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Myocardial metabolism and haemodynamic responses during high-dose fentanyl anaesthesia for coronary patients

Fentanyl (mean dose 109 $\mu\text{g}\cdot\text{kg}^{-1}$) and oxygen were given to ten patients having coronary vein grafts. Serial studies were done before, during and after operation, of central and mean arterial pressures (MAP), cardiac index (CI) and coronary sinus flow (CBF) by thermodilution, myocardial oxygen consumption (MVO_2) and lactate extraction (MLE). On induction CI and stroke work index decreased, but heart rate and MAP were unchanged as systemic resistance increased. Mean MAP and heart rate remained at the awake levels. Mean CBF remained unchanged along with stable MAP and coronary resistance. Oxygen content of CS blood increased on induction and remained elevated until the incision; it was above the awake level early postoperatively. MVO_2 was low normal when the patients were awake and remained so. Normal MLE continued with a few exceptions. High-dose fentanyl did not uniformly abolish autonomic reflexes. Heavy premedication, complete beta adrenergic blockade and a high initial doses of fentanyl plus its continued infusion, aided in retaining a hypodynamic circulation and myocardial oxygenation.

Key words

HEART: oxygen consumption; ANAESTHESIA: cardiovascular; ANAESTHETICS, INTRAVENOUS: fentanyl; SURGERY: cardiovascular.

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Since 1978¹ intravenous fentanyl, in doses of 100 $\mu\text{g}\cdot\text{kg}^{-1}$ or more, has become widely used as the major agent for cardiac anaesthesia. The primary advantage initially put forth was the smooth haemodynamic course, i.e., absence of response to sympathetic stimulation, in addition to minimal cardiovascular depression. More recent communications²⁻⁸ reported that even with high doses, hypertension and tachycardia associated with sternotomy were not uniformly prevented. Sonntag *et al.*⁹ found that 100 $\mu\text{g}\cdot\text{kg}^{-1}$ did not protect the myocardium from ischaemia in patients having coronary arterial bypass grafts (CABG). The haemodynamic pattern during fentanyl anaesthesia for cardiac surgery has been frequently documented.¹⁻⁸ Its effects on coronary sinus flow (CBF), myocardial oxygen consumption (MVO_2) and myocardial lactate extraction (MLE) have rarely been reported^{9,10} due to difficulty of measurement in the operating room. The latter can be accomplished by the Ganz coronary sinus catheter.¹¹ With it, we studied the correlation of haemodynamics to coronary flow and myocardial oxygenation before, during and after CABG, in ten patients receiving fentanyl (mean dose 109 $\mu\text{g}\cdot\text{kg}^{-1}$) and oxygen.

Methods

Our protocol was approved by the faculty ethics committee and signed informed consent was obtained. Nine of the patients were male. The mean age was 59 years and mean weight 84 Kg. Disability of NYHA Class was: II – two patients, III – six and IV – two. While all patients had regional areas of hypokinesia, ventricular function was adequate: at cardiac catheterization, mean LVEDP was 12 mmHg

(1 > 20 mmHg). Mean cardiac index was 2.00 l·min⁻¹·m⁻² (range 1.76–2.22). All patients had ejection fractions >0.50 (range 0.50–0.80); all were normotensive preoperatively and were maintained on propranolol (mean dose 42 mg q.i.d.) until operation. The patients did well postoperatively and were discharged at the usual time.

Premedication was with oral diazepam (mean 11 mg) and intramuscular morphine (mean 16 mg). After establishing an intravenous route, additional diazepam (mean 5.5 mg) was given before instrumentation, approximately 75 minutes before induction of anaesthesia.

After premedication and local anaesthesia, a 20 gauge radial arterial needle was placed, followed by two catheters via the right internal jugular vein. First was the usual PA catheter,¹² then the Ganz catheter¹¹ manipulated into the midcoronary sinus with the assistance of fluoroscopy, its position verified by an injection of contrast medium. The latter catheter measured total CBF by thermodilution. Patients with an initial pulmonary capillary occluded (wedge) pressure of less than 12 mmHg, had fluid infused to raise it to 12, so as to begin the study from a more uniform baseline of ventricular function.

After a 10–15 minute quiet period following instrumentation, a control series of measurements were done with the patient awake, breathing air. This consisted of recording arterial and central pressures, measuring CBF and cardiac output (two within ten per cent), plus arterial and CS samples for haemoglobin and oxygen saturation (co-oximetry) and lactate concentration (Boehringer Mannheim, Canada, Ltd.). Subsequent series of measurements were done after induction, after intubation, five times during operation and 1, 3 and 24 hours following surgery (see Figures).

Induction was with fentanyl (75 µg·kg⁻¹) given intravenously in a mean of 11.7 minutes (range 10–14) with pancuronium (mean 8.6 mg) given early to prevent chest wall rigidity. Ventilation throughout was with oxygen, to maintain PaCO₂ at the awake level. After intubation, fentanyl was infused (10 µg·kg⁻¹·hour⁻¹) until the end of the pump run: mean total dose, 108.6 µg·kg⁻¹ (range 104–110). Duration of anaesthesia ranged from 260–345 minutes, while whole body perfusion varied from 46 to 128 minutes at 27°C. The mean time of response to command was 5.2 hours (range

FIGURE 1 Mean arterial pressure, wedge and right atrial pressure during the awake state, at seven points during anaesthesia and operation, and at three intervals in the postoperative period.

1.2–16) and mean time to extubation was 15.5 hours (range 9.5–20.5).

Other variables were calculated from measured data by standard formulas.¹³ MVO₂, obtained by multiplying CBF by the arterial-CS difference of oxygen content, represents the oxygen consumption of myocardium drained by the coronary sinus, essentially the left ventricle. MLE is the arterial-CS difference of lactate content, divided by the arterial content, expressed as per cent. Analysis of variance and t-tests were done, relating subsequent series back to the awake control state. Only differences greater than $p < 0.05$ are reported.

Results

In the Figures, means $\pm$ standard deviation are shown for the ten patients.

Figure 1. Mean arterial pressure (MAP) did not change on induction; it was different than control only once, a decrease before the surgical incision. Nitroprusside was given to decrease blood pressure just before perfusion to three patients, during perfusion to three and postoperatively to only two patients, in each instance after the appropriate study measurements were completed.

FIGURE 2 Heart rate and vascular resistances, pulmonary and systemic.

Wedge pressure decreased before the incision was below control levels prepump. Right atrial pressure fell below control levels by the time of sternotomy.

Figure 2. Heart rate did not change until it decreased before sternotomy; postpump it was faster than when awake, to a peak of 87 ± 19 min at three hours postoperatively.

Pulmonary vascular resistance showed no change before perfusion but was higher than control postpump, through three hours postoperatively. Systemic vascular resistance (SVR) increased significantly on induction and before sternotomy rose to 1725 ± 329 , from 1339 ± 179 before the incision. The postpump mean values were highest at 1819 ± 471 before protamine was given and 2053 ± 353 after chest closure.

Figure 3. Cardiac index decreased on induction and remained below the awake value, both before pump and postpump through one hour postoperatively. Stroke work index fell on induction and remained so throughout, as also did stroke volume index.

Figure 4. The ten awake, sedated patients had a mean CBF of 126 ± 60 ml·min⁻¹ which did not change on induction or later. On induction, the oxygen content of CS blood rose from 8.3 ± 0.5 to

FIGURE 3 Cardiac index and stroke work index.

9.1 ± 0.5 ml·dl⁻¹ and remained elevated until the surgical incision; it was again elevated postoperatively. Mean MVO_2 decreased from 9.9 ± 4.0 to 7.1 ± 3.0 ml·min⁻¹ by the time of intubation; before sternotomy it fell to 6.9 ± 4.1 ml·min⁻¹.

Figure 5. Arterial and CS lactate content did not change prepump, but both were elevated early postpump and after chest closure. Mean lactate

FIGURE 4 Coronary blood flow (CBF), coronary sinus oxygen content (CSO₂) and myocardial oxygen consumption (MVO₂).

FIGURE 5 Arterial and coronary sinus lactate contents and myocardial lactate extraction.

extraction by the heart in the awake state was a normal 32 per cent; the only significant change was a decrease to six per cent immediately after the pump run. Lactate production was evident from one myocardium after induction, from three each immediately postpump and at 24 hours postoperatively.

Discussion

Myocardial oxygenation during anaesthesia for cardiac operations has been an important issue since open-heart surgery began. It had not previously been possible to quantitate myocardial oxygenation in the operating room, due to inability to measure coronary blood flow and arterial-coronary sinus differences of oxygen and other energy substrates. Moffitt, McGoon *et al.* from 1965–72 studied myocardial energy metabolism in a series of patients with various cardiac lesions, during and after open-heart operations: aortic,¹⁴ mitral¹⁵ and double valve¹⁶ replacement, congenital lesions¹⁷ and in fatal postoperative cardiogenic shock.¹⁸ The only method available was a catheter, inserted by the surgeon into the coronary sinus after thoracotomy,

making possible arterial-coronary sinus sampling of energy substrates. Left coronary artery blood flow was known only during direct coronary perfusion; MVO_2 could then be calculated.

Subsequently, determination of CBF and MVO_2 became possible, before and after induction and maintenance with various anaesthetic techniques. The argon-washin method was developed by Rau¹⁹ and used by Sonntag *et al.*,⁹ while Ganz *et al.*¹¹ created the thermodilution coronary sinus catheter for measuring total CBF. The latter quantitates essentially left coronary inflow, can be placed in the awake patient; highly reproducible CBF measurements take less than a minute. Using it, Moffitt and co-workers have correlated the haemodynamic response to CBF, MVO_2 and MLE in patients having CABG, receiving morphine or halothane,²⁰ enflurane,²¹ isoflurane²² or nitrous oxide^{23,24} with oxygen. This communication adds high-dose fentanyl and oxygen to that series, using the same protocol.

The patients selected were like those for our previous studies, having coronary disease, needing CABG, but with adequate ventricular function. All were normotensive and kept on beta blockade until operation; all had regions of hypokinesis or akinesis but no ventricular aneurysm. None had valvular or pulmonary disease or recent myocardial infarction. An induction with fentanyl $75 \mu\text{g}\cdot\text{kg}^{-1}$, followed by a continuing infusion of $10 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{hour}^{-1}$ was chosen as appropriate pharmacokinetically.⁷

Circulatory responses

Induction with $75 \mu\text{g}\cdot\text{kg}^{-1}$ of fentanyl over a 12-minute period, caused no change in heart rate or MAP, the latter because a decrease in cardiac index was countered by a rise in SVR. This reduction in cardiac output, contrary to other reports,^{1,2} was in the presence of decreased atrial pressures. MAP later decreased before the surgical incision. During anaesthesia, prepump and postpump, mean MAP stayed at the awake level, with a reduced cardiac index persisting and a SVR higher than the awake valve. Another possible influence on haemodynamics was the approximately 5 mg of IV diazepam,²⁵ given to increase sedation before placing the catheters. Since over an hour elapsed before the awake studies, we conclude its influence to be minimal. In the well-beta blocked patients, pancuronium ($0.1 \text{ mg}\cdot\text{kg}^{-1}$) caused no rate increase.

However, as found by others, this high loading dose of fentanyl did not uniformly prevent a major pressure rise associated with sternal sawing: two of the ten patients needed nitroprusside after the posternotomy measurements. More critical was the fact that two additional patients, entered into the study under the same criteria, became severely hypertensive before sternotomy. Both were younger, more vigorous patients and had inadequate effect from the premedication. One had not been completely beta blocked but the other had a heart rate while awake of 46/min. One patient needed nitroprusside before sternotomy and one soon after. Nitroprusside was continued intermittently throughout, therefore eliminating them from the study. This unpredictable inability of high-dose fentanyl to maintain a hypodynamic circulation is similar to our experience with $1 \text{ mg} \cdot \text{kg}^{-1}$ of morphine, in patients also concluded to be not fully beta blocked.²⁰ Conversely, with halothane,²⁰ enflurane²¹ and isoflurane,²² each given alone with oxygen, a steady depression of the circulation is maintainable. These agents were given in varied concentrations, anticipated to obtund autonomic responses, and not at an unvarying rate as with fentanyl. One major difference with fentanyl was clear early postoperatively: patients given $110 \mu\text{g} \cdot \text{kg}^{-1}$ did not respond for an average of 5.2 hours; only two required treatment of hypertension. After enflurane-oxygen,²¹ patients responded in an hour and eight of ten then needed a vasodilator and sedation to control hypertension.

Coronary flow and myocardial oxygenation

The mean CBF in the ten sedated, awake patients of $126 \pm 60 \text{ ml} \cdot \text{min}^{-1}$ is typical in our experience. Our enflurane patients,²¹ studied concomitantly, had a mean CBF of $116 \pm 44 \text{ ml} \cdot \text{min}^{-1}$. This wide range of coronary flow in awake man with coronary disease has been our characteristic finding. While other factors such as body size and the coronary lesions may be involved, arterial pressure appears to be the major determinant of CBF. The five patients with the highest CBF when awake ($176 \text{ ml} \cdot \text{min}^{-1}$; range 115–252) had a mean MAP of 104 mmHg (range 76–135). The five patients with lowest awake CBF ($76 \text{ ml} \cdot \text{min}^{-1}$; range 62–96) had a mean MAP of 72 mmHg (range 74–101) at that time.

Another influence on the flow measured is the catheter position; we verify, by contrast medium, its place in mid-coronary sinus, i.e., not deeply

inserted into the great cardiac vein. The measurement error is small: in 12 awake patients, duplicate flows done three minutes apart were 113.8 ± 0.1 and $112.9 \pm 0.1 \text{ ml} \cdot \text{min}^{-1}$ (unpublished data).

Whereas in this fentanyl study, no significant changes in CBF were documented throughout, with enflurane²¹ we saw a decrease on induction ($p < 0.05$) paralleling a 35 per cent reduction in MAP. With fentanyl induction, no change in coronary resistance was documented.

Fentanyl induction, with ventilation maintained with oxygen, produced an increase in CS oxygen content, which lasted until the surgical incision. This was in the presence of reduced cardiac index and stroke work and volume indices. A CS oxygen content greater than when awake, reappeared by the time of chest closure and was still present in the extubated patient the next morning, along with still decreased stroke work, but normal cardiac index. Whether this is due to better oxygen supply from vein grafts or lower oxygen extraction cannot be determined from the information available. The same picture of diminished myocardial oxygen uptake was present prepump in the enflurane study,²¹ with reduced cardiac index and work.

As calculated from CBF and the arterial-CS difference of oxygen content, MVO_2 in these ten awake, sedated patients averaged $9.9 \pm 4 \text{ ml} \cdot \text{min}^{-1}$. This fits well with 10.2 ± 3.2 in ten enflurane patients,²¹ 9.8 ± 0.3 in six receiving morphine²⁰ and 10.1 ± 2.3 in six halothane patients.²⁰ In contrast, Sonntag⁹ reported, in nine awake patients with higher MAP (mean 103) and rate (mean 76), a MVO_2 of 11.5 ± 0.8 (SEM). In 21 unpremedicated patients with a mean MAP of 113 and mean rate of 75, Reiz *et al.*²⁶ found mean MVO_2 to be $14.9 \pm 1.2 \text{ ml} \cdot \text{min}^{-1}$. Our patients before induction had been maintained on beta blockade and were well-sedated, hence we found MVO_2 to be low normal. Our data fit with those of Tinker *et al.*,²⁷ who found that in pigs the efficiency of myocardial oxygen utilization is decreased with fentanyl, compared to halothane or enflurane and that this efficiency was increased by large doses of propranolol. Since CBF did not change, the narrowed difference of oxygen content produced the decreased calculated MVO_2 after intubation. The lowest MVO_2 (6.9 ± 4.9) before sternotomy, was probably influenced by a decline in body temperature. At three hours postoperatively, MVO_2 was highest (11.6 ± 6.2), in the presence of

the highest heart rate, plus increased cardiac index and body temperature compared to the previous state.

With aerobic energy metabolism, the myocardium extracts and uses more than 20 per cent of the arterial lactate presented to it. In anaerobic ATP formation, myocardial cells produce excess lactate, which appears in CS blood. Since lactate production from regional ischaemia could be hidden in the large CS pool of venous blood from oxygenated muscle, more lactate in CS than arterial blood must mean a large ischaemic area. The ten fentanyl patients had normal MLE ratios except at three of the study times. One myocardium produced lactate after induction and not again. Three myocardia had lactate production immediately postpump, which probably included washout from the cardioplegic period, though none of the enflurane group²¹ showed this. More puzzling was that three other myocardia showed lactate production the next morning, with normal cardiac index, CBF and ECG. Two of six of our morphine patients and one of 12 halothane patients had lactate production poststernotomy;²⁰ none in the enflurane study²¹ showed it at any time. We saw no adverse haemodynamic effects at these times when myocardia were producing lactate, or postoperatively in the one patient with ECG evidence of minor infarction.

Comparison with other evidence

Our study and findings must be compared with those of Sonntag *et al.*⁹ who found lactate production during sternotomy in seven of nine patients after a total fentanyl dose of $127 \mu\text{g}\cdot\text{kg}^{-1}$. They concluded that "large doses of fentanyl ... produce incomplete anesthesia and fail to protect the myocardium from ischemia ..." Several differences exist between the two studies which explain the considerable divergence of results. While the severity of cardiac illness of both patient groups seems equivalent, our patients were given heavier sedation: 15 mg of morphine in addition to the 10 mg of diazepam given their patients. Also we conclude that their patients probably did not have complete beta blockage, on 15 mg of pindolol/day, compared to our mean dose of 168 mg/day of propranolol. In support of this is their awake heart rate (mean 76) and MAP (mean 103) compared to 62 and 96 in our patients. Further, in their patients heart rate continued to climb from intubation through sternotomy,

peaking at 96/minute. Heart rate in our patients was unchanged on intubation and slowed by the time of sternotomy, to 54/minute. A third reason for their high incidence of anaerobic myocardial energy metabolism may have been their drug protocol. They performed intubation after etomidate and only $10 \mu\text{g}\cdot\text{kg}^{-1}$ of fentanyl, adding $90 \mu\text{g}\cdot\text{kg}^{-1}$ after intubation, plus additional fentanyl after pressure and rate increases. We gave $75 \mu\text{g}\cdot\text{kg}^{-1}$ before any autonomic stimulation and continued with a maintenance dose, before any further surgical stimulation. This produced more effective blunting of autonomic reflexes and preserved myocardial aerobic metabolism in our heavily sedated and beta blocked subjects. Ten of our original 12 patients had controlled depression of blood pressure and slow heart rate prepump, with no net appearance of myocardial venous lactate, vastly different from Sonntag's experience. We firmly believe that preventing emotional stress with its endogenous catecholamine elevation and reducing the heart's ability to respond to catecholamines, are the secrets to preserving myocardial oxygen balance.

We conclude, as have others, that high dose fentanyl (over $100 \mu\text{g}\cdot\text{kg}^{-1}$) does not assure complete obtunding of the hyperdynamic response to stimulation in patients with coronary disease and preserved ventricular function. Factors contributing to preserving a hypodynamic circulatory state and myocardial oxygenation, were heavy premedication, effective beta blockade continued until operation and a high loading dose of fentanyl plus its continued infusion.

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Résumé

Le fentanyl à dose moyenne de $109 \mu\text{g}\cdot\text{kg}^{-1}$ avec oxygène, a été employé comme agent d'anesthésie chez dix malades opérés pour pontage aorto-coronarien. Les mesures suivantes ont été prises avant, durant et après l'opération: pression artérielle moyenne (MAP), pression dans l'artère pulmonaire (PAP), index cardiaque (CI), flux sanguin du sinus coronaire (CBF), consommation d'oxygène par le myocarde (MVO_2) et extraction de lactate. A l'induction, l'index cardiaque et le travail du ventricule gauche ont diminué mais la fréquence cardiaque et la pression artérielle sont demeurées inchangées de même que la résistance systémique. La pression artérielle moyenne et la fréquence cardiaque sont demeurées au niveau pré-induction. Le CBF est demeuré inchangé comme la pression artérielle moyenne et les résistances coronaires. Le contenu en oxygène du sang veineux coronaire a augmenté à l'induction et s'est maintenu élevé jusqu'au moment de l'incision. De même ce contenu était au-dessus des niveaux de contrôle dans la période post-opératoire immédiate. La consommation d'oxygène par le myocarde était basse mais normale à l'état d'éveil et s'est maintenue ainsi. A quelques exceptions près, l'extraction de lactate est demeurée normale. Le fentanyl à hautes doses n'a pas contrôlé les réflexes du système nerveux autonome de façon uniforme. Une prémédication lourde, un blocage bêta-adrénergique adéquat et une dose élevée de fentanyl au départ suivie d'une infusion continue contribuent à garder la circulation dans un état hypodynamique et à assurer l'oxygénation myocardique.