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Combined general and regional anaesthesia in pulmonary endarterectomy

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Introduction. The effects of combined high thoracic epidural with general anaesthesia in comparison with total intravenous anaesthesia in 32 patients undergoing pulmonary endarterectomy were prospectively studied.

Method. Thirty two patients were randomly allocated either to the study group or to the control group using sealed envelopes. The 16 study group patients received high thoracic epidural and general anaesthesia. The 16 control patients received total intravenous anaesthesia alone. Haemodynamic parameters, drug use, as well as time to extubation, rate of complications, postoperative pain, the length of intensive care unit stay, and mortality were recorded.

For comparison of discrete variables distribution Fisher's exact test in four-field tables was used. Distribution of continuous variables among the groups was analysed by Kolmogorov-Smirnov test and the results were verified using other tests. Repeated measure-ANOVA was used to compare the differences of the haemodynamic variables between the groups.

Results. There were no differences in demographic parameters between the groups. There was no haemodynamic instability in either group during induction of anaesthesia. The study group patients had significantly lower sufentanil consumption (2.1 ± 0.7 vs. $9.1 \pm 3.1 \mu\text{g}\cdot\text{kg}^{-1}$, $P < 0.001$), shorter period of artificial ventilation (34 ± 35 vs. 52 ± 49 hours, $P = 0.0318$) and better pain score at 3 h (0.1 ± 0.3 vs. 0.9 ± 1.4 , $P = 0.015$), 12 h (0.1 ± 0.5 vs. 0.9 ± 0.8 , $P = 0.002$) and 24 h (0.4 ± 0.5 vs. 1.3 ± 1.0 , $P = 0.007$) postoperatively.

Conclusions. This study has shown that combined epidural and general anaesthesia is a suitable and potentially beneficial anaesthetic option in patients who are selected for pulmonary endarterectomy. It can provide the same haemodynamic stability as total intravenous anaesthesia, and moreover contributes to significant shortening of tracheal intubation postoperatively, although this has not been shown to decrease either length of intensive care unit stay or mortality.

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Thoracic paravertebral block with bupivacaine 0.25% versus intrathecal morphine for pain relief after thoracotomy

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Introduction. This study aimed to compare the effects of a thoracic paravertebral block with bupivacaine 0.25% and intrathecal morphine on postoperative pain and morphine consumption after thoracotomy [1].

Method. After obtaining ethics committee approval and informed consent, 40 patients aged 18-65 years, undergoing thoracotomy were randomized to receive either thoracic paravertebral block or intrathecal analgesia for relief of post-

thoracotomy pain. In the paravertebral group, a bolus of 20 ml of 0.25% bupivacaine was given at the end of surgery through a catheter placed in the paravertebral region before thoracotomy closure, which was followed by a 24 h infusion of 0.25% bupivacaine ($0.1 \text{ ml kg}^{-1} \text{ h}^{-1}$). In the intrathecal group, intrathecal $10 \mu\text{g kg}^{-1}$ morphine was given at the end of surgery. All patients received i.v. morphine PCA after extubation. Postoperative VAS scores at rest and on coughing, morphine consumption, sedation scores (Ramsay scale), arterial pressures, heart rate, respiratory rate, oxygen saturation, arterial blood gases and peak expiratory flow rates were recorded.

Results. Morphine consumption at 24 h was significantly lower in the paravertebral group when compared with the intrathecal group ($11.4 \pm 3.3 \text{ mg}$ vs. $18.4 \pm 9.5 \text{ mg}$, respectively, $P < 0.05$). Postoperative VAS scores, haemodynamic and respiratory parameters were similar between groups. The sedation scores in the intrathecal group were higher than those in the paravertebral group at 10, 30, 60 min and 4 h postoperatively ($P < 0.05$).

Discussion. Thoracic paravertebral block with 0.25% bupivacaine and intrathecal morphine were both effective in controlling post-thoracotomy pain, but paravertebral analgesia was associated with improved sedation scores during the initial four hours after surgery and a lower morphine consumption at 24 hours after surgery.

Reference.

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Thoracic epidural anaesthesia impairs the haemodynamic response to acute pulmonary hypertension by depressing right ventricular contractility in pigs

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Introduction. Thoracic epidural anaesthesia (TEA) is increasingly used in the perioperative management of patients with increased cardiac risk. It was recently suggested that sympathetic blockade of cardiac efferents may affect right ventricular (RV) function and its response to acute pulmonary hypertension (PHT) [1]. We tested this hypothesis in an experimental model of acute RV obstruction and compared the effects of thoracic and lumbar epidural anaesthesia (LEA).

Methods. Eighteen pigs were instrumented with a microtip pressure-volume catheter in the RV, transonic flow probes around the pulmonary artery (PA) and the right coronary artery, a microtip pressure catheter in the PA, a 22G-catheter within a RV free wall coronary vein, and an epidural catheter with the tip at level TH2 (n=9) or L2 (n=9). Animals were paced with a heart rate of 100 min^{-1} . Following baseline measurements, acute PHT was induced by adjustable constriction of the PA with a tourniquet. Animals were then randomly assigned to receive TEA (n=6, 1 ml bupivacaine 0.5%), lumbar EA (LEA) (n=6, 4 ml bupivacaine 0.5%) or control (C, n=6). Final measurements were performed 30 min after injection. The results were statisti-

cally analysed using repeated measures analysis of variance and the Tukey's HSD-test for post-hoc testing.

Results. PHT resulted in all groups in an increase in the slope of preload-recruitable stroke work (Mw) ($+50\pm 24\%$) and PA effective elastance (PA-Ea) ($+264\pm 34\%$), while CO decreased by $31\pm 10\%$. Induction of EA caused a sympathetic block ranging from C7-TH5 (TEA) and from TH13-L5 (LEA) as assessed by skin temperature. TEA was associated with a decrease in Mw [1.5 ± 0.6 vs. 2.8 ± 0.5 (LEA) vs. 3.2 ± 0.9 mWatt·s·ml⁻¹ (C), $P<0.01$], and consequently in CO [1.8 ± 0.3 vs. 2.7 ± 0.4 (LEA) vs. 2.4 ± 0.3 L·min⁻¹ (C), $P<0.01$]. RV coronary flow, oxygen delivery and consumption were not different between the three groups. Systemic vasodilation occurred only in the LEA-group.

Conclusion. Selective blockade of cardiac sympathetic nerves by TEA deteriorated systemic haemodynamics in PHT. This effect was mainly attributable to the depression of RV contractility and was neither the result of impaired coronary flow dynamics nor of systemic vasodilation.

Reference.

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The effects of propofol and sevoflurane on modulation of inflammation and oxidative stress in the kidney following aortic cross clamping

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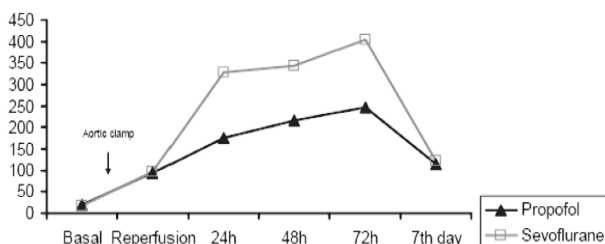
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Introduction. Propofol has been reported to provide protection against ischaemia-reperfusion (IR) injury. The aim of this study was to compare the effect of propofol with sevoflurane on kidney NFκB expression and systemic inflammatory responses induced by aortic clamping.

Method. Twenty piglets were divided into four groups: sham surgery group with propofol (group SP, n=5) or with sevoflurane (group SS, n=5); and suprarenal clamping for 30 min with aorta-aortic bypass under propofol (group CP, n=5) or sevoflurane (group CS, n=5) anaesthesia. Peripheral blood and kidney biopsies were taken at different periods. Plasma creatinine, myeloperoxidase, tumour necrosis factor-α, interleukin 1-β, kidney superoxide anion, superoxidase dismutase and the expression of inducible nitric oxide synthase and renal tissue NFκB were measured. Data were analyzed using the Fisher exact test and analysis of variance (Student-Newman-Keuls or Scheffe test or Kruskal-Wallis Z test, as indicated).

Results. Compared with CS group, animals in the CP group had lower concentrations of inflammatory markers and diminished NFκB expression and iNOS activity. (Fig.1)

Fig. 1. Representative evolution of markers of systemic inflammatory response.



Conclusions. Compared with sevoflurane, propofol administration during supra-renal aortic clamping and unclamping led to modulation of markers of inflammation and decreased NF-κB expression.

Reference.

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Lower tidal volumes do not impair oxygenation during pressure controlled one-lung ventilation

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Introduction. It is suggested that tidal volumes (TV) during one-lung ventilation (OLV) should be as high as in two-lung ventilation (TLV) to ensure adequate oxygenation [1]. However, recent studies have shown that low TV during OLV are (or can be) associated with a decreased incidence of post-pneumonectomy pulmonary oedema [2]. On the other hand, the effects of low TV on oxygenation and pulmonary shunt (Qs/Qt) have been less examined.

Method. In 31 patients undergoing pulmonary surgery, two ventilation strategies were applied in different sequences. Patients were ventilated with pressure-controlled ventilation (PCV) during the entire operation. During TLV, ventilation pressures were adjusted to obtain a tidal volume to maintain normocapnia. During OLV, TVs were adjusted either by increasing the ventilation pressure to obtain the same TV as in TLV (Normocapnic Stage: StN); or by keeping the same ventilation pressure as in TLV and permitting the TV to decrease (Hypercapnic Stage: StH). In each patient, both methods were applied in a randomized cross-over fashion. At the end of every stage (20 minutes), PaO₂, PaCO₂, TV, and Qs/Qt were measured. Data were compared with Student's *t* test and repeated measures ANOVA.

Results. There was a significant difference in TV (569 ± 180 ml vs. 399 ± 136 ml; $P<0.0001$) and in PaCO₂ (39.1 ± 6.2 mmHg vs. 46 ± 7.6 mmHg; $P<0.0001$) between StN and StH, respectively. There was a slight but insignificant decrease in PaO₂ and a slight but insignificant increase in Qs/Qt during StH compared to StN (StN vs StH: 192 ± 56 mmHg vs 176 ± 50 mmHg; and $28.7\pm 8.8\%$ vs $31.4\pm 8.4\%$).

Discussion. Lower TVs do not result in any important clinical change in oxygenation during OLV, suggesting that ventilation with lower TV can be used without any drawback.

References.

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Anaerobic metabolism during thoracic anaesthesia for hyperthermic intrathoracic chemotherapy

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