HORMONAL RESPONSES TO CARDIAC SURGERY:
EFFECTS OF SUFENTANIL, SOMATOSTATIN AND
GANGLION BLOCK

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AND S. R. BLOOM

SUMMARY
We have investigated the effect of the addition of somatostatin and trimetaphan to sufentanil
20 µg kg⁻¹ on the hormonal responses to cardiac surgery and compared the changes with a control
group receiving sufentanil and sodium nitroprusside. Eighteen patients undergoing elective
valve replacement surgery were studied. Patients who received somatostatin and trimetaphan in
addition to sufentanil had significantly smaller serum growth hormone and plasma glucagon
concentrations compared with those who received sufentanil and sodium nitroprusside. The
cortisol response to surgery was inhibited in both groups. There were no significant differences in
catecholamine concentrations between the two groups. There was no effect of the additional
inhibition of glucagon and growth hormone on circulating concentrations of glucose and lactate,
but plasma concentrations of non-esterified fatty acids increased significantly. Thus the addition of
somatostatin increased the suppression of the hormonal response to cardiac surgery by
sufentanil.

KEY WORDS

High dose fentanyl anaesthesia (50 µg kg⁻¹)
prevents the hormone response to pelvic surgery
[1] and doses of 100 µg kg⁻¹ suppress the response to
upper abdominal surgery [2]. However, there is
a considerable risk of respiratory depression in the
postoperative period, and high dose opioid tech-
niques are more suitable for cardiac anaesthesia.

During cardiac surgery, high dose fentanyl suppresses the pituitary hormone response until the
onset of cardiopulmonary bypass (CPB) [3].

The effect of a newer opioid, sufentanil, has
been studied extensively [4–10] and reviewed by
Desborough and Hall [11]. Sufentanil has a
significantly higher affinity for µ-opioid receptor
sites and slower dissociation from the receptor
than fentanyl [12]. It may be a more effective
inhibitor of secretion of pituitary hormones, for
example adrenocorticotropic hormone (ACTH) and β endorphin, even during CPB [9]. In some
studies, sufentanil has been shown also to prevent
increases in growth hormone (GH) concentration
during CPB [4, 5], but this is not a consistent
finding [9]. The catecholamine response to CPB
has not yet been blocked, even with doses of
sufentanil as great as 25 µg kg⁻¹ [8].

It appears that a high dose opioid technique
alone is unlikely to prevent all the hormonal
changes associated with CPB, and we considered
that a combination of opioid and other specific
hormonal blocking agents might be more effective.
Octreotide is a long acting (6–8 h) synthetic
octapeptide analogue of naturally occurring
somatostatin which inhibits the secretion of
intestinal peptides and GH [13]. We have
investigated, therefore, the catabolic hormonal
response to cardiac surgery, using sufentanil to
inhibit ACTH and hence cortisol secretion,
octreotide (hereafter called somatostatin) to sup-
press GH and glucagon secretion, and ganglion
block with trimetaphan to inhibit catecholamine release. This was compared with a control group receiving sufentanil and sodium nitroprusside (SNP) to control arterial pressure.

PATIENTS AND METHODS

We studied 18 patients admitted for elective aortic or mitral valve replacement. They had no history of endocrine or metabolic disease. All patients received sufentanil 20 \( \mu \)g kg\(^{-1} \) and they were allocated randomly either to receive somatostatin 100 \( \mu \)g subcutaneously (s.c.) at induction of anaesthesia and trimetaphan camsylate to achieve ganglion block (somatostatin group), or to receive SNP to control arterial pressure (control group). The study was approved by the Hospital Ethics Committee and patients gave written informed consent.

Patients were premedicated with papaveretum 15–20 mg, hyoscine 0.3–0.4 mg and droperidol 2.5–5.0 mg i.m. 90 min before arrival in the anaesthetic room. A peripheral vein was cannulated for infusion of drugs and fluids and a radial artery cannula inserted for direct measurement of arterial pressure and collection of blood samples. Anaesthesia was induced with a dose of thiopentone sufficient to obtund the eyelash reflex and the trachea intubated after administration of pancuronium 0.12 mg kg\(^{-1} \). The lungs were ventilated with 50% nitrous oxide in oxygen. Sufentanil 10 \( \mu \)g kg\(^{-1} \) was given over 5 min after the pancuronium; patients in the somatostatin group received somatostatin 100 \( \mu \)g s.c. at this time. A further sufentanil 5 \( \mu \)g kg\(^{-1} \) was given 5 min before sternotomy and 5 min before CPB. Three right internal jugular cannulae, a urinary catheter and an oesophageal temperature probe were inserted. Ventilation was adjusted to maintain \( P_{\text{a}} \text{CO}_2 \) at 4.5–5.0 kPa throughout the study. Sodium chloride 150 mmol litre\(^{-1} \) was infused at 6 ml kg\(^{-1} \) h\(^{-1} \) before CPB. An attempt was made to maintain mean arterial pressure less than 75 mm Hg in the control group with an infusion of SNP 1 mg ml\(^{-1} \) and in the somatostatin group with an infusion of trimetaphan camsylate 5 mg ml\(^{-1} \).

Heparin 3 mg kg\(^{-1} \) was administered before cannulation of the aorta and right atrium. The heart–lung machine was primed with 2 litre of Hartmann’s solution and sodium bicarbonate 25 mmol. CPB was undertaken using non-pulsatile flow with a bubble oxygenator at a pump flow of 2.2–2.4 litre min\(^{-1} \) m\(^{-2} \). Patients were cooled (mean temperature 28.3 °C after 30 min) during CPB and myocardial preservation was undertaken with 1–2 litre of ice cold cardioplegia solution into the root of the aorta. Protamine 3 mg kg\(^{-1} \) was administered to restore normal coagulation after CPB. Blood was transfused after CPB according to cardiovascular status and blood loss.

After operation, patients were transferred to the intensive care unit and ventilation of the lungs was continued. Papaveretum 5 mg and midazolam 2.5 mg were given i.v. as required for analgesia and sedation. Glucose solution 4% was infused i.v. at 1 ml kg\(^{-1} \) h\(^{-1} \).

Blood samples were collected and mean arterial pressure recorded at the times shown in table I. All samples were analysed in duplicate for glucose, lactate and non-esterified fatty acids (NEFA) concentrations and PCV by methods described previously [1]. Serum GH [14], insulin [15], cortisol [16] and plasma glucagon [17] concentrations were determined by radioimmunoassay in samples taken before induction of anaesthesia, after 30 min of surgery, after 30 and 60 min of CPB, and 2 h and 6 h after the end of CPB. Plasma concentrations of adrenaline and noradrenaline were analysed in these samples by high pressure liquid chromatography with electrochemical detection [18]. All samples were analysed in a single assay; the intra-assay coefficients of variation were 5.0% for GH, 10.3% for insulin, 4.0% for cortisol, 7.9% for glucagon, 8.2% for adrenaline and 6.8% for noradrenaline. The investigators were unaware of sample identities at the time of analysis.

Results are presented as mean (SEM), or median (range) for GH data which were not normally distributed. Within-group comparison of metabo-

<table>
<thead>
<tr>
<th>Sample</th>
<th>Time of collection</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Before induction of anaesthesia</td>
</tr>
<tr>
<td>2</td>
<td>15 min after induction of anaesthesia</td>
</tr>
<tr>
<td>3</td>
<td>After 30 min of surgery</td>
</tr>
<tr>
<td>4</td>
<td>After 30 min of CPB</td>
</tr>
<tr>
<td>5</td>
<td>After 60 min of CPB</td>
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<tr>
<td>6</td>
<td>1 h after CPB</td>
</tr>
<tr>
<td>7</td>
<td>2 h after CPB</td>
</tr>
<tr>
<td>8</td>
<td>4 h after CPB</td>
</tr>
<tr>
<td>9</td>
<td>6 h after CPB</td>
</tr>
<tr>
<td>10</td>
<td>24 h after CPB</td>
</tr>
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</table>
Table II. Details of patients studied mean (SEM)

<table>
<thead>
<tr>
<th></th>
<th>Control group (n = 9)</th>
<th>Somatostatin group (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>65.1 (3.2)</td>
<td>57.5 (3.8)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>66.8 (3.7)</td>
<td>70.0 (4.2)</td>
</tr>
<tr>
<td>Sex (M:F)</td>
<td>6:3</td>
<td>7:2</td>
</tr>
<tr>
<td>Aortic valve replacement</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Mitral valve replacement</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Aortic and mitral valve replacement</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>Duration of surgery (min)</td>
<td>165 (9)</td>
<td>188 (10)</td>
</tr>
<tr>
<td>Duration of CPB (min)</td>
<td>71 (6)</td>
<td>89 (8)</td>
</tr>
<tr>
<td>Preoperative medication</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diuretics</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Digoxin</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>β-Blockers</td>
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<td>1</td>
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<tr>
<td>Calcium channel blockers</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Nitrates</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

Results and hormones was undertaken by two-way analysis of variance and Dunnett's test, and between-group differences by one-way analysis of variance. Data not distributed normally were analysed by Mann-Whitney U test and Wilcoxon rank test.

Results

There was no significant difference between the groups with respect to age, sex, weight, preoperative medication and duration of surgery or CPB (table II).

Serum GH (fig. 1)

In the control group, GH increased significantly from a median value of < 1 to 2.1 µg litre⁻¹ after 60 min of CPB (P < 0.01) and increased further to 9.2 µg litre⁻¹ (P < 0.01) 6 h after CPB. In the somatostatin group, however, GH concentrations remained low during CPB and increased significantly only 2 h after CPB, from < 1 to 1.3 µg litre⁻¹ (P < 0.05), with a further increase 6 h after CPB to 3.1 µg litre⁻¹ (P < 0.01). The concentration of GH was significantly less in the somatostatin group compared with the control group after 60 min of CPB (P < 0.05).

Serum insulin (fig. 2)

Insulin concentrations declined from 13.9 to 7.5 µg litre⁻¹ (P < 0.01) in the control group and from 14.7 to 6.3 µg litre⁻¹ (P < 0.01) in the somatostatin group after 30 min of surgery. There was a further profound decrease after 30 min of CPB, to 1.5 µg litre⁻¹ (P < 0.01) in the control group and to 1.1 µg litre⁻¹ (P < 0.01) in the somatostatin group. Insulin concentrations then increased after 60 min of CPB, and 2 h after CPB had returned to pre-induction values. There was
no significant difference between the two groups of patients.

**Plasma glucagon (fig. 3)**

In the control group, plasma glucagon decreased from 18.1 to 14.3 pmol litre\(^{-1}\) after 30 min of surgery, but this change was not significant. Glucagon increased with the onset of CPB and reached 40.5 pmol litre\(^{-1}\) \((P < 0.01)\) 6 h after CPB. In the somatostatin group there was a significant decrease in plasma glucagon from 18.5 to 9.0 pmol litre\(^{-1}\) \((P < 0.05)\) after 30 min of surgery. During CPB, glucagon increased more slowly than in the control group, reaching 29.9 pmol litre\(^{-1}\) \((P < 0.01)\) 6 h after CPB. Plasma glucagon concentration was significantly smaller in the somatostatin group compared with the control group after 30 min of surgery \((P < 0.05)\), after 30 and 60 min of CPB \((P < 0.01)\) and at 2 and 6 h after CPB \((P < 0.05)\).

**Serum cortisol (fig. 4)**

In both groups of patients, serum concentrations of cortisol decreased during surgery before CPB, from 403 to 234 nmol litre\(^{-1}\) in the control group and from 309 to 223 nmol litre\(^{-1}\) in the somatostatin group, although this change was not significant. Cortisol concentrations remained low during CPB in both groups. They increased slowly after CPB, to 978 \((P < 0.01)\) and 753 nmol litre\(^{-1}\) \((P < 0.01)\) in the control and somatostatin groups, respectively, after 6 h. There was no significant difference between the two groups.

**Plasma adrenaline and noradrenaline (fig. 5)**

Plasma adrenaline concentrations in the control group increased significantly from 0.35 to 1.32 nmol litre\(^{-1}\) after 60 min of CPB \((P < 0.01)\) and this increase was maintained until 6 h after CPB. The increase in plasma adrenaline from 0.29 to 0.87 nmol litre\(^{-1}\) after 60 min of CPB in the somatostatin group did not reach statistical significance; however, 2 h after CPB, adrenaline concentrations increased further, to 1.39 nmol litre\(^{-1}\) \((P < 0.01)\). Although adrenaline concentrations were lower in the somatostatin group compared with the control group, there was no
TABLE III. Mean (SEM) values of metabolites, PCV and mean arterial pressure. Significance of difference from sample 1 (pre-induction): *P < 0.05; **P < 0.01. Significant difference from control group: †P < 0.05; ‡P < 0.01. For key to samples, see table I.

<table>
<thead>
<tr>
<th>Sample No:</th>
<th>Glucose (mmol litre(^{-1}))</th>
<th>Lactate (mmol litre(^{-1}))</th>
<th>NEFA (mmol litre(^{-1}))</th>
<th>PCV (%)</th>
<th>Mean arterial pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Somatostatin</td>
<td>Control</td>
<td>Somatostatin</td>
<td>Control</td>
</tr>
<tr>
<td>1</td>
<td>4.41 (0.31)</td>
<td>3.71 (0.20)†</td>
<td>0.83 (0.10)</td>
<td>0.84 (0.05)</td>
<td>1.05 (0.18)</td>
</tr>
<tr>
<td>2</td>
<td>4.21 (0.32)</td>
<td>3.50 (0.15)†</td>
<td>0.87 (0.13)</td>
<td>1.02 (0.09)</td>
<td>1.04 (0.20)</td>
</tr>
<tr>
<td>3</td>
<td>4.27 (0.17)</td>
<td>3.51 (0.17)†</td>
<td>1.01 (0.24)</td>
<td>0.92 (0.08)</td>
<td>1.00 (0.12)</td>
</tr>
<tr>
<td>4</td>
<td>5.46 (0.33)</td>
<td>4.68 (0.37)</td>
<td>1.84 (0.24)**</td>
<td>2.28 (0.24)**</td>
<td>1.56 (0.20)*</td>
</tr>
<tr>
<td>5</td>
<td>6.72 (0.36)*</td>
<td>6.28 (0.71)</td>
<td>1.94 (0.24)**</td>
<td>2.62 (0.31)**</td>
<td>1.72 (0.28)**</td>
</tr>
<tr>
<td>6</td>
<td>7.09 (0.58)*</td>
<td>6.32 (0.60)</td>
<td>1.05 (0.18)</td>
<td>0.96 (0.17)</td>
<td>22.5 (1.2)†</td>
</tr>
<tr>
<td>7</td>
<td>7.42 (0.99)**</td>
<td>7.86 (0.81)**</td>
<td>1.04 (0.20)</td>
<td>0.96 (0.13)</td>
<td>2.27 (0.38)‡</td>
</tr>
<tr>
<td>8</td>
<td>8.21 (0.77)**</td>
<td>8.02 (1.04)**</td>
<td>1.00 (0.12)</td>
<td>1.28 (0.19)</td>
<td>2.27 (0.38)‡</td>
</tr>
<tr>
<td>9</td>
<td>8.41 (0.57)**</td>
<td>9.51 (1.75)**</td>
<td>1.56 (0.20)*</td>
<td>3.11 (0.52)**</td>
<td>25.5 (0.9)†</td>
</tr>
<tr>
<td>10</td>
<td>6.58 (0.23)*</td>
<td>8.49 (0.10)†</td>
<td>1.72 (0.28)**</td>
<td>3.06 (0.53)**</td>
<td>24.5 (1.1)**</td>
</tr>
</tbody>
</table>

significant difference between the two groups throughout the study.

In the control group, noradrenaline concentrations increased from 1.36 to 3.34 nmol litre\(^{-1}\) after 60 min of CPB (P < 0.01). In the somatostatin group, the increase in noradrenaline from 1.15 to 2.62 nmol litre\(^{-1}\) after 60 min of CPB was not statistically significant, but noradrenaline increased further, to 3.57 nmol litre\(^{-1}\), 2 h after CPB (P < 0.01). There was no significant difference between the two groups.

**Metabolites** (table III)

**Blood glucose.** Similar changes in blood glucose concentration were seen in the two groups. In the control group, blood glucose concentration increased significantly, from 4.41 to 6.72 mmol litre\(^{-1}\), after 60 min of CPB (P < 0.05) and continued to increase up to 9.41 mmol litre\(^{-1}\) 4 h after CPB (P < 0.01). In the somatostatin group, blood glucose did not increase significantly until 2 h after CPB (7.86 mmol litre\(^{-1}\); P < 0.01) and was maximal 6 h after surgery (9.51 mmol litre\(^{-1}\); P < 0.01). In the somatostatin group, blood glucose concentration was significantly lower than in the control group in all three samples up to 30 min of surgery, and was significantly greater than the control group 24 h after CPB.

**Blood lactate.** There was no change in blood lactate concentration before CPB. After 30 min of CPB, lactate increased significantly, from 0.83 to
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1.84 mmol litre\(^{-1}\) \((P < 0.01)\) in the control group and from 0.84 to 2.28 mmol litre\(^{-1}\) \((P < 0.01)\) in the somatostatin group. The increase in blood lactate was maintained for 24 h after CPB in both groups. These was no significant difference in blood lactate between the two groups.

**Plasma non-esterified fatty acids (NEFA).** NEFA concentrations increased significantly in both groups of patients during CPB, from 1.05 to 1.56 mmol litre\(^{-1}\) \((P < 0.05)\) in the control group and from 0.96 to 3.11 mmol litre\(^{-1}\) \((P < 0.01)\) in the somatostatin group after 30 min of CPB. One hour after CPB, NEFA concentrations returned to pre-induction values. NEFA concentrations were significantly greater in the somatostatin group compared with the control group during CPB and 1 h after CPB.

**PCV (table III)**

In both groups of patients, PCV decreased significantly with the onset of CPB, from 39.4\% to 22.5 \% \((P < 0.01)\) in the control group and from 37.8\% to 25.5 \% \((P < 0.01)\) in the somatostatin group after 30 min of CPB. After operation, PCV increased slowly, reaching 32.3 \% \((P < 0.01)\) in the control group and 33.4 \% \((P < 0.05)\) in the somatostatin group 6 h after CPB. There was no significant difference between the two groups of patients.

**Mean arterial pressure (table III)**

Mean arterial pressure decreased in both groups after induction of anaesthesia, during surgery and with CPB. In the control group it decreased from 82 mm Hg to 60 mm Hg \((P < 0.05)\) and in the somatostatin group from 79 mm Hg to 55 mm Hg \((P < 0.05)\) after 60 min of CPB. One hour after CPB, mean arterial pressure was 71 mm Hg in the control group and 73 mm Hg in the somatostatin group. There was no significant difference between the two groups of patients, and arterial pressure was greater than the target value of 75 mm Hg on only two occasions after operation.

**Vasodilator therapy**

In the control group, the doses of SNP given during and after operation were 11.6 (2.5) mg and 56.5 (20.2) mg, respectively. In the somatostatin group, the doses of trimetaphan camsylate given during and after operation were 53.3 (11.4) mg and 132.6 (97.5) mg, respectively.

DISCUSSION

The combination of sufentanil, somatostatin and trimetaphan prevented the cortisol, GH and glucagon responses to cardiac surgery, and decreased plasma catecholamine concentrations, although this change was not statistically significant. Haemodilution occurs on CPB and may result in transient decreases in circulating concentrations of hormones and metabolites. However, the changes in PCV were comparable in the two groups (table III), showing that the addition of somatostatin increased the suppression of catabolic hormone secretion compared with a technique using opioids alone.

GH concentrations remained at basal values during CPB in patients who received somatostatin, with a significant difference from control patients after 60 min of CPB. Somatostatin is known to inhibit GH secretion in normal volunteers, although effects are variable, depending on the time of administration and food intake [13]. The increase in GH during CPB in the control group, although significant, was not as large as we expected from a previous study in which sufentanil 10 \(\mu\)g kg\(^{-1}\) or 20 \(\mu\)g kg\(^{-1}\) was given in a single dose following induction of anaesthesia with thiopentone and pancuronium [9]. In the present study, sufentanil was administered in divided doses, after induction of anaesthesia and before sternotomy and onset of CPB, which may have prevented larger increases in GH. Previous work using sufentanil in similar divided doses also showed no change in GH with CPB [5], as did a study with sufentanil given in incremental doses throughout surgery to a total dose of 13.1 \(\mu\)g kg\(^{-1}\) [4].

The significant decrease in plasma concentrations of glucagon in the patients who received somatostatin is in keeping with the inhibition of glucagon secretion for up to 6 h which occurs in normal volunteers given an s.c. injection of somatostatin 50 \(\mu\)g [13]. The decrease in insulin concentrations after 30 min of CPB followed by an increase after 60 min of CPB was similar to results found in a previous study [9]. The increase in insulin secretion occurred while there was no change in PCV and an increase in blood glucose of only 1.2–1.6 mmol litre\(^{-1}\), and could be explained partly by the effect of changes in temperature with cooling and rewarming during CPB. Although somatostatin is known to suppress insulin secretion in normal individuals [13], the injection of
somatostatin had no additional inhibitory effects in the present study. This implies that insulin was already suppressed maximally during surgery and CPB. The inability of somatostatin to inhibit insulin secretion further during cardiac surgery is fortuitous, as insulin is the key anabolic hormone [19].

In both groups of patients sufentanil 20 µg kg⁻¹ prevented any change in cortisol concentrations until 6 h after the end of CPB. This confirms the results of our previous study [9] and is in keeping with the observation that ACTH and cortisol secretion are suppressed by high dose opioid anaesthesia during cardiac surgery [11].

Circulating concentrations of catecholamines were lower in patients receiving trimetaphan, but surprisingly this did not reach statistical significance. Previous work suggested that trimetaphan effectively prevented release of catecholamines during CPB [20]. However, these investigators studied patients having coronary artery surgery who received intermittent fentanyl to a total dose of only 35 µg kg⁻¹, and either SNP or trimetaphan to control mean arterial pressure. Circulating concentrations of catecholamines during CPB were greater than in the present study, even in the patients given trimetaphan, and this may reflect the low dose of opioid used. In the present study, pre-bypass concentrations of catecholamines were comparable to those described recently in patients receiving sufentanil 17.5 µg kg⁻¹ for coronary artery surgery [10]. Sufentanil alone, in doses up to 25 µg kg⁻¹, has been found to be insufficient to prevent increases in plasma adrenaline and noradrenaline concentrations during CPB [8].

It is probable that the major stimulus for hyperglycaemia during CPB is the increase in circulating catecholamine concentrations. It has been shown in general surgery that glucose concentrations are mirrored by changes in concentrations of adrenaline and noradrenaline [21], and that blood glucose is sensitive to increases in plasma adrenaline [22]. Blood glucose concentrations were similar during CPB in both groups in the present study, supporting the primary role of the sympathetic nervous system in mobilizing glucose, and suggesting that glucagon and GH play a less important part than catecholamines in the regulation of circulating concentrations of glucose during surgery.

The highly significant difference in NEFA concentrations between groups during CPB was an interesting finding. Subcutaneous injection of somatostatin in normal man is associated with increases in concentrations of glycerol and 3-hydroxybutyrate, probably because of suppression of insulin secretion stimulating lipolysis [13]. However, insulin concentrations were similar in the two groups of patients in the present study. It is possible that a direct effect of somatostatin on lipolysis, lipogenesis, or both, or an alteration in the balance of anabolic and catabolic hormones during CPB may have been responsible for our observations.

In conclusion, the addition of somatostatin to sufentanil increased the suppression of the hormonal response to surgery. However, catecholamine secretion appears to be refractory to ganglion block during CPB and other methods of suppressing catecholamine response need to be investigated. Until the catabolic hormonal response to cardiac surgery can be totally prevented, attempts to establish a relationship between anaesthetic technique, hormone secretion and postoperative morbidity are premature [23].

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REFERENCES


