OXYGEN CONSUMPTION DURING CARDIOPULMONARY BYPASS WITH MODERATE HYPOTHERMIA IN MAN

BY

E. A. HARRIS, EVE R. SEELYE AND A. W. SQUIRE

SUMMARY

Oxygen consumption was measured during hypothermic total body perfusion in twenty-seven patients undergoing cardiac operations. In nine patients it was also measured immediately before induction of anaesthesia. Fourteen patients were kept at normal temperature for from 5 to 17 minutes after bypass began; the rest were cooled immediately to 30°C. Oxygen consumption during bypass at normothermia, whether before or after cooling, was 84 per cent of the pre-anaesthesia value, due possibly to the effects of muscle relaxation. During hypothermia, patients who were cooled immediately had an oxygen consumption which was about 15 per cent lower than that found in the patients with delayed cooling. Even in the latter group, oxygen consumption during hypothermia probably did not quite meet requirements, since it was shown to be positively correlated with blood flow.

The adequacy of perfusion in all regions of the body during extracorporeal circulation and hypothermia is variously judged by arteriovenous oxygen-content difference, by mixed venous oxygen pressure and by the concentration of lactate, "excess lactate" or lactate/pyruvate ratio in the blood. The reliability of any such criterion must stand or fall by its correlation with an independent criterion of the adequacy of tissue perfusion. In the last analysis, the only available reference value is the whole-body oxygen consumption in relation to the prevailing oxygen requirement of the tissues.

We have measured whole-body oxygen consumption under a variety of conditions in patients during cardiac operations with cardiopulmonary bypass, with the objects of (1) gaining information on the relation between oxygen consumption and oxygen requirement and (2) obtaining data against which other criteria of tissue perfusion can be assessed. In this paper we report our results on oxygen uptake (a) before anaesthesia, (b) during bypass, both normothermic and hypothermic, with immediate or delayed cooling and with varying pump flow, and (c) in relation to body size.

METHODS

Twenty-seven patients were studied. Clinical and other details are shown in table I. They formed part of three successive experimental series which differed in design.

Series I. Patients 1–7 have been described previously (Harris, Seelye and Barratt-Boyes, 1970, patients 20, 16, 13, 12, 9, 8 and 5). Patients 1 and 2 were kept at normal temperature, for 5 and 7 minutes respectively, after cardiopulmonary bypass was started, and were then cooled to 30°C. Patients 3–7 were cooled immediately. During hypothermia, flow was kept roughly constant in each case at 2.0 l./min/m² or more. These patients could be described as having perfusions with "warm" or "cold" starts and constant flow.

Series II. Patients 8–18 all had "warm starts" lasting up to 17 minutes. During hypothermia, except in patient 18, pump flow was deliberately varied at 20-minute intervals; each patient was perfused at either two or three rates (around 50, 60 and 75 ml/kg body weight/min). The order of flow rates was varied (see table I). All but one of the patients in this series thus had "warm starts" and varied flows.

Series III. These studies were similar to those of series I except that a duplicate measurement of oxygen uptake was made, in each patient, in a
TABLE I

Clinical data on patients studied.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, sex</th>
<th>Operation</th>
<th>Duration of warm start (min)</th>
<th>Duration of hypothermia (min)</th>
<th>Measurements during hypothermia</th>
<th>Duration of bypass (min)</th>
<th>Lowest temperature (°C)</th>
<th>Flow during hypothermia (ml/kg/min)</th>
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<td>132</td>
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<td>HAV, MV, TA</td>
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</table>

MSV: Mitral Starr valve.
HAV: Homograft aortic valve.
RT of F: Repair Tetralogy of Fallot.
ASV: Aortic Starr valve.
TA: Tricuspid annuloplasty.
HPV (M): Homograft pulmonary valve replacing mitral valve.
HAV (M): Homograft aortic valve replacing mitral valve.
MV: Mitral valvotomy.

calm or drowsy state just before the induction of anaesthesia. Patient 19 was perfused with a "warm start" lasting 12 minutes. In the remainder, cooling to 30°C was started as soon as bypass began. Arterial pump flow, during hypothermia, was kept roughly constant in each case at 2.0 l./min/m² or more.

The procedure used for perfusion and details of laboratory methods have been described (Barratt-Boyes, 1965; Harris, Seelye and Barratt-Boyes, 1970). Oxygen consumption was measured, pre-operatively in series III, by the standard Douglas-bag method and Lloyd-Haldane analysis of expired gas. During bypass, oxygen consumption was calculated from the flow rate of the calibrated arterial pump and the arteriovenous oxygen content difference; blood samples were drawn from the venous line before the blood reached the venous reservoir and from the arterial outflow from the pump oxygenator. Blood oxygen content was calculated from haemoglobin concentration, oxygen saturation and oxygen partial pressure, using a Höffner factor of 1.36 and the solubility coefficient appropriate to the patient's temperature, as recorded from a thermistor in the nasopharynx.

In series II, oxygen consumption was measured on bypass before cooling, during hypothermia 10
and 20 minutes after each change of flow, and again after rewarming but while still on full bypass. In series I and series III the measurements were made less regularly; “warm start” patients all had one measurement before cooling; all had at least one measurement after rewarming, and from two to seven measurements during hypothermia, depending on the duration of this phase.

Calculations.

According to the Arrhenius equation, the logarithm of the rate of a chemical reaction is inversely related to the reciprocal of the absolute temperature. Over a small range of temperature (37–30°C; 310–303 K) this relationship is almost equally well described by stating that the logarithm of the reaction rate is directly related to the temperature. In this form, it finds expression in van’t Hoff’s law, which states that a rise in temperature of 10°C increases the reaction rate by between two and three times. This proportional increase (or, in cooling, decrease) is usually denoted by the symbol Q_{10}. We have therefore considered the logarithm of oxygen consumption in relation to temperature and have expressed this relationship in terms of the Q_{10}.

From consideration of critical oxygen tensions in tissues (Stainsby, 1966) the oxygen uptake (V_{O_2}) might also be expected to vary with pump-flow (Q) independently of temperature. Clowes and associates (1958) published a curve for dogs which, on analysis, shows a correlation of 0.997 between log V_{O_2} and log Q, both corrected for body weight; V_{O_2} varied directly as the power 0.524 of Q. In considering flow we have therefore postulated a log-log relationship with oxygen consumption, and in averaging data within and between individuals (see below) we have used geometric means for both oxygen uptake and flow.

Temperatures have been averaged arithmetically. Regression analyses and tests of significance have followed standard procedures (Davies, 1961).

RESULTS

(1) Correction of oxygen consumption for body size.

The basal oxygen consumption measured just before anaesthesia in the nine patients in series III, and in two patients not otherwise included in this paper, has been correlated with body weight, surface area, weight to the power 0.6 (Hill, 1966) and weight to the power 0.75 (Kleiber, 1947). The correlation coefficients were respectively 0.770, 0.732, 0.769 and 0.769. By z-transformation these correlations do not differ significantly. For present purposes, therefore, oxygen consumption has been corrected to a standard body weight of 65 kg by multiplying it by the factor 65/body weight in kg.

(2) Effects of varying temperature and flow on oxygen consumption.

These were assessed in the following manner. Single values for weight-corrected oxygen uptake (V_{O_2}^{65}), flow in ml/kg/min (Q) and temperature (t) were obtained for each patient in each temperature state; thus, a set of three values was obtained in each of three states for the “warm start” patients (precooling, hypothermic and rewarmed) and in each of two states for the “cold start” patients (hypothermic and rewarmed). In each case, geometric means were taken for V_{O_2}^{65} and Q, and an arithmetic mean for t. The averaged data were arranged as follows:

(a) Cold to warm (“cold start” patients)
(b) Warm to cold
(c) Cold to warm (“warm start” patients)

A multiple-regression analysis for (a), (b) and (c) was then carried out of log V_{O_2}^{65} on t and log Q according to the hypothetical equation

\[ \log V_{O_2}^{65} = a + b_1t + b_2 \log Q \]

where \(b_1\) and \(b_2\) are the partial regression coefficients on \(t\) and \(\log Q\) respectively. Partial correlations, \(r_t\) and \(r_Q\), were also calculated. The results are shown in table II.

It can be seen that in neither group of patients did flow appear to affect oxygen consumption. The correlations with temperature were, however, highly significant. In the “cold start” group, the slope of log V_{O_2}^{65} on \(t\) was 0.04339, corresponding to a Q_{10} of 2.72. The “warm start” slopes of 0.02311 and 0.01942, for cooling and warming respectively, were not significantly different from each other (0.3 > 2P > 0.2) but were significantly different from the slope for the “cold start” patients (2P < 0.001). The mean slope for the “warm start” patients, 0.02127, corresponds to a Q_{10} of 1.63.
Since series I, II and III had been studied in that sequence over a period of 2–3 years, it was thought that the difference in \( Q_{10} \) between "warm" and "cold" starts might be due to a systematic technical error. Against this was the fact that most of the "warm start" patients were in series II and were thus studied in the middle of the period. To test the postulate further, the mean slopes of \( V_{26} \) on temperature for the "cold start" patients in series I and series III were calculated separately and were not found to differ significantly (0.7>2P>0.6).

The difference between the two groups of patients is illustrated in figures 1 and 2. In figure 1 the mean \( V_{26} \) for the "cold start" patients of series III is seen, at each temperature, to be insignificantly different from that for the "cold start" patients in series I and III combined. The \( V_{26} \) measured before the induction of anaesthesia in series I and series III were calculated separately and were not found to differ significantly (0.7>2P>0.6).

The difference between the two groups of patients is illustrated in figures 1 and 2. In figure 1 the mean \( V_{26} \) for the "cold start" patients of series III is seen, at each temperature, to be insignificantly different from that for the "cold start" patients in series I and III combined. The \( V_{26} \) measured before the induction of anaesthesia in series III is, however, significantly higher than the Rewarmed \( V_{26} \) in either or both series. Figure 2 shows the results in the "warm start" patients. The mean \( V_{26} \) before cooling was not significantly different from that after rewarming, and both were insignificantly different from the mean \( V_{26} \) in the "cold start" series III patients after rewarming. On the other hand, when cooled, the "warm start" patients showed a significantly smaller reduction of \( V_{26} \) in relation to temperature than the "cold start" patients of series III.

(3) Effect of flow on oxygen consumption at uniform temperature.

It seems possible that the multiple-regression analysis of all three series might have obscured a small effect of flow on oxygen consumption, since (a) the varying flows of series II were confounded with the much more uniform flows of series I and

\[
\begin{array}{cccccc}
\text{Patients} & b_1 & r_1 & b_2 & r_2 \\
1 & \text{"Cold start"} & & & \\
& \text{Cold to warm} & 0.04339 & 0.601 & -0.36499 & -0.110 \\
& n = 26 & (Q_{10} 2.72) & 0.01>2P>0.001 & 2P<0.1 \\
2 & \text{"Warm start"} & & & \\
& \text{(a) Warm to cold} & 0.02311 & 0.554 & 0.20673 & 0.112 \\
& n = 28 & (Q_{10} 1.70) & 0.01>2P>0.001 & 2P<0.1 \\
& \text{(b) Cold to warm} & 0.01942 & 0.525 & 0.33026 & 0.195 \\
& n = 28 & (Q_{10} 1.56) & 0.01>2P>0.001 & 2P<0.1 \\
3 & \text{Series II} & & & \\
& \text{Hypothermia} & 0.06280 & 0.510 & 0.21997 & 0.330 \\
& \text{(individual data)} & & & \\
& n = 47 & & 2P<0.001 & 0.05>2P>0.01 \\
\end{array}
\]

Fig. 1

Geometric means of weight-corrected oxygen consumption, and arithmetic mean of temperature were calculated for each "cold-start" patient in series I and III; these means were then averaged in the same way for series III and for series I and III combined. The bars show 2 standard errors of the mean, on each side of the mean, for each variable. The pre-operative measurements of oxygen consumption and temperature in the patients of series III are shown at X.
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1117

Fig. 2

Procedure as for fig. 1, showing the “warm start” patients (2 from series I, 11 from series II, and 1 from series III) in comparison with the 8 “cold start” patients of series III, for whom the pre-operative data are shown at X.

III, and (b) the range of temperature might have biased the correlations in favour of temperature. To test this possibility, the individual data for the hypothermic state in series II patients were considered separately; this procedure reduced the temperature range and relatively increased the range of flow. The result is shown in table II.

V₀₂₅₅ is now seen to be related directly to the power 0.220 of flow (b₅), with a significant correlation (r₂). In this test the significance of the correlation is, of course, enhanced by the larger number of data (47) compared with the analysis of means for each patient in the upper part of table II.

(4) Comparison of basal V₀₂₅₅ with the V₀₂₅₅ during normothermic perfusion.

Basal V₀₂₅₅ was measured pre-operatively in only the nine patients of series III, eight of whom had a “cold start”; it is thus impossible from the present data to make a direct comparison of oxygen uptakes before anaesthesia and immediately after the start of bypass at normal temperature. However, inspection of figures 1 and 2 shows that the mean V₀₂₅₅ during normothermic perfusion is not significantly different in the “warm start” group (whether before cooling or after rewarming) and the “cold start” group. It is thus reasonable to compare the pre-operative V₀₂₅₅ with the mean normothermic V₀₂₅₅ for all patients. The difference was significant (0.02>2P>0.01) and indicated a fall of 16 per cent in V₀₂₅₅ between the pre-anaesthesia measurement and that made during normothermic perfusion. The difference in mean temperature, 0.26°C, was inconsiderable (0.3>2P>0.2).

DISCUSSION

Measurement of temperature and oxygen consumption.

The correlation of V₀₂ with temperature depends, in the first place, on how accurately each can be measured.

(a) Temperature. It is well established that temperature gradients exist throughout the body, even in the normal state, that different regions cool at different rates, and that the pattern of cooling rates varies with the method of cooling used. Our own experience, for example, confirms that nasopharyngeal temperature is above muscle temperature, before cooling, by an average of 1.7°C (0.005>2P>0.001 by paired t-test); at around 30°C, muscle is warmer than nasopharynx by an average of 1.4°C (0.02>2P>0.01); after rewarming the nasopharynx is again the warmer by an average of 2.9°C (2P<0.001). Our use of nasopharyngeal temperature in the present studies is, therefore, open to immediate criticism on the grounds that it is unrepresentative of the whole body, but we know of no way in which this objection can be met.

(b) Oxygen consumption. The measurement of V₀₂ during perfusion, from pump flow and arteriovenous oxygen-content difference, cannot be checked by an independent measurement. Lacking this, we have examined the difference between two successive measurements of V₀₂ and 20 minutes after changing flow at the same temperature, in the patients of series II. The second measurement was, on average, 0.48 per cent higher than the first, a difference which was not signifi-
cant (0.9 > 2P > 0.8). While it cannot be assumed, a priori, that there should not have been any difference between these “duplicate” measurements, we conclude that this method for determining Vo₂ on full bypass is probably satisfactory.

**Oxygen consumption during normothermic perfusion.**

Our results indicate that oxygen consumption is the same, during normothermic perfusion, before cooling and after rewarming. In each case it is, on average, 84 per cent of the pre-operative oxygen uptake in the premedicated patient.

Theye and Tuohy (1964) measured oxygen consumption during light halothane anaesthesia, without premedication, and compared it with predicted (not measured) values for basal oxygen consumption. If a relaxant drug had not been given the values were the same. After tubocurarine, Vo₂ fell to 84 per cent of the predicted basal value.

We conclude that the fall in Vo₂ between our pre-operative measurement and that made during normothermic perfusion was probably due to the effects of muscular relaxation, and that the measured Vo₂ in each case probably represents the prevailing oxygen requirement.

**Oxygen consumption during hypothermic perfusion.**

The fall in Vo₂ observed after cooling appeared to depend, in the present studies, on the manner in which cooling was effected. If the perfusion began with a normothermic phase, Q₁₀ was 1.63; if the patient was immediately perfused with cooled blood, Q₁₀ was 2.72. It would seem that, at least in the latter group, oxygen availability fell short of requirement in the cooled state. It remains to consider whether, even in the patients who had an initial normothermic phase, oxygen requirement was being met at 30°C.

Table III shows Q₁₀ values obtained by other workers under various conditions. It can be seen that apart from the value of 1.51 observed in dogs by Clowes and his colleagues (1958), all values are higher than the 1.63 of our “warm start” patients. It is perhaps especially notable that Shapiro and Stoner (1966) obtained a value of 1.93 in two healthy subjects who were heated to 39.9°C; the peripheral vasodilatation due to heating would presumably ensure that uptake of oxygen kept pace with requirement. In the light of these data we conclude that in those of our patients whose perfusion had a normothermic start, oxygen consumption in the hypothermic state was probably a fair reflection of requirement.

Since the Q₁₀ in these patients was relatively low, the question indeed arises as to whether Vo₂ may have somewhat exceeded current requirement, as it might have done were an oxygen debt, incurred during the cooling phase, being repaid. This question is difficult to answer. We have previously shown (Harris, Seelye and Barratt-

### Table III

*Q₁₀ values from previous studies.*

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<th>Author(s)</th>
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<td>Clowes et al. (1958)</td>
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<td>Brewin (1964)</td>
<td>30</td>
<td></td>
<td>1.85</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>Man</td>
<td>2.26</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td></td>
<td>2.37</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td></td>
<td>2.55</td>
</tr>
<tr>
<td>Shapiro and Stoner (1966)</td>
<td>40 Man (heating)</td>
<td></td>
<td>1.93</td>
</tr>
<tr>
<td>Lunding and Rygg (1968)</td>
<td>33</td>
<td></td>
<td>3.18</td>
</tr>
<tr>
<td></td>
<td>28.5</td>
<td>Man</td>
<td>2.08</td>
</tr>
<tr>
<td></td>
<td>23</td>
<td></td>
<td>2.26</td>
</tr>
</tbody>
</table>
Boyes, 1970) that in this “warm start” group of patients the blood lactate concentration rose almost twice as rapidly, during the hypothermic phase, as in the “cold start” patients, and that this difference was, statistically, highly significant. This observation would hardly, at first sight, suggest that the patients in the “warm start” group were paying off an oxygen debt at the same time, but this is quite conceivable. Elsewhere (Seelye et al., 1971) we have presented clear evidence of oxygen debt repayment in babies at a time when “washout” lactic acidosis was increasing very rapidly indeed. At present we are reluctant to call upon evidence derived from “excess lactate” or lactate/pyruvate ratio, since this would beg the question of whether these indices provide reliable evidence of oxygen availability or not. There remains a possible indication that oxygen requirement was not in fact being fully met in our cooled “warm start” patients, and this will be considered below in connection with changes related to pump flow.

By contrast, the Q₁₀ for the “cold start” perfusions was, at 2.72, considerably higher than the heating Q₁₀ of Shapiro and Stoner (1966), and lies in the upper part of the range shown in table III. It seems almost certain that during hypothermia in this group the oxygen availability fell short of requirement by as much as 15 per cent. The provision of a “warm start” lasting up to 15 minutes or so would therefore seem to assure the tissues of a better oxygen supply during the hypothermic phase. In most cases this advantage is probably of minor consequence, and the “warm start” technique certainly adds to the hazards, at this stage of the operation, in patients with aortic reflux whose hearts suddenly fibrillate before the heart is cool or the coronary arteries have been cannulated. In some instances, however, it may possibly be a desirable means of reducing the metabolic insult to which the patient is exposed.

Oxygen consumption in relation to pump flow.

The data for series II shown in table II suggest a positive correlation between Vo₂ and flow over the range 50–75 ml/kg/min. From the observed relationship that log Vo₂ varies as 0.220 log Q, it may be deduced that Vo₂ increased by 10 per cent as flow increased from 50 to 75 ml/kg/min. For a 65-kg man of 1.7 m² surface area, the commonly accepted minimal flow of 2.0 l/m²/min at 30°C is equivalent to 52 ml/kg/min. It would appear that higher flows may confer some advantage in more effective tissue perfusion. If this be so, it cannot be held that the mean hypothermic Vo₂ in the “warm start” patients of series II represented the prevailing oxygen requirement, since many of the Vo₂ measurements were made at the lower two flow rates.

Comments

In the absence of continuous, accurate measurement of oxygen consumption and exact knowledge of the mass of lactate released and excreted, the oxygen requirement can never be precisely known. It is virtually certain that most estimates in the literature, of oxygen consumption during hypothermic bypass, represent not what the tissues need so much as what they can get. If one can be reasonably sure that requirement is met under a certain set of conditions, then the Q₁₀ offers a guide as to what effect a change of temperature has had, other conditions remaining essentially unchanged. The lower the Q₁₀ during cooling the more likely it is that oxygen needs are being supplied. Presumably there is, theoretically, a limiting value at which one can say that oxygen supply is just sufficient, and we have somewhat arbitrarily taken the “heating” Q₁₀ of Shapiro and Stoner (1966) as being near this limit. Unfortunately, any attempt at precision is probably doomed to failure by the impossibility of measuring “body temperature”. The present studies, nevertheless, form a possible basis for further consideration of venous Po₂, arteriovenous oxygen difference, and pyruvate and lactate concentrations as indices of adequate perfusion.

ACKNOWLEDGEMENTS

We are grateful to Sir Brian Barratt-Boyes and Mr D. S. Cole for the opportunity to study patients under their care, and for their equanimity in the operating theatre during our investigations. We also wish to thank Mrs C. Ford, B.sc, Mrs M. Barron, B.sc, and Mrs A. Kenyon, B.sc, for laboratory assistance. This work was supported by successive grants from the Auckland Medical Research Foundation, the Golden Kiwi Lottery Fund, and the Medical Research Council of New Zealand.

REFERENCES


**CONSOMMATION D'OXYGENE AU COURS D'UN “BY-PASS” CARDIOPULMONAIRE AVEC HYPOTHERMIE MODEREE CHEZ L'HOMME**

La consommation d’oxygène a été mesurée au cours d’une circulation totale sous hypothermie, mise en oeuvre chez 27 malades soumis à des interventions de chirurgie cardiaque. Chez neuf malades, cette mesure a été aussi effectuée immédiatement avant l’induction de l’anesthésie. Quatorze malades étaient maintenus à une température normale pendant les 5 à 17 minutes qui suivirent le début du “bypass”. Les autres malades furent immédiatement placés en hypothermie à 30°C. La consommation d’oxygène au cours du “bypass” en normothermie, soit avant ou après réfrigération, correspondait à 84% de la valeur mesurée avant l’anesthésie, ce qui pouvait être dû aux effets de la myorelaxation. Au cours de l’hypothermie, les malades qui avaient subi immédiatement la réfrigération présentaient une consommation d’oxygène qui était inférieure de 15% environ à celle enregistrée chez les malades soumis à une réfrigération retardée. Même dans ce dernier groupe, la consommation d’oxygène au cours de l’hypothermie n’exigeait la mise en oeuvre d’aucune mesure spéciale, étant donné qu’elle s’aurait être tout à fait en relation avec le flux sanguin.

**SAUERSTOFF-VERBRAUCH WÄHREN CARDIOPULMONALER ÜBERBRÜCKUNG MIT MÄSSIGER HIPOTHERMIE BEIM MENSCHEN**

ZUSAMMENFASSUNG

Bei 27 Herzoperationen wurde der Sauerstoff-Verbrauch während hypothermer Ganzkörper-Perfusion gemessen; in 9 Patienten auch kurz vor Beginn der Narkose. 14 Patienten wurden von 5–17 Minuten nach dem Beginn der Überbrückung bei normaler Temperatur gehalten, die restlichen sofort auf 30°C unterkühlt. Der Sauerstoffverbrauch während der Überbrückung betrug bei den Normothermen—sowohl vor als auch nach der Unterkühlung—84% des Wertes vor der Narkose, was wahrscheinlich auf Muskelrelaxation zurückzuführen ist. Während der hypothermen Phase zeigten die Patienten, die sofort unterkühlt worden waren, einen etwa 15% niedrigeren Sauerstoff-Verbrauch als die Patienten mit verzögter Unterkühlung. Selbst in dieser letzteren Gruppe jedoch reicht anscheinend die Sauerstoff-Versorgung während der Hypothermie nicht völlig aus, wie die positive Korrelation zum Blutfluss zeigt.

**CONSUMO DE OXIGENO DURANTE EL BYPASS CARDIOPULMONAR CON HIPOTERMIA MODERADA EN EL HOMBRE**

RESUMEN

El consumo de oxígeno fue medido durante la perfusión hipotérmica de todo el cuerpo en veintiisiete pacientes sometidos a operaciones cardíacas. En nueve pacientes, también fue medido inmediatamente antes de la inducción de anestesia. Catorce pacientes fueron mantenidos a temperatura normal durante 5 hasta 17 minutos después de comenzar el bypass; los demás fueron inmediatamente enfriados a 30°C. El consumo de oxígeno durante el bypass con normotermia, tanto antes como después del enfriamiento, fue de 84 por ciento del valor pre-anestesia, probablemente a causa de los efectos del relajamiento muscular. Durante la hipotermia, los pacientes que fueron enfriados inmediatamente tuvieron un consumo de oxígeno que era inferior en aproximadamente un 15 por ciento al encontrado en pacientes con enfriamiento retardado. Incluso en este último grupo es probable que el consumo de oxígeno durante la hipotermia no llegara a satisface completamente los requisitos, ya que se demostró que estaba correlacionado positivamente con el flujo sanguíneo.