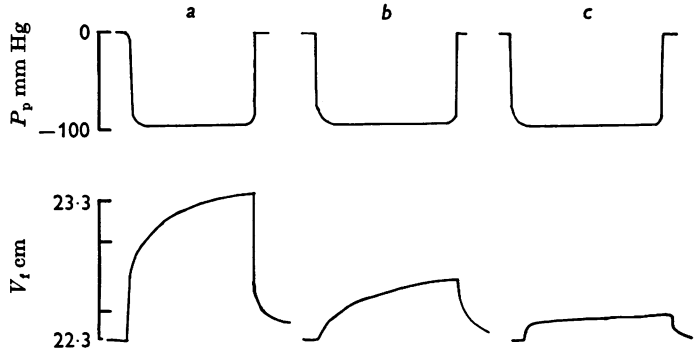

Fig. 5. Effect of reducing P_p on forearm volume. *a*, Forearm muscles relaxed; *b*, forearm muscles tensed; *c*, forearm muscles tensed with brachial artery occlusion.

sequently interpreted (see Discussion) as being due to increase of arterial inflow.

In Fig. 4 (subject S.S.) P_{bane} was measured while the upper arm cuff was abruptly inflated to 300 mm Hg. P_{bane} decayed as expected (●—●). When P_p was rapidly reduced to − 80 mm Hg, 1 sec after inflation of the cuff (○- - -○), the rate of fall of P_{bane} was increased. But when 30 sec elapsed between inflation of the cuff and reduction of P_p, change of P_p

had no detectable effect on the already slow rate of decay of $P_{ba_{ne}}$ (data not shown).

Figure 5*a* is a record of V_f (subject C.C.) when P_p was reduced to -90 mm Hg with the forearm relaxed, when the fist was clenched to

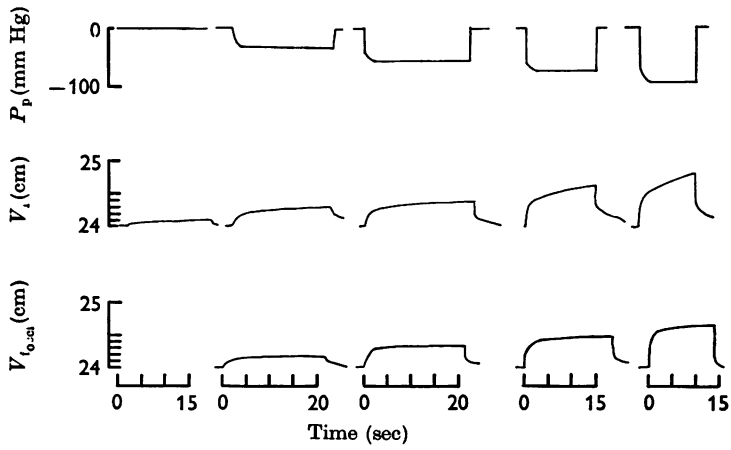

Fig. 6. Effect of reduction of P_p on V_f, with and without artery occlusion.

tense forearm muscles approximately 10 sec before reduction of P_p (Fig. 5*b*) and when the upper arm calf was inflated to 300 mm Hg with muscles tensed (Fig. 5*c*). Tensing muscles greatly reduced the initial change of V_f; but the later gradual phase persisted and the rate of change was similar to that with muscles relaxed. Inflation of the cuff reduced the later rate of increase of V_f.

In Fig. 6 (subject M.S.) graded suction was applied to the forearm over the range -30 to -90 mm Hg. The rate of later increase of V_f was greatest at the highest suction pressure. The rate of increase of V_f caused by a venous occluding cuff, i.e. resting blood flow (with $P_p = 0$), is also shown.

The lower panel of Fig. 6 ($V_{f_{occl}}$) shows the effect of applying the same suction pressures with the upper arm cuff inflated to 300 mm Hg before reduction of P_p.

Figure 7 shows curves obtained after point-by-point subtraction of the pairs of curves in the middle and lower panels of Fig. 6. The derived curves (zero time is the instant of reduction of P_p) are substantially linear and their slope in general increases with increase of P_p.

In Fig. 8 the slope of the curves in Fig. 7 is expressed as arterial blood flow (ml./min/100 ml. tissue) (see Discussion). Flow is plotted against P_p. Mean brachial artery pressure was 83 mm Hg. Resting forearm blood flow corresponds to $P_p = 0$. The relationship between P_p and flow is

non-linear, convex to the pressure axis. At $P_p = -90$ mm Hg, flow is approximately 7 times greater than the resting value.

Figure 9 resembles Fig. 8 but shows average values (plus 2 S.E.) for seven normal subjects. Mean brachial artery pressure was 86 mm Hg (S.E. 3·0) and resting flow again corresponded to $P_p = 0$. Average resting blood flow was 1·8 ml./min/100 ml. tissue (S.E. 0·2). Average blood flow with $P_p = -90$ mm Hg was 10·2 ml./min/100 ml. tissue (S.E. 1·0).

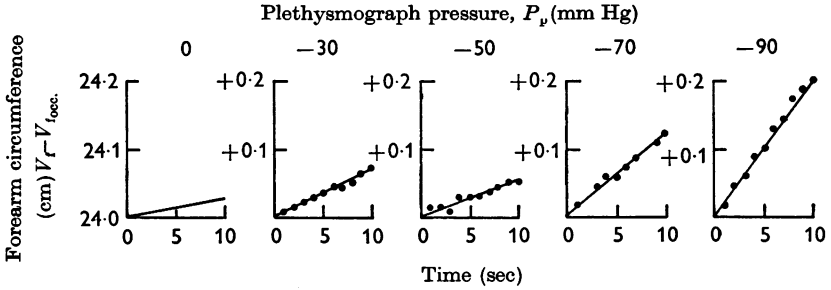

Fig. 7. Point-by-point subtraction of curves of forearm volume obtained with arterial occlusion from curves obtained without occlusion (see Fig. 6), for the first 10 sec after reduction of plethysmograph pressure.

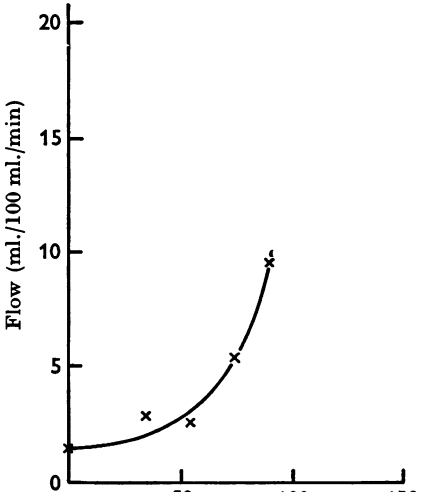

Fig. 8

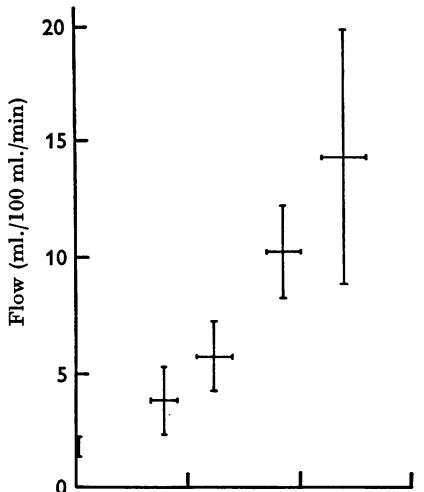

Fig. 9

Fig. 8. Blood flow ($\dot{V}_b$) plotted against P_p for subject M.S.

Fig. 9. Mean blood flow (± 2 S.E.) plotted against P_p for seven subjects.

DISCUSSION

In the system we studied, a segment of forearm was sealed into a rigid plethysmograph, by means of flexible sleeves. Wrist cross-sectional area was made comparable to that of the upper forearm, so that change of P_p caused almost no longitudinal motion of the forearm relative to the plethysmograph. The wrist cuff was kept inflated to 300 mm Hg to ensure that blood, or interstitial fluid, could enter and leave the forearm segment only in the direction of the upper arm.

Reduction of P_p caused V_f to increase. Increase of V_f could be due to inflow of soft tissue, blood and interstitial fluid from regions at higher pressure outside the plethysmograph, or to capillary filtration.

Both deep and superficial tissues and fluids would be affected by change of P_p, since P_t closely followed P_p. The pressures employed were insufficient to liberate gas bubbles (Harvey, 1951).

Consider first soft tissue. This could 'flow' in from the wrist (movement of wrist cuff) or from the upper forearm. Abrupt reduction of P_p caused a rapid, followed by a more gradual increase of V_f. The initial increase of V_f appeared to be due mainly to inflow of soft tissue, since it was greatly reduced by tensing the forearm muscles. Moreover, the initial increase of V_f was not affected by occluding arterial and venous flow in the upper arm, with a cuff. These are, however, qualitative findings and give no indication of the time course of soft tissue flow.

Consider next the effect of change of P_p on blood flow. The slope of the later increase of V_f was considerably reduced when the upper arm circulation was occluded. Clarification of the effects of P_p on blood flow came from combined studies of V_f and of vascular pressures. With upper arm circulation free, abrupt reduction of P_p caused an abrupt fall of P_{ve}, which then slowly rose. However, P_{vne} was unaffected, and $P_{ve} < P_{vne}$ for about 40 sec. While $P_{ve} < P_{vne} < P_{bane}$ there could be arterial inflow and venous backflow, but no venous outflow. It therefore remained to determine whether venous backflow occurred.

With the upper arm circulation occluded, P_{vne} was being measured in a system of veins closed at the proximal end. Yet reduction of P_p had no effect on P_{vne}, even when P_{vne} had been raised 20 mm Hg with a venous occlusion cuff before occlusion of the upper arm arterial and venous circulations. Venous backflow could therefore be excluded, and other experiments with a cuff and with digital occlusion of the brachial artery substantiated this conclusion. Absence of venous backflow must be attributed to the competence of numerous venous valves (Krogh, 1922, cited by Barcroft, 1963).

Increase of V_f, in the absence of venous backflow or of venous outflow,

must have resulted from inflow of arterial blood, of soft tissue or of interstitial fluid. The last could be derived from tissue spaces external to the plethysmograph or from capillary filtration. Further studies and arguments assist in distinguishing the contributions of these factors.

Abrupt reduction of P_p, immediately after inflating the upper arm cuff, caused P_{ve} to fall to equal P_p, but P_{ve} then rose more slowly than with the upper arm circulation free. When reduction of P_p was delayed, 30 sec after inflating the upper arm cuff, the later rise of P_{ve} was even slower—a finding consistent with there then being almost no inflow of blood into the veins.

Observations of $P_{ba_{ne}}$ support this conclusion. Abrupt reduction of P_p, immediately after inflating the upper arm cuff, increased the rate of decay of $P_{ba_{ne}}$ over that seen when there was no reduction of P_p. However, when reduction of P_p was delayed 30 sec after inflation of the upper arm cuff, the rate of decay of $P_{ba_{ne}}$, already slow, was hardly changed. Thus arterial 'run off' may have been nearly complete, and little affected by change of P_p.

We now write expressions to denote the changes of V_f that occurred following abrupt change of P_p. Let ΔV_f be the amount by which forearm volume has increased at any time t, after the instant of reduction of P_p and let $\Delta V_{f_{occl}}$ be the volume increase at the corresponding time, with upper arm circulation occluded.

We have argued that ΔV_f will at time t include increments of soft tissue volume (ΔV_{st}), of blood volume (ΔV_b), and of interstitial fluid volume (ΔV_{if}).

Hence with upper arm circulation free we write for time t:

$$\Delta V_f = \Delta V_{st} + \Delta V_b + \Delta V_{if}. \quad (1)$$

The effect of reduction of P_p on the movement of soft tissue and interstitial fluid, from regions external to the plethysmograph, was not expected to be influenced by occlusion of the upper arm circulation. However $P_{ba_{ne}}$ fell to about 35 mm Hg 10 sec after such occlusion and capillary pressure too presumably fell. The hydrostatic gradient for capillary filtration would therefore be smaller with upper arm circulation obstructed rather than with it free.

Since the magnitude of change in this gradient is unknown we consider the possible rate of filtration with a gradient between normal capillary pressure and $P_t = -90$ mm Hg. No precisely relevant observations have been found but Landis & Pappenheimer (1963) quote filtration coefficients ranging from 0·0057 to 0·0105 ml. min^{-1}. 100 g $tissue^{-1}$. mm Hg^{-1}. If these are applicable, the maximum filtration rate in the present experiments would be of the order of 1 ml./min/100 ml tissue. As subsequently shown

blood flow rates 10 times as high were recorded with this level of suction, so that it seems permissible, as a first approximation, to discount capillary filtration here.

Adopting this course we can write for time t after reduction of P_p, with upper arm circulation occluded,

$$\Delta V_{f_{occl}} = \Delta V_{st} + \Delta V_{b_{occl}} + \Delta V_{if}. \quad (2)$$

We thus assume that the first and third terms on the right are the same as in equation (1).

Subtracting we obtain

$$\Delta V_f - \Delta V_{f_{occl}} = \Delta V_b - \Delta V_{b_{occl}}. \quad (3)$$

We cannot define $\Delta V_{b_{occl}}$, but we have shown that there is, particularly with delay between inflation of the upper arm cuff and reduction of P_p, a very slow rise of P_{ve}. We argue that $\Delta V_{b_{occl}}$ then approaches zero. Under these circumstances subtraction of (2) from (1) yields ΔV_b at various instants of time after reduction of P_p. The slope of increase of ΔV_b with time gives, therefore, the volume inflow rate of blood, $\dot{V}_b$. Other evidence has been advanced that $\dot{V}_b$, while $P_{ve} < P_{vne} < P_{ba_{ne}}$, and before distension reduces the extensibility of capacity vessels, is the volume inflow rate of arterial blood. The subtraction procedure also cancels any error signal arising from change of temperature of the strain-gauge during suction.

Consider mechanisms by which change of P_p affects $\dot{V}_b$. These are complicated and will require additional studies for their elucidation. However, P_t closely followed P_p, and we assume that pressure at the surface of vessels in the segment was close to P_t. Then with abrupt reduction of P_p all vessels will experience increase of transmural pressure and start to dilate. The time taken for completion of dilatation may, if inertial factors can be ignored, substantially be determined by the product of the interposed resistance to blood flow and the compliance of each vessel. From this argument dilatation of arteries will be completed almost instantaneously. The later phase of increase of V_f is therefore presumed to represent dilatation of vessels having greater compliance and offering a higher resistance to inflow.

The relationship between P_p and $\dot{V}_b$ was non-linear, convex to the pressure axis. With $P_p = -90$ mm Hg, $\dot{V}_b$ was on average (seven normal subjects) about 5 times greater than resting forearm blood flow.

Since P_t closely followed P_p, we can assume that reduction of P_p is equivalent to increasing pressure at the surface of the rest of the body, with P_p held constant. Once the flow of soft tissue is complete, reduction of P_p is then analogous to increasing both arterial inflow pressure ($P_{ba_{ne}}$)

and venous outflow pressure (P_{vne}) by P_p. The pressure gradient across the segment is unaltered. But $P_{bane} - P_{vne}$ cannot be the appropriate pressure gradient, since there was, while $P_{ve} < P_{vne} < P_{bane}$, no venous outflow.

An alternative and more realistic gradient is $P_{bane} - P_{ve}$. Under these conditions, if mean $P_{bane} = 90$ mm Hg and $P_p = -90$ mm Hg, the gradient is at its maximum doubled. Yet flow was increased 5-fold, showing that there was increased conductance $[\dot{V}_b/(P_{bane} - P_{ve})]$ of the segment. Similar changes of conductance were observed by Green, Lewis, Nickerson & Heller (1944) on altering perfusion pressure in isolated perfused animal limbs. Other studies are however needed to determine whether the gradient responsible for flow is $P_{bane} - P_{ve}$, or whether a 'waterfall' mechanism (Permutt & Riley, 1963) exists.

The absence of change of mean P_{bane}, with increase of arterial flow deserves comment. The brachial artery catheter measured lateral plus stagnation pressure. But even assuming a resting mean blood velocity of 10 cm/sec, which was increased by suction to 50 cm/sec, the recorded pressure would not increase by more than about 1·3 cm H_2O. Increase of blood inflow could be achieved, without appreciable fall of P_{bane}, by virtue of the low resistance to flow in larger arteries, and of blood being diverted from parallel channels.

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REFERENCES

BARCROFT, H. (1963). Circulation in skeletal muscle. In *Handbook of Physiology*, vol. II, sect. 2, *Circulation*, ed. HAMILTON, W. F. & DOW, P. Washington, D.C.: American Physiological Society.

BAYLISS, W. M. (1902). On the local reaction of the arterial wall to change of internal pressure. *J. Physiol.* **28**, 220–231.

BLAIR, D. A., GLOVER, W. E., GREENFIELD, A. D. M. & RODDIE, I. C. (1959). The increase in tone in forearm resistance blood vessels exposed to increased transmural pressure. *J. Physiol.* **149**, 614–625.

COLES, D. R. (1956). Observations on the responses of human blood vessels to local alterations in transmural pressure. M.D. Thesis, University of Bristol.

COLES, D. R., KIDD, B. S. L. & PATTERSON, G. C. (1956). The reactions of the blood vessels of the human calf to increases in transmural pressure. *J. Physiol.* **134**, 665–674.

FOLKOW, B. (1949). Intravascular pressure as a factor regulating the tone of the small blood vessels. *Acta physiol. scand.* **17**, 289–309.

FOLKOW, B. (1953). A study of the factors influencing the tone of denervated blood vessels perfused at various pressures. *Acta physiol. scand.* **27**, 99–117.

GREEN, H. D., LEWIS, R. N., NICKERSON, N. D. & HELLER, A. I. (1944). Blood flow, peripheral resistance and vascular tonus, with observations on the relationship between blood flow and cutaneous temperature. *Am. J. Physiol.* **141**, 518–536.

GREENFIELD, A. D. M. & PATTERSON, G. C. (1954). Reactions of the blood vessels of the human forearm to increases in transmural pressure. *J. Physiol.* **125**, 508–524.

HARVEY, E. N. (1951). In *Decompression Sickness*, ed. FULTON, J. F. Philadelphia and London: W. B. Saunders.

LANDIS, E. M. & PAPPENHEIMER, J. R. (1963). Exchange of substances through the capillary walls. *Handbook of Physiology*, vol. II, sect. 2, *Circulation*, p. 990, ed. HAMILTON, W. F. & DOW, P. Washington, D.C.: American Physiological Society.

PERMUTT, S. & RILEY, L. R. (1963). Haemodynamics of collapsible vessels with tone: the vascular waterfall. *J. appl. Physiol.* **18**, 924–932.

WHITNEY, R. J. (1953). The measurement of volume changes in human limbs. *J. Physiol.* **121**, 1–27.