Reactive Hyperemia in the Human Finger

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Reactive hyperemia in the finger was investigated in 16 experiments on 5 normal subjects, using venous occlusion plethysmography. Since the flow is so variable from moment to moment, the simultaneous "corrected" flow in a control finger, not occluded, was subtracted from the flow in the test finger, so the difference gave the effect of occlusion free from fluctuations of central origin. The absolute amount and the duration of extra blood flow increased with the period of occlusion. The percentage of the calculated "blood flow debt" repaid was variable, with a mean of 51 per cent ± 36 S.D. (SEM ± 6 per cent). This is greatly in excess of nutritional debt of the skin.

REACTIVE hyperemia of tissues, following occlusion of the blood supply, has interested physiologists for a long time. It has been studied extensively in the muscle. Although known to occur in the skin, there is very little quantitative data available on the subject; Lanier and colleagues have even denied its presence in any significant amount in the hind leg of a dog.

We have attempted to study some quantitative aspects of reactive hyperemia in the skin, by measuring blood flows of both index fingers (test and control) simultaneously with a plethysmograph. It is assumed that the finger blood flow mainly represents the blood flow in the skin of the finger and that the small bone flow may be neglected. The normal fluctuations in blood flow from second to second in the finger are very great and obscure any experimental changes. However, Burton has shown that these fluctuations relative to mean flow are simultaneous and nearly equal in all digits. This suggests the possibility of using the blood flow of another finger at the same instant as control. In order to do this we have multiplied the flow values of the control finger by a factor which equates the resting flows of the 2 fingers. This will henceforth be referred to as the "normalized control flow." We feel that the difference between the test and the normalized control flows is the best estimate of the magnitude of changes due to the experimental procedure.

METHOD

The plethysmographs consisted of light glass test tubes which enclosed the finger up to the proximal interphalangeal joint. The plethysmograph was not externally damped. Some plastic sealing compound (Tremco strip-seal, Tremco Manufacturing Co., Canada) was used for sealing and was found to be very satisfactory. The outlets of the tubes were connected by Tygon plastic tubing (the distensibility of which is low) to a metal capsule with a thin butyl rubber membrane (Polymer, Sarnia, Ontario, Canada). The deflection of a mirror on the edge of the membrane was recorded on a photokymograph. A venous occlusion cuff was wrapped around the adjacent phalanx of the finger and was inflated suddenly from a large pressure bottle with a collecting pressure usually from 30 to 60 mm. Hg. For occluding the blood supply a pressure of 200 mm. Hg. was used. This method has been described in detail by one of us.

Sixteen experiments were performed on 5 normal subjects. They rested for about 15 min. before each experiment in a constant temperature room (25 C. ± 0.5). The subject was in a supine position, the hands resting on a wooden bridge slightly above the heart level. Flow measurements after release of occlusion were made every 6 sec. continuously for 5 min. and then at 7 and 10 min. Rates of flow could be estimated from the records with an accuracy of about 5 per cent, except in unusual cases. The volume of the finger within the plethysmograph was measured by displacement of water.

RESULTS AND DISCUSSION

A representative plot of 1 experiment is shown in figure 1. The absolute amount of reactive hyperemia is expressed, in ml./100
cc. of finger, as the difference between the flow in the test and the normalized control fingers. The total debt acquired is calculated in the same units from the time of occlusion in the test finger, and the mean normalized flow during an experiment in the control finger. It is presumed that this might represent the best estimate of the flow in the test finger had it not been occluded.

Two other possible ways of estimating these flows were rejected. The resting blood flow taken before applying occlusion was not suitable because of normal fluctuations in flow found in a digit; the use of the control flow during occlusion of the test finger had an added objection that the application of the occlusion to the test finger might be a general stimulus affecting the control flow.

The percentage of the debt paid back is calculated as the ratio:

\[
\text{amount of reactive hyperemia} \times 100
\]

\[
\text{amount of debt acquired}
\]

Reactive hyperemia in the finger does occur after release of occlusion. In all experiments except no. 15, an increase in blood flow was noted following an occlusion of 2 min. or more. It was demonstrated once in subject D.P. after 1 min. occlusion but he failed on another occasion.

The absolute amount of reactive hyperemia in a given experiment seemed to increase with the period of occlusion (fig. 2). For example, a 5 min. occlusion always produced a greater amount than a 2 min. occlusion during the same experiment (by statistics of paired data p 0.02) and this was independent of the order in which these were done. The mean values for the amount of reactive hyperemia after 2 and 5 min. occlusions were 22.2 ± 11.5 and 46.4 ± 17.8 ml./100 cc. finger respectively.

The duration of reactive hyperemia, which was arbitrarily determined as the time of cessation of the major portion of the increase
in blood flow, also increased with the period of occlusion, except in 2 experiments. This can be seen from figure 3.

Whether this increase in flow has the function of repaying some debt acquired during the occlusion period is difficult to answer from this study. The skin flow in a finger has a twofold function, nutrition of the skin and temperature regulation of the body. From oxygen consumption studies on skin the minimal flow required for nutrition may be estimated as about 1 ml./100 cc tissue/min. Thus repayment, from our data, would be far beyond the nutritional requirements. The total debt incurred was also calculated in the manner explained earlier and the percentage repaid is tabulated. In a given experimental run the results show only a slight tendency to repay a certain percentage of total debt irrespective of the period of occlusion, since the difference between percentage debt paid back after 2 and 5 min. occlusions (in 10 experiments) was not significant (statistics of paired data \( p = 0.25 \)). The overall mean percentage of debt paid back was 51 per cent, with a standard deviation of \( \pm 36 \), and \( \sigma_M = 6 \) per cent.

It was noted that the percentage debt repaid for 1 of the subjects (G.F.) was always less than for 2 other subjects (D.P. or B.H.), where there were 8 or more comparable experiments. An analysis of variance for these showed that the variance between these 3 subjects was significantly greater (\( p = 0.05 \)) than the variance within subjects. This indicates that the degree of repayment of a blood flow debt by reactive hyperemia tends to be an individual characteristic. Individual differences in the anatomy and innervation of the arterial-venous anastomoses, the opening of which probably plays an important role in the hyperemia, might be responsible.

In 1 other experiment (M.S.), the room temperature was 21 C. and the patient complained of cold. His resting flows were markedly reduced and no reactive hyperemia was observed even after a 5 min. occlusion. This would indicate the inability of the mechanism to open up the constricted vessels. Similar findings are reported by Doupe (3).

A paradoxical feeling of cold was noted by all subjects when warm blood rushed into the finger after release of the occlusion. Whether this was due to an increased temperature gradient for the heat flow, or due to change in sensitivity of the thermal receptors due to oxygen lack or to some other mechanism, is under investigation.

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