Peripheral vasoconstriction after open-heart surgery

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Most patients at the conclusion of open-heart surgery show some degree of peripheral vasoconstriction which persists for a variable period of time until vasodilatation occurs and normal skin perfusion is restored. Ross, Brock, and Aynsley-Green (1969), measuring the temperature of the great toe, showed that peripheral vasoconstriction after open-heart surgery could be prolonged by hypovolaemia. The recognition of abnormal vasoconstriction, however, must ultimately rest on a definition of the degree of vasoconstriction that is normal after open-heart surgery. No such definition exists in the literature.

In the hope that a knowledge of the normal behaviour of the peripheral circulation might permit the early detection of postoperative circulatory impairment a study was undertaken to determine what peripheral temperature changes occur routinely in patients after open-heart surgery. This paper presents the results.

MATERIALS AND METHODS

The postoperative toe temperature pattern was recorded on 148 patients following cardiopulmonary bypass (including emergencies) between 1 January 1971 and 31 December 1972. Operations were performed under ischaemic arrest using a plastic bubble oxygenator and Hartmann’s solution prime, with nitrous oxide, oxygen, halothane, and relaxant anaesthesia. Patients were not actively cooled during operation but the mean lowest oesophageal temperature was 33.1±0.9°C. The mean operating room temperature was 20.8±1.1°C. Patients were warmed to above 34°C (oesophageal) before discontinuing bypass.

Postoperatively patients were nursed in an intensive care unit, flat in bed, with the trunk and lower limbs covered with blankets. Pulse, blood pressure, central venous pressure, electrocardiogram, and hourly urine output were recorded in all patients. Blood gases, acid-base state, and serum electrolytes were estimated four-hourly during the warm-up period and more often if necessary. Mean temperature in the intensive care unit was 23.6±0.9°C. Skin and rectal temperatures were measured at the time of return to the intensive care unit and thereafter at half-hourly or hourly intervals using an electrical resistance thermometer (Tempkey, Gallenkamp, London). Skin temperatures were recorded from the plantar surface of the great toe. Pedal pulses were present in all cases. All readings were taken to the nearest 0.5°C and are reported in degrees Centigrade.

Group values are expressed as mean ± standard deviation and were analysed by Student’s t test with the application of a 5% level of significance. Chauvenet’s criterion ((x̄−8)/Sx, Documenta Geigy Scientific Tables, 1956) was used to identify values lying outside the range of a normal distribution.

In 60 of the 148 cases the warm-up curve may have been modified by drugs such as isoprenaline, lignocaine, etc. (48 cases), or by an episode of peripheral
cooling after the warm-up had begun but before it was complete (12 cases). These subjects were considered unsuitable for determination of the normal or 'basal' degree of postoperative vasoconstriction.

The remaining 88 patients all had an uninterrupted peripheral warm-up curve and required no specific circulatory support. Their results are presented here. Thirty-six were male and 52 female. Ages ranged from 16 to 63 (42±13) years. Twenty-five had aortic valve replacement, 36 had mitral valve replacement, 12 had correction of congenital heart defects, and 15 had miscellaneous operations—double valve procedures (8), open mitral valvotomy (3), pericardectomy (2), saphenous vein aortocoronary bypass graft (1), and removal of foreign body from heart (1). After operation 65 patients were breathing spontaneously and 23 were on intermittent positive-pressure ventilation (IPPV).

RESULTS

CIRCULATORY STATUS Of the 88 patients only 10 had a systolic blood pressure below 100 mmHg at any time during the warm-up period; seven of these 10 had only one (hourly) reading below 100 mmHg and three had two readings below 100 mmHg, but all otherwise maintained their pressure above this level. Of the 88 patients eight required a diuretic during the warm-up period. Five patients (6%) died while still in hospital.

TYPICAL WARM-UP PATTERN To explain the following analysis a typical temperature record is shown (Fig. 1). On return to the intensive care unit after mitral valve replacement the patient was breathing spontaneously. The toe temperature was initially 26-5° (rectal 37°) and remained within 0-5° of this for 3-5 hours. It then rose within two hours to 34° and eventually reached a peak at 36-5° (rectal 38-5°) after which it remained constant within 1-5°.

The curve is seen therefore to consist of three distinct phases which together constitute the warm-up pattern. An initial cold period has been followed by a relatively rapid rise of temperature across an intermediate zone to a final temperature 2-3° lower than the core. Further, the warm-up once begun has proceeded without interruption.

THE NORMAL WARM-UP PATTERN The normal degree of postoperative peripheral vasoconstriction was determined by analysing these three phases of the warm-up curve separately.

(a) The cold period The toe temperature on return to the intensive care unit was 25-9±1-6° (rectal 36-3±0-9°, toe/core gradient 10-4±1-5°). The temperature lay between 23° and 28° in 85 of the 88 patients, the three exceptions all being warmer—28-5°, 29°, and 33-5°. There was no significant difference between patients with or without IPPV. The normal limit of toe temperature during this period was therefore taken to be 23°.

(b) The warm plateau The temperature at which the warm-up curve levelled off was calculated for each patient over six hours. This temperature for the group was 35-9±0-9° (rectal 38-4±0-5°, toe/core gradient 2-4±0-9°) and in all cases lay between 34° and 38°. There was no significant difference between patients with or without IPPV. The normal lower limit of peripheral warmth was taken to be 34°.

Using this value the warm-up time (from arrival in the intensive care unit to 34°) was determined for each patient. There was a significant difference between patients with or without IPPV. The 65 patients without IPPV had a warm-up time of 4-6±2-5 hours, while the 23 patients with IPPV had a warm-up time of 5-8±2-6 hours (Fig. 2).

Of the 65 patients without IPPV, 60 warmed within 6-5 hours; the remaining five warmed at 8-25, 10-5, 13, 13-25 and 13-5 hours, all of which values exceed Chauvenet's criterion for a normal distribution. Retrospective analysis revealed a probable pathological cause in four of these. The limit of normal warm-up for patients without IPPV was therefore taken as 6-5 hours.

Of the 23 patients with IPPV, 21 warmed within eight hours; times in the other two cases were 9-5 and 13-25 hours. The latter value lies outside the normal distribution by Chauvenet's criterion, and in the former case the central venous pressure recordings were misleading due to an incorrectly placed catheter, and the patient was probably
hypovolaemic. For clinical purposes, therefore, eight hours was taken as the limit of normal warm-up for patients with IPPV.

(c) Intermediate zone The pattern for each patient during this phase was represented on a time/temperature graph by a line joining the point at which the curve left the cold zone (28°) with the point at which it entered the warm zone (34°). For any specific group the normal limit of warm-up during this phase will be given by the slope from the individual taking the longest time to warm in that group. For the 21 normal patients with IPPV this slope was from 28° at 6.75 hours to 34° at 8 hours (X–Y in Fig. 3). For the 60 normal patients without IPPV it was from 28° at 5.25 hours to 34° at 6.5 hours.

FIG. 2. Warm-up times (from arrival in the intensive care unit to a toe temperature of 34°) of 23 patients with and 65 patients without IPPV after open-heart surgery.

FIG. 3. Peripheral warm-up slopes (28–34°) of 23 patients on IPPV after open-heart surgery.

VARIABLES No significant difference in warm-up pattern was found between patients having different operations provided they were in the same category with respect to IPPV. The warm-up pattern was also independent of many other variables. No significant difference was found in respect of age (< > 40 years), sex, pre-operative systolic pulmonary artery pressure (< > 40 mmHg), heart size (C–T ratio < > 1 : 1), bypass time (< > 60 min), postoperative central venous pressure (< > 10 cm H₂O), heart rhythm (sinus v atrial fibrillation) or lactic dehydrogenase levels (< > 700 international units/l).

A CLINICAL NOMOGRAM

By putting together the separate limits for each phase of the warm-up curve a nomogram was prepared which indicates time limits for the normal peripheral circulatory recovery from open-heart surgery (Fig. 4). Line A represents the lower limit for toe temperature of 23°, and line D represents the normal lower limit of peripheral warmth of 34°. Slopes C and B represent the limits for the rise in temperature for patients with or without IPPV respectively.

FIG. 4. Nomogram showing the normal limits for peripheral vasoconstriction after open-heart surgery in this unit. Lines ACD and ABD apply to patients with and without IPPV respectively.

The nomogram is independent of the operation performed. In clinical use any patient whose toe temperature at any given time lies to the left of or above the line appropriate to his ventilatory state is recovering normally. Conversely, if his temperature curve crosses these limits he is abnormally constricted—for whatever reason.

DISCUSSION

Cold extremities are so common during critical illness that they are virtually ignored, and the precise quantitation of toe temperature for clinical
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Ibsen (1967) has reported an extensive experience with this measurement as a guide to fluid replacement during the treatment of shock with vasodilators, and Ross et al. (1969) demonstrated its value in detecting hypovolaemia after cardiac surgery. Joly and Weil (1969) found a significant correlation between toe temperature and cardiac output in shocked patients and found that persistent peripheral coldness frequently predicted a fatal outcome.

The factors that govern the temperature of the body surface have been extensively investigated in the laboratory. The temperature at any given site depends on (1) the ambient temperature to which the part is exposed, (2) the quantity of heat that is being delivered to the area, which depends jointly on blood flow and blood temperature, and (3) whether any heat is being generated locally in the area, for example by muscular activity or metabolic organs such as the liver.

Digital temperature measurement is clinically valuable simply because ambient temperature can be easily controlled, and the amount of metabolic heat generated locally in the digits is insignificant and can be ignored. Under experimental conditions the digital blood flow per unit time can be quantitated solely from the digital temperature (Felder, Russ, Montgomery, and Horwitz, 1954), but for clinical purposes it is sufficient that low digital temperatures indicate peripheral vasoconstriction and high temperatures indicate peripheral vasodilatation. The toe is preferred to the thumb for clinical use as it is more convenient for the patient and registers the state of the circulation with maximum sensitivity (Ibsen, 1967; Joly and Weil, 1969). In the postoperative situation, where the patient is already confined to bed, control of ambient temperature at the recording site is achieved simply by keeping the limb covered with blankets.

The principal finding to emerge from this study is that the peripheral warm-up after open-heart surgery is not a random event. Eighty-one of the 88 patients warmed in a remarkably similar manner despite all the variables that theoretically might have affected the pattern. Moreover this pattern was sufficiently constant for meaningful time limits to be deduced for the normal restoration of the peripheral circulation after operation. Patients reached a toe temperature of 34° within 6-5 hours if they were breathing spontaneously and within 8 hours if they were on IPPV, the limits at lower temperatures being defined by the nomogram. The reason for the difference between patients with or without IPPV is not clear; it may be due to the actual use of the ventilator or to the fact that the patients were ill enough to require one.

It is to be stressed that the precise limits of warm-up defined here may apply specifically only to adults operated on and cared for in this unit. Its validity in other units where different techniques may be employed during or after operation remains to be investigated. What may be anticipated, however, is that in any cardiac surgical unit with a well-established routine there will be an equally constant warm-up pattern in patients with a normal circulation, whether the details correspond exactly with those given here or not.

It is recognized that toe temperature is affected by body core temperature and some workers regard the peripheral circulation as normal only when the toe/core gradient is less than 2° (Ross et al., 1969). In this series the gradient was 24±0-9°, but for practical purposes we are satisfied with the state of the peripheral circulation if the toe temperature is above 34°. This single limit can be applied broadly to all cases and eliminates the need for rectal temperature monitoring which may be a potential source of infection for the patient.

The cause of the peripheral vasoconstriction that follows major surgery is not definitely known. Possible mechanisms are a low cardiac output, excessive sympathetic nervous system activity, including release of catecholamines, heat loss during operation or an otherwise unrecognized fluid deficit. Any theory must explain why all patients are cold after open-heart surgery and why similar patterns occur after non-cardiac operation, for example, Fig. 5. On this basis a low cardiac out-

![Fig. 5. Toe temperature curve of a patient aged 71 on IPPV after emergency oesophagogastrectomy for bleeding gastric ulcer, plotted against the appropriate cardiac nomogram.](http://thorax.bmj.com/)

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put is unlikely to be responsible, particularly in fit young subjects after closure of simple septal defects and in non-cardiac cases. Circulating catecholamines are not the cause (Turton and Matthews, 1974), but this does not exclude the possibility of increased vasoconstrictor tone in sympathetic nerves. The suggestion that an occult fluid deficit is the cause is not supported by our finding that the normal warm-up pattern is unaffected by the postoperative level of central venous pressure.

Loss of total body heat has been shown by various workers (Pembrey and Shipway, 1918; Dyde and Lunn, 1969; Lunn, 1969) to occur during many forms of surgery, and postoperative vasoconstriction could be explained on the basis that it protects against further heat loss until muscle and metabolic activity have made good the debt. If all patients developed an approximately similar debt then all would require an approximately similar time to rewarm (as seen here) provided the circulation was adequate. If there was any degree of circulatory impairment then delay in warming would be expected as the necessary increase in metabolism could not be sustained. The proof of this explanation will rest on the demonstration that measures to conserve heat during operation significantly shorten the duration of postoperative vasoconstriction, and studies to investigate this are currently being conducted in our unit.

Whatever the cause of postoperative vasoconstriction, however, the recognition of a consistent warm-up pattern has considerable practical value as it opens up possibilities for the early detection and treatment of circulatory insufficiency. The application of the nomogram to clinical care is described separately (Matthews, Meade, and Evans, 1974).

REFERENCES


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*Thorax* 1974 29: 338-342
doi: 10.1136/thx.29.3.338

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