RESPONSES OF SUPERFICIAL AND DEEP BLOOD VESSELS TO COOLING

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SUMMARY

Dilation of blood vessels seems desirable from a therapeutic point of view. Results suggest that application of icepacks may not be the ideal technique to use, as icepacks, although lowering skin temperature to levels at which skin vessels will dilate, will not lower muscle temperatures to levels at which deep vessel constriction is inhibited. Results further show that to achieve dilation of vessels, it is not necessary to apply icepacks for longer than 8 - 10 minutes.

INTRODUCTION

One of the basic assumptions of rehabilitation of musculoskeletal injuries is that blood flow to the injured area must be increased (Knight and Londeree, 1980). Increases in blood flow can only be achieved if the diameter of the blood vessel increases and perfusion pressure remains constant.

Various techniques are used to cause vasodilatation without affecting blood pressure. Most of these techniques are based on the observation that contractility of vascular smooth muscle can be affected by a number of physical factors such as local temperature, light intensity and sonic vibration (Vanhoutte and Verbeuren, 1981). Of these, temperature is known to have marked effects. Thus applications of heat (Abramson *et al.* 1958) and cold (Allwood and Burry, 1954; Laing *et al.* 1973) have been used to induce vasodilatation. In both cases it is assumed that temperature affects the interaction between catecholamines and the vascular smooth muscle (Vanhoutte and Verbeuren, 1981). However, the precise effects of changes in temperature are unclear.

The technique most widely used to induce cooling is application of icepacks. If icepacks are to be successful in increasing blood flow to injured tissue they must lower superficial and deep temperature to an extent that will cause blood vessels to dilate. We have examined, therefore, the effect of icepack application on skin temperature and whether the temperatures produced by icepacks will affect blood-vessel diameter.

METHOD

To determine the effect of icepacks on skin temperature, an icepack made by wrapping crushed ice in a wet towel was applied to the skin of 8 healthy human volunteers (5 females, 3 males). In all cases the icepack was applied to the anterior surface of the left thigh.

Before application the surface temperature was measured using a calibrated alcohol thermometer. The bulb of the thermometer was placed approximately in the centre of the quadriceps muscle mass. After obtaining a steady value for skin temperature, the icepack was applied and skin temperature was recorded at 8, 15 and 20 minutes after

OPSOMMING

Dilatasie van bloedvate blyk wenslik te wees van 'n terapeutiese standpunt. Resultate dui daarop dat die aanwending van yspakke nie die ideale tegniek is nie aangesien yspakke, wat wel die veltemperatuur verlaag tot vlakke waar die huidvate sal dilateer, nie spiertemperatuur verlaag tot vlakke waar diepvaatsametrekking inhibeer word nie. Resultate dui ook aan dat, om dilatasie van vate te bewerkstellig, dit nie nodig is om yspakke langer as 8 - 10 minute aan te wend nie.

application of the icepack. After 20 minutes the icepack was removed. Skin temperature was then recorded at 2 minute intervals for 10 minutes and at 5 minute intervals for the next 10 minutes.

To assess the effects of cooling on blood vessels, abdominal aortas of rabbits were cooled in vitro. In these experiments 9 rabbits were given an overdose of thiopentone sodium (Intraval, Maybaker). A segment of the aorta was removed, attached to a 14 gauge needle, suspended in an electrolyte solution (Plasmalyte B, Baxter) at 37°C and aerated using a 95% O_2, 5% CO_2 gas mixture (Fig. 1).
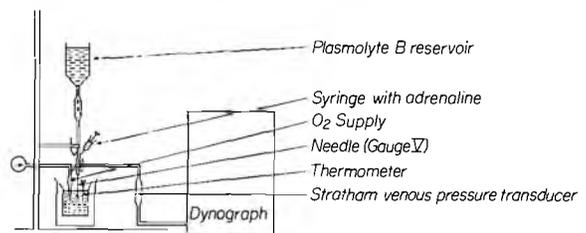
Figure 1.

The first series of experiments was designed to assess the effect of a fall in temperature from 37°C to 0°C on the resistance to fluid flow through vessels. In these experiments the electrolyte solution bathing the vessel was gradually cooled over a period of 30 minutes. The perfusing fluid was also cooled. Resistance to fluid flow was calculated from the formula $R = {}^{DP}/_F$, where R = resistance, DP = perfusion pressure and F = fluid flow.

Perfusion pressure was measured by attaching a Statham P23 AA venous pressure transducer proximal to the artery. Pressure was recorded on a Beckman Dynograph calibrated to measure pressures between 1 and 40 mmHg. Perfusion pressure was kept constant by maintaining the height of the reservoir containing the electrolyte solution at a constant distance above the artery. Flow (ml/min) at each temperature was determined by measuring the time taken for 20 ml of Plasmalyte B to pass through the vessel. In these experiments the effect of cooling was assessed from changes in resistance. Basal resistance for each vessel was considered to be the resistance calculated at 37°C. Changes in the resistance of each vessel to flow were calculated by subtracting the resistance measured at temperatures below

37°C from basal resistance. The values obtained for change in resistance for all 9 arteries in the temperature ranges 36 - 30°C, 29 - 20°C, 19 - 10°C, 9 -5°C and 4 - 0°C were pooled, and a value for the mean change in resistance was obtained.

The second series of experiments on isolated vessels was designed to separate any direct effect of cooling on blood vessels from effects of cooling on adrenergic neuroeffector interaction. In these experiments 0,25 μg of adrenaline was injected into the perfusion fluid immediately proximal to each of the 9 arteries at temperatures of 37°C, 30°C, 20°C, 10°C and 0°C. The change in resistance induced by injections of adrenaline were calculated as described above. A mean value for change in resistance at each temperature was obtained from changes in each of the arteries at each temperature.

RESULTS
The effects of icepacks on skin temperatures

Figure 2 shows the mean skin temperature of 8 subjects during and after icepack application. Basal skin temperature was 29,5 ± 0,4°C ($\bar{x}$ ± S.E.M.) Application of an icepack reduced skin temperature significantly to 7,8 ± 1,0°C within 8 minutes (P < 0,01, 't' test). Although the icepack was applied for a further 12 minutes no further significant decrease in skin temperature occurred. These results indicate that icepacks reduce skin temperature to a minimum of approximately 7°C in about 10 minutes.

After removal of the icepack, skin temperature increased to 20°C within 8 minutes. Thereafter skin temperature increased relatively slowly. Extrapolation of the warming curve suggests that skin temperature would reach starting values (29,5 ± 0,4°C) approximately 100 minutes after removal of the icepack.

Icepacks and skin temperature

Mean temp.	29·5	7·8	5·9	7·0	15·3	19·4	22·5	22·8
SEM	0·4	1·0	0·5	1·2	1·0	1·0	1·0	1·2
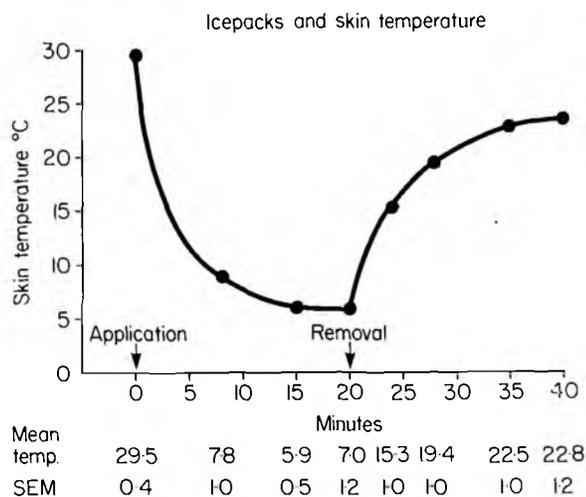
Figure 2.

The effect of temperature on blood vessels

Figure 3 shows that compared to resistance at 37°C (R = 0) a progressive decrease in mean resistance of 0,5; 2,4; 3,2; 2,8; and 3,9 mmHg/ml. min^{-1} occurred in the temperature ranges 37 - 30°C, 29 - 20°C, 19 - 10°C, 9 - 5°C, and 4 - 0°C. These changes represented a highly significant linear correlation between temperature and resistance (r = 0,90; P < 0,05).

Effect of cooling

$$y=(0.09 \pm 0.03)x-(4.0 \pm 0.3)$$
$$r^2=0.81$$
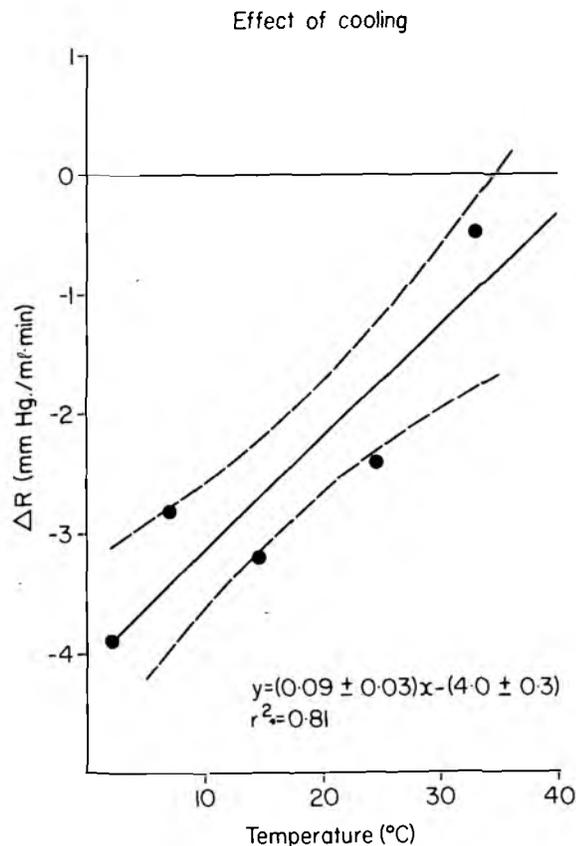
Figure 3.

Most of the change in resistance (2,9 mmHg/ml.min^{-1}; 75%) occurred in the temperature range 37 - 20°C).

Figure 4 shows that at temperatures above 20°C injections of adrenaline are likely to cause a vasoconstriction; resistance increased in all arteries at temperatures of 30°C and 37°C. However, injection of adrenaline into arteries at 0°C and 10°C did not produce an increase in resistance compared to resistance at 37°C. Figure 3 shows however a highly significant linear correlation between decreasing temperature and resistance in the presence of adrenaline (r = 0,98, P < 0,05) which suggests that cooling decreases vessel sensitivity to adrenaline.

Cooling of vessels has both direct and indirect effects on vessels. This is shown in Figure 5 which was obtained by plotting Figures 3 and 4 on the same axes. In this figure it is clear at all temperatures above 13,5°C injections of adrenaline are able to cause a significant increase in resistance despite the decrease in resistance caused by cooling. Thus vascular smooth muscle still responds to injections of adrenaline although its ability to contract is reduced at low temperature.

DISCUSSION

Several physiological mechanisms regulate blood vessel diameter. Stimulation of ß-adrenergic receptors in vascular smooth muscle or inhibition of α-adrenergic receptors will cause relaxation of smooth muscle and blood vessel diameter will increase. Dilatation will also occur if vasoactive

Effect of adrenaline

Figure 4.

Effect of adrenaline and cooling
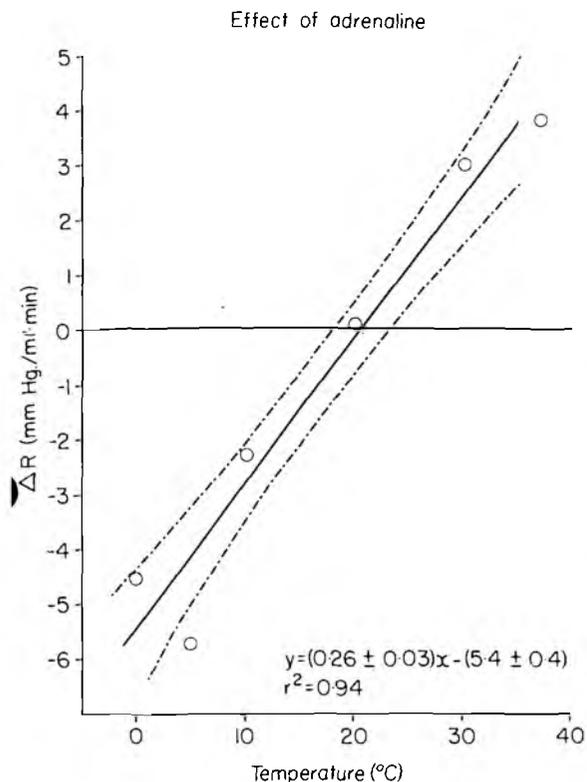
Figure 5.

metabolites are released into the immediate environment of smooth muscle cells. Our results confirm previous findings (Allwood and Burry, 1954; Laing et al., 1973) that cooling blood vessels will also induce vasodilatation although the mechanism is not clear.

Our results, however, suggest that vasodilatation is partly because of direct effects of low temperature on blood vessels. The evidence which supports this idea is that in the absence of nerves and neuroeffector substances resistance to blood flow decreases linearly with decreases in temperature (Fig. 3). The increase in diameter associated with the fall in resistance could be explained by a reduction in the elasticity of the vessel. It has been shown previously (Scandola and Pezzin, 1978) that elastin relaxes when it is cooled and contracts when warmed. Another explanation is that smooth muscle metabolism is decreased at low temperatures. However this explanation seems unlikely if the vessel is cooled moderately. Our results show that at 30°C vascular smooth muscle contracts normally in the presence of adrenaline. More severe cooling to temperatures of 15 - 20°C also does not abolish vascular smooth muscle response to adrenaline, although the response is diminished. However, below 13,5°C smooth muscle does not contract in the presence of adrenaline which suggests that muscle metabolism could be inhibited at low temperatures.

Our results also indicate that cooling may indirectly cause vasodilatation. In vivo the indirect vasodilator effect of cooling on vascular smooth muscle could result from changes in nerve function. Synthesis and storage of transmitter are depressed by cooling and cooling decreases spontaneous efflux of transmitter substances (Vanhoutte,
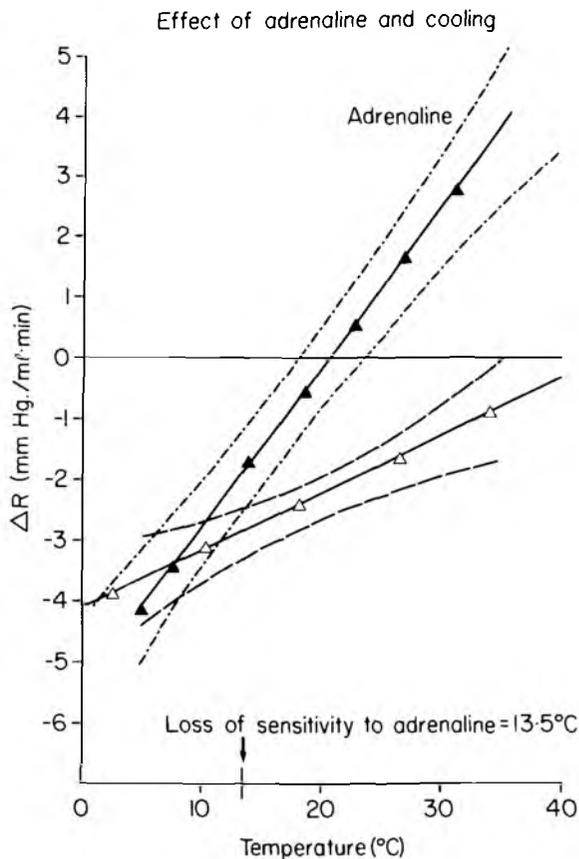
Verbeuren and Webb, 1981) all of which will reduce vasoconstrictor tone and allow the vessel to dilate. In addition, as the vessels showed a decreasing response to adrenaline, our results suggest that the sensitivity of α-adrenergic receptors may be reduced by cooling (Fig. 4). Furthermore, adding adrenaline does not cause the vessel resistance to fall to levels below that found by cooling alone, which suggests that there is not an enhanced ß-response at low temperatures.

All of these results indicate that, whatever the underlying mechanism, cooling to 13,5°C or below, causes blood vessels to dilate. Application of icepacks cools superficial vessels to 7°C within 10 minutes. Thus icepacks should cause skin vessels to dilate after an initial period of constriction (Vanhoutte, Verbeuren and Webb, 1981). Further, since most of the decrease in resistance which follows cooling occurs in the temperature range 37 - 20°C (Fig. 2) it is possible that deep vessels will dilate during icepack therapy. Calf muscle temperature is reduced to 30 - 35°C during icepack application and forearm muscle falls to about 30°C when the forearm is immersed at 12°C (Barcroft and Edholm, 1943). However, since our results suggest that deep vessels are sensitive to adrenaline at all temperatures above 13,5°C, stimulation of sympathetic nerve activity which will follow application of icepacks might cause constriction and reduce blood flow to injured muscles.

In summary dilation of blood vessels seems desirable from a therapeutic point of view. However, our results suggest

that application of icepacks may not be the ideal technique to use as icepacks, although lowering skin temperature to levels at which skin vessels will dilate, will not lower muscle temperatures to levels at which deep vessel constriction is inhibited. Our results further suggest that to achieve dilatation of vessels it is not necessary to apply icepacks for longer than 8 -10 minutes.

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THE MEASUREMENT OF PAIN - A BRIEF REVIEW

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Summary

Physiotherapists are now measuring pain when they assess their patients. Pain has been considered to be unmeasurable by some, but a number of subjective and objective methods have been devised. Subjective methods appear to be more satisfactory than objective methods. Several methods of subjective measurement are reviewed. Studies suggest that the Numerical Rating Scale (N.R.S.) may be an appropriate subjective scale for general use. Several methods of measuring pain relief are also reviewed. Patients tend to express themselves more in terms of pain relief than in terms of pain measurement. The principles of the Signal Detection Theory for quantification of pain are outlined.

Opsomming

Fiosioterapeute meet nou pyn wanneer hulle hul pasiënte evalueer. Pyn is deur sommige as onmeetbaar beskou, maar 'n aantal subjektiewe en objektiewe metodes is ontwerp. Subjektiewe metodes blyk meer bevredigend te wees as objektiewe metodes. Verskeie metodes van subjektiewe meting word hersien. Studies stel voor dat die Numeriese Waardebepalingskaal (Numerical Rating Scale, N.R.S.) 'n geskikte subjektiewe skaal vir algemene gebruik mag wees. Verskeie metodes om pynverligting te meet word ook hersien. Pasiënte is geneig om hulself eerder in terme van pynverligting as pynmaat uit te druk. Die beginsels van die Sein Vasstellingsteorie (Signal Detection Theory) van pyn kwantifisering word geskets.

INTRODUCTION

One of the most important developments in the physiotherapy profession during the last few years has been the increasing emphasis on good assessment prior to physiotherapeutic intervention.

Measurement is the essence of scientific method and during their assessment of patients, physiotherapists routinely measure such things as muscle strength and joint motion, but until recent times it has not been usual for them to measure the major accompaniment of so many of the conditions they treat, namely pain.

According to Huskisson (1974) "pain cannot be said to have been relieved unless pain or pain relief has been directly measured". Thus the question of the feasibility of pain measurement must be raised. Pain is an abstraction and therefore has been considered by many to be unmeasurable.

However, psychologists have been confronted by similar problems relating to the measurement of personality, depression and sleep, and they have coped with this since the early part of the century.

When measurement of pain is being considered, a distinction must be made between experimental and clinical pain. Measurement of pain in the laboratory is relatively easy. Specific and graded stimuli can be used and the responses analysed. It is thus possible to obtain a large degree of sameness in experiments and relatively reproducible results can be obtained (Rosen, 1977). However, with clinical pain the nature of the stimulus may not be obvious and in some cases pain levels give no indication of the severity of the disease. In addition pain may be modified by many behavioural factors (Huskisson, 1974).

The problem of pain measurement has been approached basically in two ways. The most used is the "subjective" method in which the patient is asked to report his pain experience directly. Alternatively an "objective" or indirect measure (visible or non-verbal) can be used if there is a relationship between the measure and relief of pain (Rosen, 1977).

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