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Hemodynamic Changes Prior to and After Sternotomy in Patients Anesthetized with High-dose Fentanyl

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High-dose fentanyl-oxygen anesthesia (50–100 $\mu\text{g}/\text{kg}$) produces little change in cardiovascular hemodynamic function.^{1,2} However, little information is available concerning the effects of painful stimuli, *e.g.*, sternotomy, on the hemodynamics of patients receiving fentanyl anesthesia for cardiac operations. An investigation of the hemodynamic responses to cardiac surgery during fentanyl anesthesia is described below.

METHODS

Following approval of this investigation by the Human Experimentation Committee, twelve patients scheduled for coronary artery bypass grafting were studied. Ten of the patients were taking propranolol (40–120 mg, po daily) and all took nitroglycerin occasionally for angina. All patients were premedicated with diazepam, 0.1–0.2 mg/kg orally, morphine sulfate, 0.1 mg/kg intramuscularly, and scopolamine, 0.4 mg intramuscularly 60 min prior to arriving in the operating room. The last dose of propranolol was given with the preoperative medication. Intravenous infusions were begun along with insertion of a catheter into a radial artery, and a Swan-Ganz catheter into the pulmonary artery via the right internal jugular vein.

After inhaling 100 per cent oxygen for ten min, fentanyl, 50 $\mu\text{g}/\text{kg}$ was given as a single intravenous bolus. When the patient lost consciousness as evidenced by inability to answer verbal commands, pancuronium, 0.15 mg/kg was given, the trachea intubated, and ventilation controlled to maintain a PaCO_2 of 35–40 torr.

Measurements of cardiovascular dynamics included cardiac output (CO), heart rate (HR), mean arterial pressure (MAP), pulmonary capillary wedge pressure (PCWP), and central venous pressure (CVP). Cardiac

output was measured utilizing the thermodilution technique and an Edwards #9520 computer. Cardiac index (CI), left ventricular stroke work index (LVSWI), and systemic vascular resistance (SVR) were calculated from standard formulas.

Cardiovascular dynamics were measured prior to induction of anesthesia (Period 1), five min after intubation of the trachea (Period 2), five min after sternotomy (Period 3), and five min after stabilization of cardiovascular variables using a vasodilator (Period 4).

All patients received vasodilator treatment, Period 4, for elevations in MAP following sternotomy. This consisted of either nitroglycerin, 32–128 $\mu\text{g}/\text{min}$ or sodium nitroprusside, 0.5–2.0 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$.

Data were analyzed for statistical significance utilizing Student's paired *t* test comparing each measurement with the awake measurements (Period 1).

RESULTS

Fentanyl, 50 $\mu\text{g}/\text{kg}$, Period 2, produced no significant changes in any of the measured cardiovascular variables from the awake state, Period 1 (table 1). Sternotomy, Period 3, resulted in significant increases in MAP and SVR ($P < 0.005$ and $P < 0.001$, respectively) while CO and CI decreased significantly ($P < 0.001$). HR, PCWP, CVP, and LVSWI remained unchanged.

Therapeutic vasodilatation, Period 4, resulted in a return of MAP, SVR, CO, and CI to near pre-induction values, although there did remain small statistical differences between Period 1 and Period 4 ($P < 0.005$). HR, PCWP, CVP, and LVSWI remained unchanged throughout the study (table 1).

No patient remembered any aspect of his or her operation, and the tracheas of all patients were extubated within 12 hours of operation.

DISCUSSION

Previous investigators have recommended the use of high-dose fentanyl anesthesia in patients with coronary artery disease because it produces very little alteration in cardiovascular dynamics.^{1,2} Our study confirmed that fentanyl induces no significant hemo-

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TABLE 1. Cardiovascular Responses During Fentanyl Anesthesia (Mean \pm SEM)

	Period			
	1	2	3	4
CO (l/min)	4.4 \pm 0.1	4.4 \pm 0.1	3.1 \pm 0.1†	4.3 \pm 0.1
HR (beats/min)	71 \pm 1	72 \pm 1	71 \pm 2	70 \pm 1
MAP (torr)	79 \pm 2	79 \pm 2	108 \pm 2†	81 \pm 2*
PCWP (torr)	12 \pm 1	12 \pm 1	13 \pm 1	13 \pm 1
CVP (torr)	8 \pm 1	8 \pm 1	8 \pm 1	8 \pm 1
CI (l/min/m ²)	2.44 \pm 0.07	2.44 \pm 0.07	1.73 \pm 0.06†	2.38 \pm 0.07*
LVSWI (g·m/m ²)	37 \pm 2	37 \pm 2	37 \pm 2	37 \pm 2
SVR (dyne·s/cm ²)	1281 \pm 51	1274 \pm 52	2574 \pm 54†	1373 \pm 43†

* $P < 0.005$.

† $P < 0.001$.

dynamic alterations prior to sternotomy. However, sternal splitting was found to increase MAP dramatically in the fentanyl-anesthetized patients. This hypertension was produced by a marked increase in SVR which resulted in a significant reduction in CO. This finding seems contrary to the results of Stanley *et al.*³ who reported no change in cardiovascular variables following surgical stimulation. However, it is not known whether their data included sternotomy; if sternotomy had not yet been performed, the two sets of data cannot be directly compared.

These findings are consistent with those of others who observed increased sympathetic responses under narcotic anesthesia. Conahan *et al.* noted that the occurrence of hypertension during cardiac surgery was associated primarily with morphine anesthesia.⁴ Arens *et al.* also noted a high incidence of hypertension in their series of patients anesthetized with morphine for coronary artery surgery.⁵ Both the sympathetic nervous system and the renin-angiotensin mechanism apparently contribute to the hypertension.^{6,7}

Although 50 μ g/kg of fentanyl was sufficient to induce loss of consciousness and prevent recall of any part of the surgical procedure in every patient, larger doses of fentanyl may be necessary to prevent the hypertension seen after sternotomy. Ablation of sternotomy-induced hypertension has not been entirely realized in patients receiving sulfentanyl, a synthetic narcotic 5–10 times as potent as fentanyl.⁸ This hypertension, if prolonged, may become deleterious to the patient. Butchart *et al.*⁹ established the importance of the prebypass inotropic state of the left ventricle. In their study, increased prebypass contractility induced with isoproterenol resulted in severe depression of postbypass ventricular function. In our study, the absence of an increased heart rate after surgical stimulation may reflect a certain degree of beta-adrenergic blockade present at the time of sternotomy.

Vasodilatation returned SVR, MAP, and CO to normal or near normal values indicating an effective mode of treatment. Kaplan and Jones,¹⁰ have shown that intraoperative treatment of hypertension with nitroglycerin or nitroprusside resulted in reduction of

myocardial oxygen demand and improvement of ST-segment elevation. Thus, in fentanyl-anesthetized patients having cardiac surgery, a sternotomy-induced hypertension should be anticipated which may quickly be treated by way of vasodilator therapy and thus minimize this idiopathic stress to an already compromised heart.

These patients did not develop bradycardia with fentanyl, which may have been due to the large dose of pancuronium given to prevent muscle rigidity. Pancuronium's ability to produce tachycardia may have negated an HR response to fentanyl.

In conclusion, these data reveal that sternotomy in patients anesthetized with fentanyl results in hemodynamic changes related to an increase in SVR. Vasodilatation proved to be a rapid and effective method of treatment. These results should be a consideration in contemplating anesthesia for patients undergoing coronary artery surgery.

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Treatment of Stress-induced Increases in Pulmonary Capillary Wedge Pressure Using Volatile Anesthetics

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Except for excessive or acute fluid administration intravascularly, a marked increase in pulmonary capillary wedge pressure (PCWP) or left atrial pressure usually indicates myocardial dysfunction.¹ Traditional treatment of such dysfunction commonly involves use of vasodilators or myocardial inotropes (*e.g.*, dopamine); myocardial depressants are usually considered inappropriate.² However, surgical stimuli may increase heart rate, systemic arterial and pulmonary capillary wedge pressures, and coronary and systemic vascular resistances. Depression of the ST segment, cardiac arrhythmias, and *pulsus alternans* may occur, perhaps due to myocardial dysfunction from an increased workload. Our study tests the possibility that the disadvantage of anesthetic-induced myocardial depression may be offset by the benefits, *i.e.*, decreases in arterial blood pressure, in peripheral and perhaps coronary vascular resistances, and in release of vasoactive substances.³⁻⁶

METHODS

From December 1978 to May 1979, 44 patients who underwent elective abdominal aortic reconstruction for either aneurysm or atherosclerosis of the aorta granted permission to be studied. Of these patients, 12 (50-88 years of age) had increased PCWP that exceeded the normal range when a surgical stimulus was initiated. These 12 patients constitute the study group of this report.

The preoperative electrocardiograms of nine patients had been normal. The other three patients had electro-

cardiograms that were compatible with left ventricular hypertrophy with strain. These three patients, and three of the other patients, were receiving diuretics for treatment of hypertension. One patient took propranolol, two patients received hydralazine, and five took potassium chloride. Diazepam or flurazepam was taken by 10 of the 12 patients. All patients received their antihypertensive medication on the morning of surgery, as well as small doses of diazepam and morphine. No patient had any diastolic blood pressure reading greater than 95 torr prior to their scheduled surgery. None of the patients had symptoms suggestive of prior myocardial ischemia.

Anesthesia was induced in all patients with a small dose (1.0-2.5 mg/kg) of thiopental and halothane or enflurane in 60 per cent nitrous oxide, 40 per cent oxygen. Paralysis was induced using 0.08 mg/kg pancuronium given at least 18 min prior to our first measurement period. After insertion of an endotracheal tube, ventilation was controlled to maintain end-tidal CO₂ in the 30-40 torr range. Before incision, anesthetic dose was manipulated to keep systemic systolic blood pressure 10-20 per cent below the lowest preoperative systolic blood pressure as long as PCWP remained within the normal range. (If it had not, sodium nitroprusside would have been administered. However, in every case, PCWP remained normal.)

Inspired and end-tidal anesthetic concentrations of anesthetic and respiratory gases were monitored continuously using mass spectrometry. Direct systemic arterial, pulmonary arterial, and central venous pressures and a modified chest lead V electrocardiogram were transduced to a Grass® Model 5 recorder. Cardiac output was measured in triplicate using an Edwards® thermodilution cardiac output monitor. Cardiac output and PCWP were measured at end expiration while the ventilator was disconnected for 10 s.

Each increase in systolic blood pressure and PCWP, with or without ST-segment depression, was treated only by increasing the dose of volatile anesthetic administered. (If abnormalities did not improve, sodium nitroprusside would have been administered. However, in every case abnormalities improved.) Cardiovascular variables were measured, and blood samples were obtained for determination of acid-base status prior to incision and at least

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