SHORT COMMUNICATION

Effect of locally applied subatmospheric pressure on subcutaneous blood flow during the course of acute myocardial infarction

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Summary

1. The effect of locally applied subatmospheric pressure on subcutaneous blood flow was studied in six patients with acute myocardial infarction.
2. Blood flow was measured by the local $^{133}$Xe washout technique.
3. Application of subatmospheric pressures of $-80$ and $-150$ mmHg to the labelled area induced no vasoconstriction on day 1. On day 7 pressures of $-40$, $-80$ and $-150$ mmHg induced a decrease in blood flow of about $40\text{--}50\%$.
4. The absence of a vasoconstrictor response to locally applied subatmospheric pressure on day 1 could not be due to decreased venous distensibility. The underlying mechanism may be neuronal inhibition, probably antidromic, in sympathetic fibres.

Key words: Acute myocardial infarction, local suction, skin, subatmospheric pressure, subcutaneous blood flow, veno-arteriolar reflex.

Introduction

Local regulation of subcutaneous blood flow during the course of acute myocardial infarction has been studied previously [1]. It was found that the local sympathetic veno-arteriolar reflex mechanism responsible for the vasoconstrictor response to increase in venous transmural pressure was abolished during day 1 of myocardial infarction but reappeared on the following days, and was normal by day 7.

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The absence of the local veno-arteriolar reflex on day 1 cannot be explained by a reduced reactivity of the arteriolar smooth muscle cells to sympathetic impulses, since centrally elicited increase in sympathetic activity induced by head-up tilt caused vasoconstriction in subcutaneous tissue even on day 1 [2].

The arteriolar constriction underlying the local venoarteriolar reflex is elicited by venous distension induced by an increase in venous transmural pressure of 25 mmHg or more [3]. Therefore the abolition of the local reflex on day 1 could be due to reduced venous distensibility caused by neurogenically induced vasoconstriction [4]. In this case it would be expected that an increase in venous transmural pressure of 150 mmHg, which is six times the threshold pressure for the reflex, would elicit the reflex even on day 1.

The purpose of the present study was to investigate whether locally applied subatmospheric pressure on the skin surface was able to induce vasoconstriction in subcutaneous tissue during the course of acute myocardial infarction. This might further elucidate the mechanisms underlying the observed absence of the local veno-arteriolar reflex during day 1 of myocardial infarction.

Material and methods

Patients

Five males (mean age 54 years, range 42--60 years) and one female (65 years old) with electrocardiographic signs of acute transmural myocardial infarction (five anterior and one
inferior) were examined as soon as possible after admission to the Coronary Care Unit. On arrival duration of chest pain was less than 6 h in all patients. None of the patients received antiarrhythmic drugs before admission or during the investigation period and pethidine was not used within 4 h before the investigation. None showed clinical signs of congestive heart failure or increased venous pressure in the investigation period. All patients were studied on days 1 and 7. Before the investigation informed consent was obtained.

Experimental procedure

Subcutaneous blood flow was measured by the local $^{133}$Xe washout technique as previously described [1] and expressed in the same units. A single investigation consisted of five to seven measurements each lasting 6-10 min. The patient was in a supine position, and the labelled area was 5 cm proximal to the wrist at heart level. Subatmospheric pressures of $-40$, $-80$ and $-150$ mmHg were applied to the labelled area with a suction glass (40 mm in diameter) connected to a mercury manometer.

Before and after suction, reference measurements were performed and the relative blood flow and vascular resistance calculated.

On day 1 only suctions at $-80$ and $-150$ mmHg were used.

In all patients arterial blood pressure (arm cuff) and heart rate were measured several times during the investigation; as they were fairly constant throughout the investigation periods only mean values ± SEM are given.

Statistics

Student’s $t$-test for paired samples was used to assess significance ($P < 0.05$ significant).

Results

Mean arterial blood pressure and heart rate

Mean arterial blood pressure was $98 ± SEM 3$ and $97 ± 4$ mmHg and mean heart rate was $73 ± 5$ and $78 ± 5$ min$^{-1}$ on days 1 and 7 respectively.

Locally applied subatmospheric pressure

During day 1 locally applied pressures of $-80$ and $-150$ mmHg revealed no vasoconstrictor response in subcutaneous tissue (Fig. 1), as blood flow increased and decreased by 3% ($P > 0.5$) respectively, and the corresponding relative vascular resistance decreased by 2% ($P > 0.6$) and increased by 4% ($P > 0.6$) respectively.

On day 7 application of $-80$ mmHg pressure induced a decrease in blood flow of 52% ($P < 0.001$), corresponding to an increase in vascular resistance of 116% ($P < 0.001$). Application of $-150$ mmHg pressure decreased subcutaneous blood flow by 34% ($P < 0.005$), corresponding to an increase in vascular resistance of 67% ($P < 0.05$). A pressure of $-40$ mmHg induced vasoconstriction as blood flow decreased by 42% ($P < 0.001$) and vascular resistance increased by 78% ($P < 0.005$).

Discussion

It has been reputed that about 90% of the locally applied subatmospheric pressure is transmitted to the underlying tissues up to $2.5$ cm deep [5].
Therefore it is reasonable to assume that the locally applied pressure of -150 mmHg increased venous transmural pressure to five to six times the pressure for eliciting the local veno-arteriolar reflex.

The present study indicates that this manoeuvre has no effect upon subcutaneous blood flow on day 1 of myocardial infarction. The absence of the local reflex on day 1 cannot be explained by an inability of the arterioles to constrict against the applied increase in transmural pressure as further vasoconstriction occurred during head-up tilt even on day 1 [2].

Wood et al. [4] found a decreased venous distensibility of about 30–40% in patients with congestive heart failure. As the patients in the present study were without signs of congestive heart failure, the decrease in distensibility was probably less. This, taken together with the fact that applications of 150 mmHg subatmospheric pressure to the skin did not induce vasoconstriction, rules out the possibility that decreased venous distensibility could abolish the local reflex on day 1.

The underlying mechanism is probably therefore a neuronal inhibition of the impulses in the sympathetic fibres, probably an antidromic impulse inhibition.

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References