Abstracts from the literature

Effects of extradural morphine or bupivacaine on endocrine response

The endocrine response, and the relief of pain, following the extradural administration of morphine or bupivacaine (0.5%) were studied for 24 hours after abdominal surgery and compared with a control group given conventional i.v. morphine after operation. Samples were taken before and at 2, 4, 6, 12 and 24 hours after skin incision. Pain relief in both extradural groups was significantly better when compared with the control group. In all three groups, the plasma concentration of cortisol was increased immediately after surgery. Thereafter, significantly lower values were seen in the extradural groups. Plasma adrenaline concentration was lower immediately after surgery only in the group given the extradural local anaesthetic. Plasma noradrenaline concentration remained unchanged after extradural local anaesthesia while an intermediate increase occurred after extradural morphine. Plasma noradrenaline concentration was significantly greater in the controls compared with both extradural groups. These results indicate that extradural analgesia with a local anaesthetic drug can suppress the increases in the plasma concentration of catecholamines and cortisol after surgery. In contrast to extradural local anaesthetic extradural morphine cannot suppress the endocrine response immediately after surgery. However, later in the postoperative period, extradural morphine can suppress the endocrine response, thus indicating that postoperative pain is a factor involved in the stress response following surgery. *Rutberg H, Hakanson E, Anderberg L, Jorfeldt J, Martensson J, Schildt B. Effects of the extradural administration of morphine, or bupivacaine, on the endocrine response to upper abdominal surgery. Br J Anaesth 1984; 56: 233–8.*

Differential lung ventilation with unilateral PEEP

Differential lung ventilation with positive end expiratory pressure (PEEP) improves pulmonary gas exchange when used in the supportive care of patients with severe unilateral or asymmetrical lung disease. Once the provision of selective PEEP to the two lungs is accomplished, the best method of partitioning the tidal volume between the two lungs is unknown. Twelve mongrel dogs were given a unilateral hydrochloric acid (HCL) aspiration injury. A computer controlled differential lung ventilation system was used to ventilate four dogs with equal volumes to each lung, four dogs with equal driving pressure (end inspiratory pressure-PEEP) to each lung, and four dogs with equal end-tidal CO_2 fraction from each lung. The respiratory rate was feedback controlled to maintain $PaCO_2$ at 4.67 kPa. The dogs were kept supine and ventilated with 30 per cent O_2. Following injury, the PEEP was set at 0 kPa for one hour. The dogs were then given 1.36 kPa and 2.72 kPa PEEP to the injured lung for two hours in a cross-over fashion. The assignments of the tidal volume controller, the side of injury, and the PEEP sequence was random. Oxygen tension fell and pulmonary venous admixture increased after giving the HCL injury. In all three groups considered simultaneously, unilateral PEEP improved PaO_2 and venous admixture. The equal tidal volume distribution was the only group to show a significant improvement in PaO_2 at both PEEP increments (0 to 1.36 kPa and 2.72 kPa). There was a significant difference in tidal volume allocation between the three groups with the equal end-tidal and equal pause pressure groups only minimally ventilating the injured lung. With differential lung ventilation and unilateral PEEP, equal partitioning of tidal volume provides the highest PaO_2, compared to the other two methods of partitioning tidal volume. *East TD, Pace NL, Westenskow DR, Lund K. Differential lung ventilation with unilateral PEEP following unilateral hydrochloric acid aspiration in the dog. Acta Anaesthesiol Scand 1983; 27: 356–60.*

Reversal by naloxone of the antihypertensive action of clonidine

The effects of clonidine, naloxone, and their combination on arterial blood pressure (BP), heart rate

(HR), and haemodynamic and biochemical parameters were examined in 29 patients with essential hypertension. Treatment for three days with 0.3 mg/day clonidine reduced BP and HR, and these effects were quickly reversed by a single injection of 0.4 mg iv naloxone in 17 of the patients (responders), but not in the remaining 12 (nonresponders). Responders had higher control values for cardiac output, stroke index, plasma renin activity (PRA), and plasma epinephrine levels than did nonresponders. Basal BP was similar in the two groups, but clonidine decreased BP, PRA, and plasma epinephrine more in responders. Naloxone given during placebo treatment had no significant effects. During clonidine treatment naloxone increased BP, HR, total peripheral resistance, PRA, and plasma epinephrine and norepinephrine, and decreased stroke volume in responders, whereas in nonresponders its only effect was a small increase in HR. It is concluded that in a subset of hyperadrenergic, hypertensive patients the antihypertensive effect of clonidine involves a naloxone-reversible inhibition of central sympathetic outflow, probably mediated by the release of an endogenous opioid. *Farsang C, Kapocsi J, Vajda L et al. Reversal by naloxone of the antihypertensive action of clonidine: involvement of the sympathetic nervous system. Circulation 1984; 69; 461–7.*

Contaminated condensate in mechanical ventilator circuits

Ventilator circuit colonization and condensate formation were studied in 30 mechanical ventilators during the first 24 hours after a circuit change. Parts of the circuit nearest the patient were more frequently contaminated and had the highest levels of colonization. There was rapid colonization of tubing after a circuit change; 33 per cent of the ventilators were colonized at 2 hours, 64 per cent at 12 hours, and 80 per cent at 24 hours. The median level of colonization at 24 hours was 7×10^4 organisms/ml. Water condensate collected in the ventilator circuits at a mean rate of 30 ml/hours (range, 10 to 60 ml/hour). At 24 hours, 80 per cent of the condensate samples were contaminated at a median level of 2×10^5 organisms/ml. The bacteria isolated from the condensate usually correlated with organisms previously isolated from the patient's sputum, suggesting that the patient's oropharyngeal flora is the primary source of circuit colonization. Highly contaminated condensate in the ventilator circuit may be a significant risk factor for nosocomial pneumonia. The authors suggest that circuit condensate be emptied regularly, handled as infectious waste, and that special efforts be taken to prevent contaminated condensate from inadvertently washing into the patient's tracheobronchial tree. *Craven DE, Goularte TA, Make BJ. Contaminated condensate in mechanical ventilator circuits. A risk factor for nosocomial pneumonia? Am Rev Respir Dis 1984; 129: 625–8.*

Permeability pulmonary edema caused by venous air embolism

A 22-yr-old man developed severe pulmonary edema after blowing air into tubing connected to a catheter inserted in a vein in his forearm. Pulmonary edema was rapid in onset, peaking in intensity about 12 hours after the air had been insufflated. The patient's edema fluid to plasma protein concentration ratio of 0.79 showed that the edema fluid was rich in protein. Vascular pressures were normal except for mild pulmonary arterial hypertension and systemic hypotension. The patient's clinical course, edema fluid protein concentrations, and vascular pressures were characteristic of an increased microvascular permeability type of pulmonary edema. The patient recovered fully within 48 hours of the air infusion. His response to venous air embolism was similar to findings in an experimental model of pulmonary edema in sheep infused with venous air emboli. As long as air is not infused continuously for long periods or in very large amounts, this disorder is probably self-limiting, and supportive care may be the only treatment necessary. *Clark MC, Flick MR. Permeability pulmonary edema caused by venous air embolism. Am Rev Respir Dis 1984; 129: 633–5.*