REDUCED SYSTOLIC BLOOD PRESSURE IN FINGERS OF PATIENTS WITH GENERALIZED SCLERODERMA (ACROSCLEROSIS)

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Abstract. Segmental systolic blood pressure was measured on upper arm, wrist, proximal and middle phalanx in 10 patients suffering from generalized scleroderma of the acrosclerosis type and in 5 normals. Systolic pressure was measured by the strain-gauge technique before and after abolition of sympathetic vasoconstrictor activity by local heating and by nerve block. Arm and wrist pressures were equal in the two groups, but a significant reduction in finger pressure was found in the patients. Finger pressure was unaltered after sympathetic blockade, both in the patients and in the normals. The pressure drop from wrist to finger in the patients suggests an increased flow resistance in the palmar arch and digital arteries which was probably not caused by an augmented sympathetic vasoconstrictor activity, as sympathetic blockade did not influence finger systolic pressure. Sympathectomy cannot be expected to relieve Raynaud's attacks in these patients.

Key words: Strain-gauge technique; Raynaud's phenomenon; Sympathetic blockade

Raynaud's phenomenon in the hand is a common finding in patients with generalized scleroderma. Lewis and Landis concluded that the vascular crises in scleroderma are caused by a local vascular defect (11). Prinzmetal suggested that the tight skin constricts the blood vessels (16). Mendlowitz and Naftchi obtained evidence indicating that Raynaud's attacks are produced either by vascular narrowing acted upon by normally reacting vascular smooth muscle cells, or by an increased reactivity to sympathetic neural discharge (12). Angiographic findings have revealed injury to the digital arteries with narrowing and complete obstruction (3, 17, 19). If this is so, it might be expected that local arterial perfusion pressure in the fingers of these patients would be reduced. The finding of a decreased finger pulse volume (3) and a reduced maximum blood flow during reactive hyperemia following circulatory arrest (5) in these patients, suggests that digital arterial pressure is reduced.

This study dealt with the segmental systolic pressure in the upper extremity as measured by the strain-gauge plethysmography technique (13). Measurements were carried out before and during sympathetic blockade in order to evaluate the possible role of sympathetic vasoconstrictor activity upon the finger systolic pressures.

MATERIAL AND METHODS

The study comprises 10 patients with generalized scleroderma of the acrosclerosis type. Informed consent was obtained before each experiment. Ages ranged from 29 to 64 years. Disease duration from 2 to 15 years. All had a history of Raynaud's syndrome. Hand and finger sclerosis was severe in 5, moderate in 3 and slight in 2 patients. Five normal controls aged 20 to 50 years were also investigated.

Experimental procedure and calculations

The subject, normally dressed, was placed in a supine position. Room temperature remained constant at 24°C. Systolic pressure was measured by the strain-gauge technique (13). Cuffs were placed around the upper arm, wrist and around the proximal and middle phalanx of the third finger. The finger cuffs were 2.4 cm wide, as recommended by Krähenbühl et al. (10). The strain gauge was placed on the finger tip, avoiding compression of the finger. Skin temperature on the fingertips ranged from 22° to 28°C.

In order to test the influence of finger blood flow upon finger systolic pressures, abolition of sympathetic vasoconstrictor activity was induced by local heating of the skin of the finger to more than 36°C. Nerve block was induced by infiltrating the base of the finger with lidocaine (1 mg/ml) without vasoconstricting.

Cutaneous blood flow was measured dorsally on the distal phalanx and on the finger tips by the local 133Xe technique (5, 6, 18).

Statistics

Student's t-test for paired samples and the rank-sum test were used to test for significance. A significance level of 0.05 was chosen.
The segmental systolic pressures recorded for the patients and the normal subjects are shown in Fig. 1. Average pressures ±1 S.E. were 120±11, 101±3, 92±11, and 77±9 mmHg on the upper arm, wrist, phalanx I and phalanx II, respectively, in the patients, and 114±4, 125±5, 116±4, and 107±3 in the normals. There was no inter-group difference in the pressures obtained on the upper arm and wrist. By contrast, the pressures measured on phalanx I and II were considerably reduced in the patients (p<0.01). The relative decrease in systolic pressure from the proximal to the middle phalanx was 9 and 17% in the normals and the patients, respectively. This difference was significant (p<0.05).

In Fig. 2 the patients are divided into three groups based on clinical judgement (slightly, moderately and severely affected). Although the material is too small to permit of statistical analysis, there seems to be a tendency to a greater reduction in the finger systolic pressures in the more severely affected patients.

There was no difference between the $^{133}$Xe washout rate constants obtained dorsally on the distal phalanx in the two groups, whereas the wash-out rate of $^{133}$Xe from the pulp of the finger tip was reduced in the patients.

Sympathetic blockade induced by nerve block at the base of the finger and local heating increased blood flow in the finger tips by 700% in the normals, whereas blood flow increased by only 100% in the patients. Dorsally on the finger tip, blood flow increased by about 70% in both groups.

Neither in the normals nor in the patients did sympathetic blockade and local heating alter the relative systolic pressures in the fingers (Fig. 3).

The main finding in the present study is that systolic pressure in the fingers is reduced at normal ambient room temperature in patients with generalized scleroderma with Raynaud’s phenomenon. Furthermore, the relative gradient from the proximal to the middle phalanx was greater in the patients than in the controls.
The decrease in systolic pressures in the patients may be due to differences in finger blood flow between patients with generalized scleroderma and normals. Cutaneous blood flow on the dorsum of the distal phalanx did not vary in the two groups, whereas cutaneous blood flow on the pulps was reduced in the patients. This suggests that finger blood flow in the patients is similar to or less than seen in normals under the present conditions. This indicates that the reduced pressures in the middle phalanx, taken together with the increased pressure drop from the proximal to the middle phalanx, is due to an increased flow resistance in the digital arteries of patients with generalized scleroderma.

Furthermore, the increased pressure drop from the wrist to the proximal phalanx similarly suggests an increased flow resistance in the palmar arch in these patients.

Thus, the evidence presented suggests the presence of obliteratorive changes of the palmar arch and digital arteries of the patients with generalized scleroderma. This is compatible with the finding of thickening of the intima causing a permanent decrease in the lumen of the small arteries (1, 2, 15) and with the angiographic findings of narrowing and complete obstruction of the digital arteries in patients with generalized scleroderma (3, 17, 19). The reduced pressure in the digital arteries of the patients explains at least partly the finding of a diminished maximum blood flow in cutaneous and subcutaneous tissues of the fingers during reactive hyperemia following circulatory arrest in patients with generalized scleroderma (6).

Neither in the patients in the controls did sympathetic blockade and local heating influence the relative systolic pressures of the fingers. This indicates that sympathetic vasoconstrictor tone under the present conditions is without importance for the reduced systolic pressure observed in the patients’ fingers. This is compatible with the observation that the normal reaction of the vascular smooth muscle cells to an increase in discharge rate in sympathetic vasoconstrictor fibres is abolished in the fingers of patients with generalized scleroderma (7). Thus, Raynaud’s phenomenon observed in these patients is probably not a result of augmented sympathetic vasoconstrictor activity, but rather of the fact that in partly stenosed digital arteries even a small increase in the vasoconstrictor activity of the vascular smooth muscle cells induced by cold will create significant lumen changes.
in analogy with what is seen in primary hypertension (4).

This indicates that sympathectomy is not suitable for relieving Raynaud's attacks in such patients.

REFERENCES


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