FINGER SKIN PERFUSION PRESSURE IN GENERALIZED SCLERODERMA

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Abstract. Skin perfusion pressure in fingers in 6 normal persons and 12 patients suffering from generalized scleroderma was estimated as the minimum external counterpressure required to stop $^{133}$Xenon wash-out. The isotope was introduced atraumatically into the skin, and local counter-pressure was exerted by a miniature blood pressure cuff. Local perfusion blood pressure in normal fingers was $100.5 \text{ mmHg} \pm 3.3 \text{ SEM}$, while in the patients the value was $72.5 \text{ mmHg} \pm 8 \text{ SEM} (p<0.001)$.

Lewis & Landis advocated the idea that, in generalized scleroderma of the acrosclerosis type, structural changes of digital vessels result in vasospastic attacks during exposure to cold (14). Prinzmetal stressed the importance of the tight inelastic skin of the fingers in effecting the reduced capillary blood flow in acrosclerosis, although the results of his study might equally well be explained by a decreased perfusion pressure due to vascular changes (18). Later studies have reached the conclusion that partial vascular obstruction acted upon by a normal vasomotor tone results in Raynaud’s phenomenon (16). Hand or finger blood flow measurements using plethysmography or clearance of radioactive isotopes usually yields results within the normal range in generalized scleroderma (1, 12, 17), whereas functional investigations reveal defects in local blood flow regulation (7, 9, 10). The reaction of hand blood vessels of normals to direct or indirect cold is very similar to that of patients exhibiting Raynaud’s phenomenon (4, 5, 17). However, patients with Raynaud’s phenomenon do not vasodilate in response to central heating, as much as normals do (4) and a markedly delayed and reduced recovery of hand blood flow following exposure to cold has been observed (5, 13). Direct observation of nail fold capillary blood flow during cold provocation has revealed a complete cessation of blood flow in scleroderma patients, whereas the flow never stops completely in normals (15).

MATERIAL AND METHODS

The experiments were done in 6 normal persons aged between 35 and 80 years and in 12 patients suffering from generalized scleroderma of the acrosclerosis type. The patients were between 25 and 73 years of age, and the history of the disease was 3 to 15 years. All patients had Raynaud’s phenomenon triggered by exposure to cold. None of the fingers investigated exhibited ulceration, but in 3 of the patients ulcers were present on adjacent fingers. Arm blood pressure was measured before and after the experiment and mean arm blood pressure calculated as diastolic pressure plus one-third of the pulse pressure. A skin area immediately proximal to the nail fold of the second left finger was labelled with $^{133}$Xenon using the epicutaneous labelling technique (19). The labelled area was covered immediately with a miniature blood pressure cuff, 20 mm in width, connected to a mercury manometer. The pressure in the cuff was brought to 300 mm Hg to stop isotope wash-out. A Nal (Tl) scintillation detector was placed about 10 cm from the radioactive field. The pulses from the detector were fed into a printing γ-spectrometer. The activity was recorded every 10 sec. Count values were plotted on semilogarithmic paper covering one decade. When the decline in the radioactivity of the depot had stopped, pressure in the cuff was lowered in a stepwise manner. For each pressure level, wash-out was followed for 5 min. Wash-out was expected to start near a cuff pressure equalling the mean brachial blood pressure (8). Near this level, the steps were diminished to 10 mmHg. Local perfusion pressure was taken as cuff pressure when wash-out started plus $5 \text{ mmHg}$ (Fig. 1). Skin temperatures measured after the experiments were observed to range from 34 to 36 both in normals and in patients.

Student’s $t$-test for paired of impaired samples was used.
Fig. 1. Recording of local perfusion blood pressure in a finger of a 45-year-old man with Raynaud's phenomenon and scleroderma. The wash-out starts at 80 mmHg (local perfusion blood pressure 85 mmHg). The columns indicate the external counterpressure.

RESULTS

The results are summarized in Table I. It was observed that in normals the finger blood pressure did not differ from the mean arm blood pressure (p > 0.6). In the patients, local perfusion blood pressure in the fingers was significantly decreased in comparison with mean brachial blood pressure (p < 0.001) and also significantly decreased as compared with local perfusion pressure of normal fingers (p < 0.001).

DISCUSSION

The method described has been used previously to measure local perfusion blood pressure in muscle, employing a 133Xenon-histamine depot (3). Local blood pressure values close to the intra-arterial diastolic blood pressure were found. The method proved of limited value when it was desired to measure skin perfusion pressure in legs (8). Due to accumulation of the tracer in subcutaneous fatty tissue, the slope of the wash-out curve became too flat. In a previous study, it was observed that accumulation of 133Xenon in subcutaneous tissue did not occur when labelling the distal part of the finger (9). The method was expected to be suitable for the present purpose, in which it was desirable to use an atraumatic technique. Using 131I-antipyrine mixed with histamine, local skin perfusion pressures 10 mmHg lower than the simultaneously measured intra-arterial mean blood pressure was regularly recorded (8). The blood pressure values in this kind of measurement are related to the cuff width in such a way that a smaller cuff width tends to overestimate the blood pressure (8). This observation could possibly explain why, in the present study, local perfusion blood pressure values in normals were found near the mean brachial blood pressures. On the other hand, this phenomenon would not explain the difference between normals and patients.

A phenomenon, which has previously been described for oedematous cutaneous tissue of legs (8), was observed in the investigations of oedematous fingers of patients. When a pressure of 300 mmHg was applied and wash-out expected to stop, a decrease in radioactivity was still recorded due to the flow of oedema-fluid away from the detector. In such cases, the decrease in radioactivity stopped when pressure had been applied for about 10 min.

The blood pressure values in normal fingers found by the present technique agree with the values obtained during direct observation of capillary flow in the nail fold (6). The results indicate that local perfusion blood pressure in fingers is reduced in generalized scleroderma, a finding which is consistent with the changes in digital arteries found by arteriography (2, 20). This decrease in arterial perfusion pressure head, together with an increase in blood viscosity during cooling, might account for a considerable decrease in blood flow. The longer rewarming period could readily be explained on this basis, and so could a slighter increase in the finger temperature in response to central heating (4, 5). In a previous paper, a very considerable decrease in maximum blood flow in response to an ischemic stimulus was described for scleroderma patients as compared with normals (9). Part of this difference might be explained by an increased resistance to blood flow in digital arteries in generalized scleroderma, although local alterations in the

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<td>BP = Blood pressure, SEM = Standard error of the mean</td>
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<td>Normals</td>
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<td>1. Mean arm BP (mmHg)</td>
<td>98.5 ± 4.0 SEM</td>
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<tr>
<td>2. Local perfusion BP</td>
<td>100.5 ± 5.3 SEM</td>
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<td>3. Mean arm BP (mmHg)</td>
<td>99.8 ± 5.2 SEM</td>
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<td>4. Local perfusion BP</td>
<td>72.5 ± 1.8 SEM*</td>
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* Significantly different from 2 and 3 (p < 0.001).
microcirculation such as an intrinsic vascular smooth muscle defect (10) or a reduced capillary density (11) might also be involved.

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REFERENCES


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