Epidural analgesia with bupivacaine does not improve splanchnic tissue perfusion after aortic reconstruction surgery†

O. VÄISÄNEN, I. PARVIAINEN, E. RUOKONEN, M. HIPPELÄINEN, E. BERG, H. HENDOLIN AND J. TAKALA

Summary

Inadequate splanchnic tissue perfusion is relatively common during and after aortic surgery. We hypothesized that vasodilation caused by thoracic epidural analgesia improves splanchnic blood flow and tissue perfusion after aortic surgery. In this prospective, randomized, controlled study, we studied 20 patients undergoing elective aortic-femoral or aortic-iliac reconstructive surgery. Gastric and sigmoid colon mucosal $P_{CO_2}$ and pH were measured during surgery. An epidural bolus of bupivacaine 40 mg was followed by infusion of 15 mg h$^{-1}$ was started after operation in 10 patients. After operation, splanchnic blood flow and gastric and sigmoid colon mucosal $P_{CO_2}$ and pH were measured before and 2 h after the start of epidural analgesia. During surgery, the gastric mucosal–arterial $P_{CO_2}$ difference remained stable, whereas the sigmoid mucosal–arterial $P_{CO_2}$ difference increased during aortic clamping but returned to pre-clamping values after declamping. After operation, epidural analgesia had no effect on gastric or sigmoid mucosal–arterial $P_{CO_2}$ differences or on splanchnic blood flow. (Br. J. Anaesth. 1998; 81: 893–898)

Keywords: analgesic techniques, epidural; surgery, vascular; gastrointestinal tract, blood flow; anaesthetics local, bupivacaine

The incidence of bowel ischaemia diagnosed by sigmoidoscopy varies between 1.1 and 10% after reconstructive surgery of the abdominal aorta: this is increased at least two-fold after aortic surgery for ruptured aortic aneurysm. Bowel ischaemia may result from interruption of the blood supply caused by surgery, reduced systemic blood flow, regional perfusion abnormalities or a combination of these.

Gastric mucosal acidosis has been shown to be a sign of poor prognosis in critically ill patients and is a predictor of poor outcome after aortic surgery for ruptured abdominal aortic aneurysm, suggesting that inadequate tissue perfusion is related to the development of organ dysfunction. In aortic surgery, sigmoid colon mucosal tonometry may reveal ischaemia caused by decreased cardiac output and arterial pressure, in addition to ischaemia caused by local vascular occlusion. Several studies have shown that sigmoid mucosal acidosis in aortic surgery is associated with poor outcome. In aortic surgery, low sigmoid mucosal pH is associated with high endotoxin concentrations and remote organ dysfunction.

Prevention of gastric mucosal acidosis has been suggested to improve prognosis in critically ill patients. Peri- and postoperative supranormal systemic oxygen delivery ($DO_2$) has improved outcome in selected groups of high-risk surgical patients. Although increased systemic $DO_2$ may improve the perfusion of the gut, interventions to increase whole body blood flow may worsen the gastric mucosal acidosis. An attractive alternative to manipulate visceral perfusion is epidural block, which is used widely in postoperative pain management after aortic surgery. Sympathetic block after thoracic epidural anaesthesia should cause mesenteric vasodilatation and increase intestinal motility, and therefore may be beneficial for gut perfusion after aortic surgery.

We hypothesized that epidural analgesia improves splanchnic blood flow and tissue perfusion after abdominal aortic surgery. To test this hypothesis, we measured the effect of epidural analgesia on splanchnic blood flow and tissue perfusion after operation in patients undergoing elective aortic reconstructive surgery.

Patients and methods

The study was approved by the Hospital Ethics Committee and written informed consent was obtained from each patient. Gastric and sigmoid mucosal pH was monitored in the peri- and postoperative periods in 20 patients undergoing elective abdominal aortic surgery because of aorto-iliac occlusive disease or abdominal aortic aneurysm. There were no exclusion criteria. To test the effect of epidural analgesia on splanchnic blood flow, patients were allocated randomly to one of two groups: 10 patients received postoperative epidural analgesia with bupivacaine after baseline measurement of hepatosplanchnic blood flow, and the remaining 10 patients served as controls. Clinical data of the patients are shown in table 1.

Before induction of general anaesthesia, an epidural catheter was inserted in each patient, except for one patient in the control group who received chronic anticoagulant therapy. The epidural catheter was used for postoperative analgesia in

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these 19 patients after completion of the study. The epidural catheter was inserted at the T12–L1 interspace using a standard loss of resistance technique and the catheter was inserted 5 cm inside the epidural space. A test dose of 50 mg of 1% lidocaine with epinephrine (adrenaline) 5 μg kg⁻¹ was injected through the catheter. General anaesthesia was induced with fentanyl 20 μg kg⁻¹ and a mixture of alcuronium 0.125 mg kg⁻¹, pancuronium 0.075 mg kg⁻¹ and midazolam 0.07 mg kg⁻¹. Anaesthesia was maintained with continuous infusion of fentanyl 0.07 μg kg⁻¹ min⁻¹, alcuronium 1 μg kg⁻¹ min⁻¹ and midazolam 0.6 μg kg⁻¹ min⁻¹, and was supplemented with thiopental (thiopentone) and isoflurane. Monitoring included pulmonary artery catheterization and we aimed to maintain cardiac index >2.0 litre min⁻¹ m⁻² by infusing crystalloids, colloids and blood. None of the patients received inotropic agents. During the surgery, sodium nitroprusside was used to treat systemic hypertension when systolic arterial pressure exceeded 160 mm Hg, despite supplementation of anaesthesia with thiopental or isoflurane. After operation, sodium nitroprusside was also used to treat hypertension when systolic arterial pressure exceeded 160 mm Hg. The arterial pressure limit was defined by the surgeon in charge of the operation. One patient received nitroglycerine in the perioperative period for a short time because of ST-elevation in the ECG, but after operation there were no signs of myocardial injury and recovery was uneventful. The aorta was clamped above the renal arteries in one patient and below the renal arteries in the remaining 19 patients. Duration of aortic clamp was 94 ± 35 min. In three patients (two in the control group and one in the epidural group) the aorta was occluded only partially. No reimplantations of the inferior mesenteric artery were performed.

**TONOMETRY**

To assess mucosal perfusion, both gastric mucosal PCO₂ and pH, and sigmoid mucosal PCO₂ and pH were measured in the peri- and postoperative periods by gastric and sigmoid tonometers (Tonomitor, Tonometrics, Worcester, MA, USA). Equilibration time was 30 min during aortic clamping and 60–90 min for other samples.²⁰ Gastric and sigmoid mucosal pH was calculated using a modification of the Henderson–Hasselbach equation. Intramucosal–arterial PCO₂ difference was calculated as gastric/sigmoid intramucosal PCO₂–arterial PCO₂, and arterial–intramucosal pH difference as arterial pH–gastric/sigmoid intramucosal pH. The patients in the epidural group and one patient in the control group received a H₂ receptor antagonist at induction of anaesthesia because of a previous history of gastric ulcer. In critically ill patients, the use of H₂ receptor antagonists has not been demonstrated to influence gastric mucosal pH values or to improve the reproducibility of determination of gastric mucosal pH.¹⁷¹⁸ The gastric tonometer was inserted after induction of anaesthesia, and the correct position was verified by the surgeon. The sigmoid tonometer was inserted into the rectum and manipulated to the mid-sigmoid position at laparotomy. The correct position of the tonometers was confirmed again just before closure of the abdomen, and for the gastric tonometer also by chest x-ray obtained in the intensive care unit.

Systemic haemodynamic variables, arterial and mixed venous blood-gas tensions and oxygen saturation, and haemoglobin and plasma lactate concentrations were measured before induction of anaesthesia, before aortic occlusion, during aortic occlusion and at the end of surgery. Plasma lactate concentrations were measured enzymatically (Stat Plus 2300, Yellow Springs Instrument Co., OH, USA). Blood-gas tensions and PCO₂ from the tonometers were measured with the same blood-gas analyser (ABL 500 Blood Gas System, Radiometer A/S, Copenhagen, Denmark) and corrected for actual body temperature. Cardiac output was measured by thermodilution in triplicate using saline 10 ml at room temperature. During operation, systemic oxygen consumption (VO₂) was calculated according to Fick’s principle. Gastric and sigmoid mucosal PCO₂ and pH were measured before aortic occlusion, during occlusion (30 min after clamping the aorta) and at the end of operation.

The study was performed in the intensive care unit where the patient’s lungs were ventilated using controlled mechanical ventilation (Servo 900C, Siemens AB, Solna, Sweden) with an FiO₂ of 0.4–0.5 and a PEEP of 5 cm H₂O. Adequacy of sedation was confirmed by careful bedside monitoring and, if necessary, bolus doses of thiopental or oxycodone chloride were given. The hepatic vein was cannulated via the right internal jugular vein, and the correct position verified with fluoroscopy using a small amount of contrast dye. After baseline measurement of splanchnic blood flow, 10 patients in the epidural group received an epidural 40-mg bolus of 0.5% bupivacaine followed by an infusion of 15 mg h⁻¹. In the control patients, no epidural injection or infusion was given. In order to detect successful sympathetic block, mean arterial pressure was evaluated 50 min after the start of epidural analgesia (epidural: mean 90 (SD 12) vs 71 (9) mm Hg; control: 94 (15) vs 89 (12) mm Hg; P<0.05 between groups, two way analysis of variance for repeated measurements). Measurement of splanchnic blood flow was repeated 2 h after baseline measurements. During splanchnic blood flow measurement, systemic VO₂ was measured continuously from the inspired and expired gases by open circuit indirect calorimetry (Deltatrac, Datex-Engström Corp., Helsinki, Finland). The device has been validated in this laboratory and it has a relative error of less than 5% under the study conditions.¹⁹

Splanchnic blood flow was measured using the dye dilution method, described previously.²⁰ In brief, after a priming dose of 12 mg of indocyanine green was injected into a peripheral vein, a constant infusion of 1.1 mg min⁻¹ was continued for 30 min. Blood was sampled for measurement of indocyanine green concentrations in arterial and hepatic venous blood after 20, 25 and 30 min of infusion. In order to evaluate if steady state dye concentrations were achieved, the coefficient of variation was calculated for each individual during each blood flow measurement. Indocyanine green concentrations were in steady-state plateau, as indicated by a coefficient of variation of 2.8±2.1% in radial arterial blood and 4.0±3.0% in hepatic venous blood. Indocyanine green extraction was 71.0±10.9% (range 41–89%). Within-subject coefficient of variation for splanchnic blood flow, based on three consecutive blood samples of each measurement period, was 4.6±3.6%.
Epidural analgesia does not increase splanchnic blood flow

STATISTICAL ANALYSIS
For sample size calculations, we assumed an SD of 0.2 litre min⁻¹ m⁻² for splanchnic blood flow and 0.068 for mucosal pH, and a within-subject coefficient of variation of 4% for splanchnic blood flow measurements based on our previous studies.20 Using a two-sided α of 0.05, this would allow us to detect a change of 0.18 litre min⁻¹ m⁻² in splanchnic blood flow and 0.06 pH units in mucosal pH with 80% power with 10 evaluable patients per group.

Friedman one-way analysis of variance was used to detect changes during surgery. If statistically significant changes were found, the Wilcoxon signed ranks test was used post hoc to locate the changes. The tonometry data were compared with preclamping values and haemodynamic data with preoperative values. The Bonferroni correction was used for multiple comparisons. Postoperative data were tested using two-way analysis of variance for repeated measurements. The values that failed the normality test were tested using the Kruskal–Wallis non-parametric test (sigmoid–arterial $P_{CO_2}$ difference in the second blood flow measurement). Findings were considered significant if $P<0.05$. All results are presented as mean (SD), unless otherwise stated.

Results
Clinical data of the patients are presented in table 1. Three patients required intensive care for 2–4 days because of postoperative respiratory failure (two patients in the epidural and one patient in the control group) and two patients in the control group and one patient in the epidural group had postoperative wound infection.

Peri- and postoperative haemodynamic and oxygen transport data are shown in tables 2 and 3. During surgery, the gastric–arterial $P_{CO_2}$ difference and arterial–gastric pH-gap remained unchanged. In contrast, sigmoid–arterial $P_{CO_2}$ difference and arterial–sigmoid pH gap increased after aortic occlusion but returned to preclamping values by the end of surgery (fig. 1).

At baseline, in the intensive care unit, there were no differences between the epidural and control groups for haemodynamic variables, splanchnic blood flow or oxygen transport (tables 3, 4, fig. 2). Mean arterial pressure decreased in both groups after 2 h but there were no significant differences between groups (table 3). There were no differences in gastric–arterial or sigmoid–arterial $P_{CO_2}$ differences or arterial–gastric or arterial–sigmoid pH-gaps between the groups after operation (table 3).

Hepatic venous oxygen saturation decreased and hepatic venous lactate and splanchnic oxygen extraction increased, but there were no differences between the groups (table 4). Hepatic lactate uptake was higher in the epidural group.

Six patients in the epidural group and nine patients in the control group received sodium nitroprusside in the intensive care unit during splanchnic blood flow measurements and the infusion rate was maintained constant during the measurements. The need for sodium nitroprusside in the epidural group decreased during epidural analgesia (epidural: mean: 1.2 (SD 1.8) (range 0–6.0) vs 0.3 (0.6) (0–1.6) μg kg⁻¹ min⁻¹; control: 1.6 (1.7) (0–4.7) vs 2.0 (2.3) (0–7.2) μg kg⁻¹ min⁻¹).

Table 1 Clinical data of the patients (mean (SD or range) or number)

<table>
<thead>
<tr>
<th></th>
<th>Epidural group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (M/F)</td>
<td>9/1</td>
<td>7/3</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>65 (54–76)</td>
<td>61 (51–68)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aorto-iliac occlusive disease</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Abdominal aortic aneurysm</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Preoperative disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Pulmonary disease</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Preoperative medication</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Nitrates</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Ca blockers or ACE inhibitors</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Acetylsalicic acid</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Type of surgery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aorto-femoral bypass</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>Aorto-iliac bypass</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Duration of aortic clamp (min)</td>
<td>101 (36)</td>
<td>87 (34) (54–166) (30–141)</td>
</tr>
<tr>
<td>Inferior mesenteric artery occluded before operation</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Duration of intensive care (days)</td>
<td>1.4 (1.0)</td>
<td>1.1 (0.3) (1–4) (1–2)</td>
</tr>
<tr>
<td>Duration of hospital stay (days)</td>
<td>10 (4)</td>
<td>8 (2) (7–17) (6–13)</td>
</tr>
</tbody>
</table>

Discussion
The main finding of the study was that epidural analgesia after abdominal aortic reconstruction surgery did not improve splanchnic tissue perfusion, as assessed by mucosal tonometer and measurements of total hepatosplanchnic blood flow. Mucosal acidosis was uncommon and mucosal perfusion or metabolism was evidently not affected by epidural analgesia.

There are few studies on the effect of epidural block on intestinal perfusion. Johansson and colleagues concluded that during colorectal surgery, epidural bupivacaine had a favourable effect on intestinal blood flow measured by laser Doppler flowmetry.22 Sutcliffe and colleagues found that perioperative gastric mucosal acidosis was less common with epidural anaesthesia in a heterogenous group of major surgery patients.23 Because data on haemodynamics and volume status were not given, interpretation of this observation is difficult. Increased volume substitution because of hypotension induced by epidural anaesthesia may have prevented splanchnic hypoperfusion caused by persistent hypovolaemia.24 Animal studies have demonstrated that the haemodynamic response is dependent on the spread of epidural block.14 15 Hogan and colleagues showed that hypotension after lumbar epidural anaesthesia causes splanchnic vasoconstriction because of baroreceptor activation, while thoracic epidural anaesthesia blocks sympathetic activity, resulting in mesenteric vasodilatation. Hepatic arterial blood flow did not change in response to epidural anaesthesia in their animal model. It is conceivable that in humans also, volume status and hypotension may modify the response of blood flow distribution to epidural block.

In our study, assessment of neural block was not feasible because of residual general anaesthesia.
Table 2 Systemic haemodynamic state and oxygen transport during surgery. *P<0.05 vs before induction; †P<0.05 vs before clamping (Wilcoxon) (mean (sd))

However, the acute reduction in mean arterial pressure in the epidural group and the reduced need for sodium nitroprusside suggests successful sympathetic block. The dose of bupivacaine used in our study was likely to produce and maintain at least partial thoracic sympathetic block.25 Nydahl and colleagues have shown that cephalad spread of epidural anaesthesia is more extensive in elderly patients than in the young.26 In their study, 0.5% bupivacaine 20 ml injected at L2–3 into the epidural space caused analgesia to pinprick at and below T4. Recently, Visser and co-workers showed that low thoracic epidural block with lidocaine 60 mg injected at T7–9 spread 7.7±1.8 segments upwards.27 We cannot exclude the possibility that sympathetic block up to T5 was not achieved. Nevertheless, most of the local anaesthetic should have spread into the area of the coeliac and superior and inferior mesenteric ganglions. Accordingly, insufficient sympathetic block is unlikely to explain the lack of changes in splanchic blood flow and mucosal PCO₂ or pH. The peri- and postoperative changes in sigmoid mucosal pH during aortic surgery have been well described.6 7 27 Sigmoid mucosal pH decreases after clamping and reaches its nadir of approximately 7.1 at 4–6 h after clamping and returns to preoperative values by the first postoperative morning. Complications can be expected if sigmoid mucosal pH does not return to pre-clamping values in 6–12 h. A sigmoid mucosal pH persistently less than 7.1 has been proposed as an early warning of problems.7 Notably, multiple organ dysfunction syndrome (MODS) is the most common complication associated with prolonged sigmoid mucosal acidosis after aortic surgery. In our study, except for clamping-induced changes at the sigmoid mucosa, any perioperative changes in mucosal–arterial PCO₂ difference or arterial–gastric pH gap were related to changes in arterial PCO₂ or systemic acid–base balance.

In contrast with sigmoid mucosal perfusion, gastric mucosal perfusion should not be affected by vascular occlusion related to aortic surgery, and it should better reflect the patient’s overall haemodynamic state. Indeed, the only mechanisms likely to influence gastric mucosal perfusion are anaesthesia and changes in systemic haemodynamics and blood volume. Our patients had no perioperative gastric mucosal acidosis, and the small reduction in gastric mucosal pH was related to changes in systemic acid–base status. This demonstrates that careful management of perioperative haemodynamics prevents gastric mucosal hypoperfusion.

In our study both gastric and sigmoid mucosal perfusion were well maintained after operation and

Table 3 Systemic haemodynamic state and oxygen transport after surgery (mean (sd)). *Group time interaction, P<0.05, two-way analysis of variance for repeated measurements; †time effect, P<0.05, two-way analysis of variance for repeated measurements

![](image)
Table 4 Splanchnic blood flow and oxygen transport after surgery (mean (SD)). * Epidural group vs controls, P<0.05, two-way analysis of variance for repeated measurements; †time effect, P<0.05, two-way analysis of variance for repeated measurements

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>2 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Splanchnic blood flow</td>
<td>Epidural</td>
<td>0.72 (0.28) 0.84 (0.41)</td>
</tr>
<tr>
<td>(litre min⁻¹ m⁻²)</td>
<td>Control</td>
<td>0.70 (0.21) 0.70 (0.15)</td>
</tr>
<tr>
<td>Fractional splanchnic</td>
<td>Epidural</td>
<td>0.25 (0.08) 0.22 (0.05)</td>
</tr>
<tr>
<td>blood flow</td>
<td>Control</td>
<td>0.22 (0.06) 0.21 (0.07)</td>
</tr>
<tr>
<td>Splanchnic O₂ consumption</td>
<td>Epidural</td>
<td>42 (13) 39 (10)</td>
</tr>
<tr>
<td>(mmol litre⁻¹)</td>
<td>Control</td>
<td>37 (6) 37 (9)</td>
</tr>
</tbody>
</table>

not influenced by epidural analgesia. Measurement of splanchnic blood flow was performed at the time when, according to previous studies, mucosal acidosis should have been common. Compared with previous studies, mucosal acidosis or increased mucosal–arterial CO₂ difference was remarkably rare in our study and even patients with a high sigmoid–arterial PₐCO₂ difference did not develop serious complications. This may be because none of our patients had signs of insufficient systemic perfusion and oxygen delivery, judged either by the information provided by the invasive haemodynamic monitoring, gastric tonometry or arterial blood lactate concentrations. Unfortunately, most of the previous studies provide insufficient haemodynamic data for evaluation of systemic haemodynamic state, oxygen transport and vasoactive drugs used, and therefore it is difficult to assess if the mucosal acidosis observed in previous studies was simply a result of inadequate systemic blood flow and oxygen delivery. In our study, sigmoid–arterial PₐCO₂ difference increased remarkably in one patient during epidural analgesia. Recovery of this patient was also uneventful. The possibility that the tonometer had slid into the rectum cannot be excluded.

Changes in total hepatosplanchnic blood flow after aortic surgery have not been reported previously. The method we used does not allow assessment of the contribution of hepatic arterial and portal blood flow to total blood flow. Because of multivessel influx of the splanchnic bed and the possible heterogeneity of blood flow responses within the splanchnic bed, we cannot exclude changes in small intestine blood flow in response to epidural analgesia. Nevertheless, the lack of changes in total hepatosplanchnic blood flow and local perfusion of the mucosa, both in the upper and lower gastrointestinal tract, suggest that epidural analgesia had no effect on splanchnic perfusion in this clinical setting.

Interactions of general anaesthesia and sedation on the effects of epidural analgesia on splanchnic blood flow are unknown. Drugs used for general anaesthesia may depress the myocardium and reduce both cardiac output and splanchnic blood flow. On the other hand, vasodilatation caused by general anaesthesia may influence the splanchnic circulation. If the patients had been awake, the result may have been different because increased sympathetic activity would have been eliminated more effectively in the epidural group.

The higher lactate uptake in the epidural group was related mainly to lower hepatic venous lactate values. This may be associated with more efficient hepatic lactate uptake by the liver or lower intestinal lactate production, already at a minimum in the epidural group.

As the individual responses to any antihypertensive drug are highly variable and as both arterial pressure and its treatment may interfere with regional blood flow, we found no way of avoiding the use of different doses of sodium nitroprusside. Moreover, treatment of hypertension was considered necessary by the surgical team. We found the use of sodium nitroprusside a reasonable compromise, as we have recently shown that it does not influence fractional splanchnic blood flow in coronary artery bypass patients after surgery. Mean arterial pressure decreased to a greater extent in the epidural group but it also decreased in the control group. There were no significant differences between the groups, suggesting that the mean arterial pressure had no effects on splanchnic blood flow or on gastric or sigmoid pH. Another
potential confounding variable is the presence of both aneurysmatic and aorto-iliac occlusive patients. Despite this, the individual responses in the epidural group did not suggest any difference in the pattern of responses to epidural analgesia. Our data suggest that epidural analgesia did not improve splanchnic perfusion in elective postoperative aortic surgery patients. Our results also showed that both gastric and sigmoid mucosal acidosis were uncommon in invasively monitored patients with a stable haemodynamic state and that short-term mucosal acidosis has no apparent effect on recovery.

Acknowledgement

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References


