The effects of shivering on oxygen consumption and carbon dioxide production in patients rewarming from hypothermic cardiopulmonary bypass

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Oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), end–tidal carbon dioxide partial pressure ($P_{ET}CO_2$), mixed venous oxygen saturation ($S\bar{v}O_2$) and haemodynamic variables were recorded every 30 min for four hours in 15 patients recovering from hypothermic cardiopulmonary bypass (CPB). All patients had been anaesthetised with fentanyl 40 $\mu g \cdot kg^{-1}$, supplemented with isoflurane, and pancuronium 0.15 $mg \cdot kg^{-1}$ for muscle relaxation. Three of the 15 patients (20 per cent) shivered, defined as intermittent or continuous, vigorous movements of chest or limb muscles. Patients who shivered had a $\dot{V}O_2$ of 159 ± 16.4 $ml \cdot min^{-1} \cdot m^{-2}$ on arrival in the ICU which rose to a maximum value of 254 ± 28.3 $ml \cdot min^{-1} \cdot m^{-2}$ by 150 min post-CPB. In contrast, patients who did not shiver had a significantly lower $\dot{V}O_2$ of 93.1 ± 6.9 $ml \cdot min^{-1} \cdot m^{-2}$ on arrival in the ICU which rose to a maximal value of only 168 ± 11.5 $ml \cdot min^{-1} \cdot m^{-2}$ by 180 min post-CPB. Maximal $\dot{V}O_2$ in both groups was reached when the nasopharyngeal temperature (NPT) was approaching normal. $\dot{V}CO_2$ paralleled the increase in $\dot{V}O_2$ in both groups. By four hours there was no significant difference between the two groups; however, the $\dot{V}O_2$ in both groups (160.5 ± 21.3 $ml \cdot min^{-1} \cdot m^{-2}$ and 173.9 ± 12.3 $ml \cdot min^{-1} \cdot m^{-2}$ respectively) was approximately twice values commonly measured in anaesthetized patients. Patients who shivered had a significantly higher heart rate and cardiac index and significantly lower $S\bar{v}O_2$. We conclude that the high $\dot{V}O_2$ and $\dot{V}CO_2$ associated with shivering causing increased myocardial work may be detrimental to patients who have impaired cardiac function post-coronary artery surgery (CAS).

Key words

ANAESTHETIC AGENTS: fentanyl; SURGERY: cardiac; PHYSIOLOGY: oxygen consumption, carbon dioxide production, hypothermia; MONITORING: mixed venous O_2 saturation, end–tidal CO_2 concentration.

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Following hypothermic cardiopulmonary bypass (CPB), patients usually arrive in the Intensive Care Unit (ICU) with a nasopharyngeal temperature (NPT) of 34–36°C.[1] Sladen *et al.* showed that over the subsequent 8 hr patients rewarm to normothermia, with the maximal rate of rewarming occurring 2–4 hr after admission to the ICU.[2] During this period of very rapid rewarming, marked changes have been suggested in both metabolic rate and myocardial work.[3,4] Increases in O_2 consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) are undesirable in post-CPB patients because they lead to increases in heart rate (HR), mean arterial pressure (MAP) and rate pressure product (RPP)[5] causing an increase in myocardial oxygen consumption.[6] Moreover, if the extent of these metabolic changes is not recognized then both respiratory and metabolic acidosis may occur.

The present study was designed to determine the extent of these metabolic changes and their haemodynamic consequences during the first four hours after CPB. We also wished to determine the effects of shivering on these variables, if these effects are significant and how best to follow their trend.

Methods

Fifteen patients scheduled for elective cardiac surgery were studied. Patients with symptomatic peripheral vas-

cular disease, insulin-dependent diabetes or body weight greater than 20 per cent above their ideal weight were excluded from the study. All patients received a premedication of diazepam (0.15 $mg \cdot kg^{-1}$), morphine (0.15 $mg \cdot kg^{-1}$) and scopolamine(0.006 $mg \cdot kg^{-1}$). Prior to induction of anaesthesia, and under local anaesthesia, an intravenous cannula, a radial artery cannula and a triple lumen pulmonary artery catheter with fibreoptics for continuous monitoring of mixed venous oxygen saturation ($S\bar{v}O_2$) (Opticath, Oximetrix, Inc., Mountain View, California) were inserted. Anaesthesia was induced with fentanyl (40 $\mu g \cdot kg^{-1}$) and pancuronium (0.15 $mg \cdot kg^{-1}$) for muscle relaxation. Anaesthesia was then maintained with 100 per cent oxygen and isoflurane was used, if necessary, to maintain the systolic blood pressure within 20 per cent of ward values. Further increments of pancuronium were administered as required up to the end of cardiopulmonary bypass (CPB), after which no further doses were given until the end of the study period. Temperature probes were inserted into the nasopharynx and rectum prior to the start of surgery. Cardiopulmonary bypass was performed with a Sarns model 7000 Modulator Pump with Optiflow II or Bentley BOS–105 oxygenator, and patients were cooled to 24–28° C NPT. A crystalloid pump prime and flow rates of 2.4 $L \cdot m^{-2}$ at normothermia, reduced by 50 per cent at 28° C, with a haematocrit maintained above 20 per cent was used in all patients. During rewarming on CPB, isoflurane or an infusion of nitroglycerine was used to maximize flow rates until the NPT was 37–38° C and the rectal temperature (RT) was greater than 34° C.

After transfer to the ICU, heart rate (HR), systolic and mean blood pressures (SBP, MBP), mean pulmonary artery pressure (MPAP), pulmonary capillary wedge pressure (PCWP),central venous pressure (CVP), $\dot{V}O_2$, $\dot{V}CO_2$, cardiac output (CO), NPT, $S\bar{v}O_2$ and $P_{ET}CO_2$ were determined at 30-minute intervals from 90 min (used as a baseline value) to 4 hr after termination of CPB. The $\dot{V}O_2$ was calculated by directly measuring the arterial and venous oxygen contents using a Lex O_2 CON-TL oxygen analyser (Lexington Instruments Corp., Waltham, Massachusetts) and multiplying the difference by the cardiac output. $\dot{V}CO_2$ was calculated using the formula

$$\dot{V}CO_2 = F_ECO_2 \times \dot{V}_E(STPD)$$

The fractional expiratory CO_2 concentration (F_ECO_2) was measured by mass spectrometry (Spectronic 710, Bausch and Lomb) after collecting expired gases for 2 min. This collection was made from the expiratory port of the ventilator using unidirectional valves to prevent contamination by inspiratory gases. The expired minute volume ($\dot{V}_E$) was calculated as half the collected volume after correcting to standard temperature and pressure (STPD).

Cardiac output was determined by averaging three consecutive measurements derived by the thermodilution technique. End–tidal CO_2 ($P_{ET}CO_2$) was displayed continuously using a capnometer (Model 14360A, Hewlett Packard, Waltham, Massachusetts) and the patient's tidal volume was adjusted after each collection period to maintain a $P_{ET}CO_2$ between 35–40 mmHg. Shivering was defined as intermittent or continuous vigorous movements of the chest or limb muscles. Any patients who shivered were included in group S at all time intervals, and those who did not shiver in group NS. Muscle relaxants were not administered in the ICU unless the degree of shivering interfered significantly with ventilation. No overt methods were used to rewarm patients and all fluids were administered at room temperature. Morphine was administered to treat hypertension or restlessness as it has been shown not to be effective in the treatment of shivering in postoperative patients.[7] No patient received any medication that would stimulate their adrenergic system and therefore increase $\dot{V}O_2$.

Mean values were calculated for each variable at each time interval. Correlation coefficients were calculated between the rate of rise of NPT and both the $\dot{V}O_2$ and $\dot{V}CO_2$. Total dose of pancuronium received and the time from the last dose of muscle relaxant until the start of the study was recorded. Differences between the two groups were compared using the Student's t test and the null hypothesis was rejected when $p < 0.05$.

Results

The three patients who shivered were placed in group S, and the 12 who did not shiver in group NS. There was no significant difference in the demographic data between the two groups (Table). The fall in NPT after the termination of CPB (afterdrop) that occurred in both groups and the subsequent return of this temperature to normal over the following 2–4 hr is shown in Figure 1. Afterdrop was comparable in both groups but patients who shivered had a significantly higher NPT from 210 to 240 min. The mean values of $\dot{V}O_2$, $\dot{V}CO_2$, $S\bar{v}O_2$, $P_{ET}CO_2$ and NPT in the two groups at each time interval

TABLE Demographic data (mean ± SEM)

Group	*Age (Yr)*	*BSA (M)*	*Sex*	*Operation*	*Bypass time (mins)*	*Rewarming time (mins)*
Non-shivering	59.5 +2.5	1.8+ 0.06	M:8 F:4	ACBP:8 Others:4	104.9 +12.6	44.9 +3.0
Shivering	71.3 +5.9	1.9+ 0.06	M:3 F:0	ACBP:2 Others:1	125.0 +18.0	46.7 +6.1

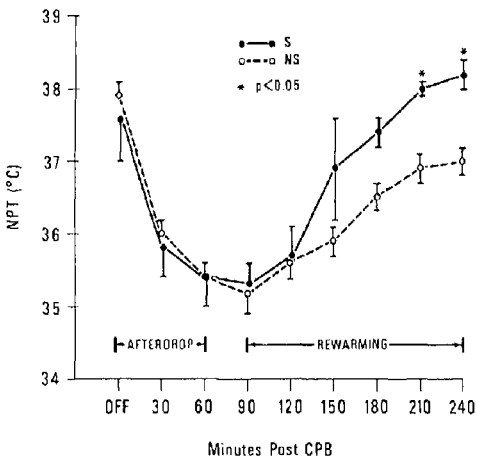

FIGURE 1 The fall (afterdrop) and subsequent rise (rewarming) in nasopharyngeal temperature (NPT) from termination of CPB (off) to 240 min post-CPB. S = patients who shivered; NS = patients who did not shiver.

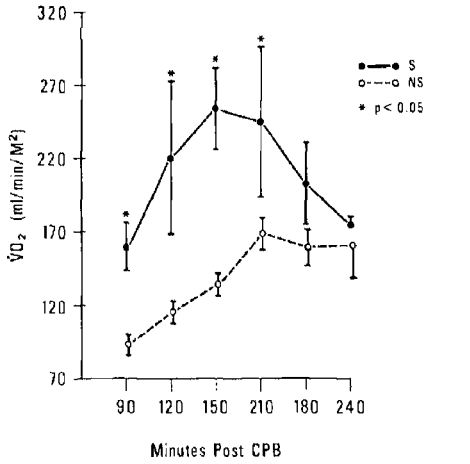

FIGURE 2 Mean values + SEM of oxygen consumption ($\dot{V}O_2$) at the times shown post-CPB in patients who shivered (S) and those who did not (NS).

are shown in Figures 1–4. There were significant differences in $\dot{V}O_2$ and $\dot{V}CO_2$ from 90 to 180 min, in $S\bar{v}O_2$ from 120 to 180 min and in $P_{ET}CO_2$ from 90 to 150 min. On arrival in the ICU the mean $\dot{V}O_2$ was significantly higher (71 per cent) in group S compared to group NS (159 ± 16.4 ml · min^{-1} · m^{-2} vs 93 + 6.9 ml · min^{-1} · m^{-2}) (Figure 2). Maximal mean value of $\dot{V}O_2$ was seen at 150 min in group S and at 180 min in group NS (254 ± 28.3 ml · min^{-1} · m^{-2} vs 168 ± 11.5 ml · min^{-1} · m^{-2}), when the mean NPT was 36.9 ± 0.2° C and 36.5 ± 0.2° C respectively. By the end of the study no patient was shivering and the $\dot{V}O_2$ was comparable in the two groups although 72 per cent above NS baseline values (Figure 2).

$\dot{V}CO_2$ increased gradually over the study period by 28 per cent in group NS. In group S baseline values were elevated by 55 per cent above NS values (136 ± 15.7 ml · min^{-1} · m^{-2}) and rose a further 35 per cent to 184 ± 40.5 ml · min^{-1} · m^{-2} at 120 min then gradually decreased to NS values by the end of the study (Figure 3).

Patients in the NS group had a mean $S\bar{v}O_2$ above 60 per cent at all time intervals. In Group S, $S\bar{v}O_2$ was never greater than 60 per cent, the minimal mean value reaching 50 per cent at 150 min when the mean $\dot{V}O_2$ was maximal for this group. Cardiac index was significantly higher in Group S from 90 to 210 min compared with Group NS (Figure 4). The only other significant differences in haemodynamic variable between the two groups was a significantly higher HR (120–180 min) (Figure 5) and MPA (90 min) in Group S.

There was no correlation with $\dot{V}O_2$ nor $\dot{V}CO_2$ and the rate of rise of the NPT. The temperature at which maximal $\dot{V}O_2$ occurred was variable; however greater than 85 per cent of patients who had a $\dot{V}O_2$ greater than 150 ml · min^{-1} · m^{-2} had a NPT above 36° C.

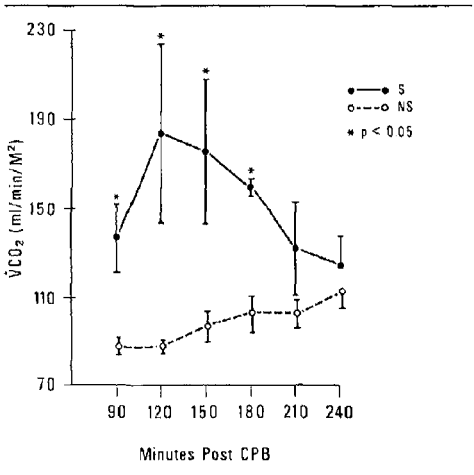

FIGURE 3 Mean values + SEM of carbon dioxide production ($\dot{V}CO_2$) at the times shown post-CPB. S = shivering patients; NS = non-shivering patients.

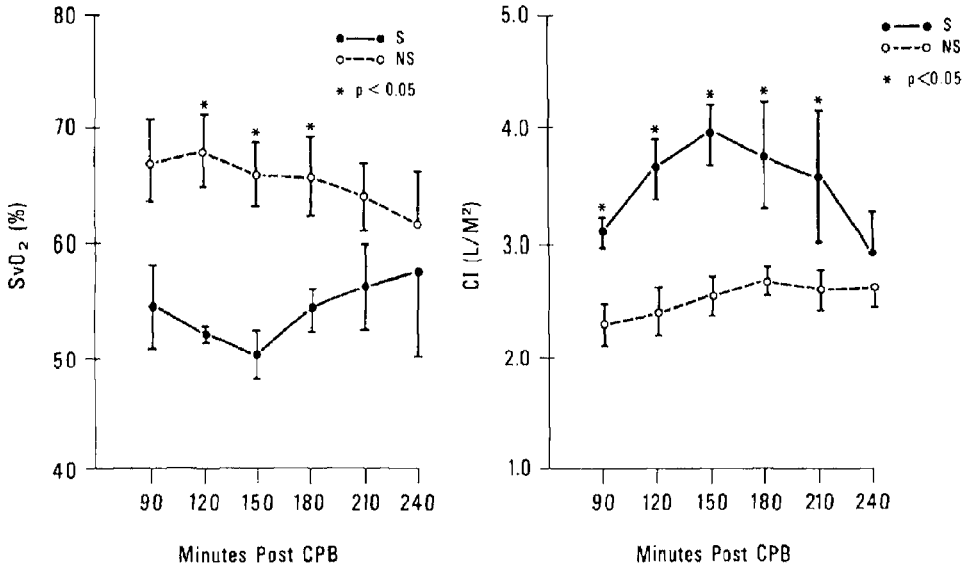

FIGURE 4 Mean values + SEM of mixed venous O_2 saturation ($S\bar{v}O_2$) (left graph) and cardiac index (CI) (right graph) over the time period shown post-CPB. S = shivering patients; NS = non-shivering patients.

There was no significant difference between the two groups in the mean total dose of muscle relaxant received (0.16 ± 0.006 mg·kg^{-1} in group S vs 0.15 ± 0.009 mg·kg^{-1} in group NS) nor in the mean time from last dose of pancuronium until the start of the study (163.3 ± 19.2 min in group S vs 174.1 ± 24.7 min in group NS). No patient received a muscle relaxant in the ICU during the period of the study.

Discussion

This study demonstrates that the early hours post-CPB are associated with significant elevations in $\dot{V}O_2$, $\dot{V}CO_2$, HR and CO, which are markedly further increased if patients shiver. Basal $\dot{V}O_2$ in the resting awake patient approximates 130 ml·min^{-1}·m^{-2},[8] and under fentanyl anaesthesia ranges from 85–100 ml·min^{-1}·m^{-2}.[9] On arrival in the ICU NS patients had $\dot{V}O_2$ values within the expected range for patients anaesthetised with fentanyl. In contrast patients in group S on arrival in the ICU had a significantly higher $\dot{V}O_2$ despite the fact that none of these patients were visibly shivering at this time. It is possible that the higher $\dot{V}O_2$ seen in group S at this time may be a predictor of patients who will shiver, although this is yet to be confirmed.

The maximal values of $\dot{V}O_2$ in the NS group reached twice normal anaesthetised values. Although these

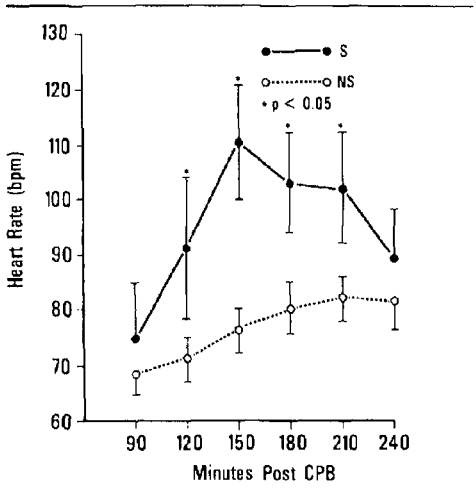

FIGURE 5 Mean values + SEM of heart rate (HR) over the time period shown post-CPB. S = shivering; NS = non-shivering.

patients were not visibly shivering they may have had increased muscular activity not detected by the methods used in this study. A more sensitive method of detection

of muscular activity, such as the use of electromyography may have demonstrated this activity. However the definition of shivering as used in this study is commonly used in the ICU and adequately identified those patients who had a significant increase in $\dot{V}O_2$ and $\dot{V}CO_2$ associated with a significant rise in CO and decrease in $S\bar{v}O_2$. These findings agree with those of Guffin *et al.*,[7] who although used a more sensitive grading of shivering only treated those who shivered to a similar degree as our patients.

By 4 hr post-CPB, $\dot{V}O_2$ was the same in both groups, but still above expected anaesthetized values.[9] This could be due to a return of the $\dot{V}O_2$ to awake values in patients emerging from anaesthesia, or more likely to the ongoing repayment of the residual thermal debt of hypothermic CPB, despite the normal NPT. At the end of CPB there is a "heat deficit" of over 300 KJ, which is gradually repaid over the subsequent hours after CPB.[10] Although by the end of 4 hr much of this deficit has been repaid, there may still be areas of muscle or subcutaneous fat that have not yet equilibrated with the core temperature.

We were unable to demonstrate any factor that indicated when $\dot{V}O_2$ would be maximal. Benzinger *et al.*[11] showed that maximal heat generating effects occur only when the temperature of the blood reaching the hypothalamus is within $-0.7°C$ of the hypothalamic set point (37.1°C). The hypothalamic set point is the blood temperature which the thermoregulatory centre of the hypothalamus recognizes as normal. This narrow range below the set point that appears to trigger increased metabolic activity was confirmed by Roe *et al.* in postoperative general surgical patients,[4] who showed a range of temperature over which $\dot{V}O_2$ was maximal. When the fall in RT was between -0.3 and $-1.2°C$, $\dot{V}O_2$ was 90 per cent above normal, and temperature variations above or below this range were associated with a significantly lower $\dot{V}O_2$ (17 and 40 per cent respectively). Our results agree with these studies as 85 per cent of the patients with a $\dot{V}O_2$ above 150 $ml \cdot min^{-1} \cdot m^{-2}$ had a NPT of 36°C or above. This suggests that maximal rates of $\dot{V}O_2$ occur when the core temperature of the patient is approaching normal.

Shivering in response to hypothermia can increase tissue oxygen demands by as much as 400–500 per cent.[12,13] As metabolic rate is the major determinant of CO,[14] it is not surprising that, in this study, patients who shivered had a significantly higher CI and HR during the period of maximal muscular activity. The $S\bar{v}O_2$ in patients who did not shiver was within normal values, while patients who shivered showed a significant reduction in $S\bar{v}O_2$, despite a significant increase in their cardiac output. A reduction in $S\bar{v}O_2$ in the presence of an increase in cardiac output indicates an imbalance between whole body oxygen supply and demand. When this imbalance causes the $S\bar{v}O_2$ to fall below 50 per cent anaerobic metabolism may occur.[15] In patients unable to significantly increase their cardiac output to meet the extra demands of shivering, anaerobic metabolism associated with a higher morbidity may occur. The continuous recording of $S\bar{v}O_2$ makes it possible to assess the ability of each patient to match O_2 supply to this increased O_2 demand.

In this study, $\dot{V}CO_2$ showed marked increases only in those patients who shivered. If this increased CO_2 production is not anticipated, ventilation may be inadequate resulting in respiratory acidosis. Despite attempts to anticipate these increases in $\dot{V}CO_2$ and adjust ventilation appropriately we failed to do so in patients who shivered during the period of peak CO_2 production. Better control of $PaCO_2$ could have been accomplished if ventilation had been adjusted more frequently than every 30 min as dictated by the protocol. Sladen *et al.* have shown a high incidence of postoperative respiratory acidosis in ventilated patients recovering from CAS. They attributed this to either increased CO_2 production or increased dead space. This study supports their hypothesis that increased $\dot{V}CO_2$ is the responsible aetiological factor. An elevated $PaCO_2$ stimulates endogenous catecholamine release. When $PETCO_2$ monitoring is not available, increases in pulmonary and/or systemic blood pressure may be an early indicator of an undesirably high $PaCO_2$.

Muscle activity was not monitored and it is possible that in some patients who did not shiver residual muscle paralysis may have prevented them from doing so. However, we feel that this is unlikely as although pancuronium requirements to maintain twitch height at 5–15 per cent of control height have been shown to be significantly reduced after hypothermic CPB, patients still required 63 per cent of the dose required prebypass to maintain the same degree of twitch depression.[16] Therefore it is likely that during the period of the study sufficient muscle activity would have returned to allow the presence of shivering to be manifest. Furthermore, there was no significant difference in the average dose received nor the mean time from the last increment of pancuronium between the two groups. Shivering as defined in this study identified all patients who developed significantly higher $\dot{V}O_2$ compared with those in the non-shivering group. In the study by Guffin *et al.*,[7] increased $\dot{V}O_2$ was only identified in patients who shivered to a similar degree as characterised in our study. The $\dot{V}O_2$ in their patients was not as high as recorded in this study, presumably as they intervened early with meperidene or muscle relaxants which sucessfully reduced the $\dot{V}O_2$. Only three patients shivered in our study, but despite their known good left ventricular function, the increased $\dot{V}O_2$ although associated with a significant increase in cardiac output, lead to a significant fall in $S\bar{v}O_2$ which could have

lead to unnecessary morbidity. The consistently high $\dot{V}O_2$, with its inherent risk to the patient of producing anaerobic metabolism, which can be effectively prevented with the use of muscle relaxants[5] was the reason why only three shivering patients were studied. We now administer muscle relaxant to all our post-CPB patients who shiver during the rewarming period.

In conclusion, this study demonstrates that the early hours after CPB are associated with marked alteration in metabolic demands. Maximal $\dot{V}O_2$ occurs 2–3 hr post CPB and when the NPT is 36° C or above. When shivering occurs it produces a significant increase in $\dot{V}O_2$, $\dot{V}CO_2$, HR and CO associated with a significant decrease in $S\bar{v}O_2$ which in many cases may be unacceptable. Continuous monitoring of the $P_{ET}CO_2$ and the $S\bar{v}O_2$ enables the clinician to assess the ability of each patient to cope with this increased metabolic activity.

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Résumé

La consommation d'oxygène ($\dot{V}O_2$), la production de CO_2 ($\dot{V}CO_2$), la PCO_2 en fin d'expiration ($P_{ET}CO_2$), la saturation d'oxygène du sang veineux mixte ($S\bar{v}O_2$) et les données hémodynamiques ont été enregistrées chaque 30 min pour quatre heures chez 15 patients ayant subi une CEC hypothermique (CPB). Tous les patients ont été anesthésiés avec du fentanyl 40 $\mu g \cdot kg^{-1}$ supplémenté d'isoflurane et de pancuronium 0.15 $mg \cdot kg^{-1}$ pour le relâchement musculaire. Trois des 15 patients (20 pour cent) ont présenté des frissons, définis comme étant des mouvements vigoureux intermittents ou continus des muscles thoraciques ou des muscles périphériques. Les patients ayant présenté des frissons avaient une $\dot{V}O_2$ de 159 ± 16.4 $ml \cdot min^{-1} \cdot m^{-2}$ à l'arrivée aux soins intensifs augmentant à une valeur maximale de 254 ± 28.3 $ml \cdot min^{-1} \cdot m^{-2}$ après 150 min de l'arrêt de la CEC. Par contre, les patients n'ayant pas présenté des frissons avaient des valeurs de $\dot{V}O_2$ significativement plus basses de 93.1 ± 6.9 $ml \cdot min^{-1} \cdot m^{-2}$ à l'arrivée aux soins intensifs augmentant à une valeur maximale de 168 ± 11.5 $ml \cdot min^{-1} \cdot m^{-2}$ 180 min, après l'arrêt de la CEC. La valeur maximale de la $\dot{V}O_2$ dans les deux groupes était atteinte quand la température nasopharyngée (NPT) s'est approchée de la normale. Les valeurs de $\dot{V}CO_2$ augmentèrent en parallèle avec celles de la $\dot{V}O_2$ dans les deux groupes. Après quatre heure il n'y avait aucune différence significative entre les deux groupes, cependant la $\dot{V}O_2$ dans les deux groupes (160.5 ± 21.3 $ml \cdot min^{-1} \cdot m^{-2}$ et 173.9 ± 12.3 $ml \cdot min^{-1} \cdot m^{-2}$ respectivement) était approximativement le double de celle qu'on mesure habituellement chez des patients anesthésiés. Les patients ayant présenté des frissons avaient une fréquence cardiaque et un index cardiaque significativement plus élevés et une $S\bar{v}O_2$ significativement plus basse. On conclut que les valeurs élevées de la $\dot{V}O_2$ et de la $\dot{V}CO_2$ accompagnant les frissons amènent une augmentation du travail myocardique et peuvent être néfastes chez les patients ayant une fonction cardiaque diminuée après la chirurgie coronarienne (CAS).