TITLE: REDUCTION OF SIGNIFICANT CARDIAC MORBIDITY BY EPIDURAL MORPHINE IN NON-CARDIAC SURGERY

AUTHORS: W.S. Beattie, F.R.C.P.(C),D.N. Buckley, F.R.C.P.(C), J.B. Forrest, F.R.C.P.(C)
AFFILIATION: Anaes. Dept., McMaster University, Hamilton, Ontario, CANADA L8N 3Z5

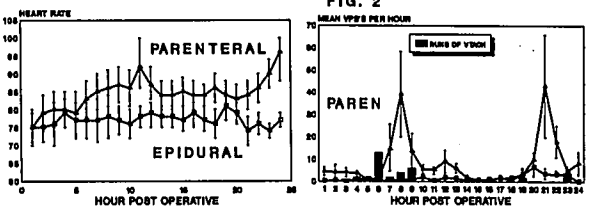
Peri-operative morbidity and mortality is related to cardiac complications in adults who undergo major non-cardiac surgery.[1] While the cause of post-operative myocardial infarction (MI) is not known, evidence suggests that MI is related to increased sympathetic nervous system activity (SNSA).[2] Epidural analgesia attenuates increased SNSA and the correlates of ischemia.[3] This study compares epidural to parenteral analgesia in patients undergoing major upper abdominal, thoracic or peripheral vascular surgery.

Twenty patients with 2 or more risk factors for coronary artery disease were prospectively studied with IRB approval after informed consent. Hr, arrhythmias and ST segment (real time) were continuously measured peri-operatively with a Holter Monitor. Pre-op management was not altered by the study. Epidural consisted of placement of a catheter in the epidural space, a test dose of 3 cc of 2% xylocaine followed by morphine in a dose of 0.1 mg/kg. An epidural block was then established with local anaesthetic to the appropriate level for the intended surgery. All patients were given a general anaesthetic (thiopental, narcotic, muscle relaxant) and ventilated intra-operatively. Inclusion in the study did not affect any other aspect of care (ie. patients received ß- or Ca-blocker as deemed necessary by their attending staff physician). Patients wore a calibrated, FM, recorder monitoring lead II and V_5. Tapes were analyzed by an independent blind observer on a Cardiodata Prodigy Computer at 60 times real speed. ST depression was defined as ≥ 1 mm (compared to baseline) persisting more than 0.06 seconds after J point for more than 30 seconds. Parametric and non-parametric statistics were used as required.

Patients in the epidural and parenteral narcotic groups were not different with respect to age, weight, number of cardiac risk factors, number taking anti-anginals, history of CHF or arrythmia. Pre-operative heart rates were similar 73.5 ± 3 (EPI) 76.6 ± 4 (PAR). Post-operatively, there was a significant progressive increase in HR (Fig 1) with peaks at 11 hrs post-op at 24 hrs. Patients receiving epidural narcotic did not exhibit this rhythm. Fig 2 illustrates the ectopic activity, 2 peaks are seen at 8 and 21 hours. Ischemia and VT were seen only in patients receiving parenteral narcotics. Fourteen episodes of VT in 5 patients were all associated with the increase in ectopic activity. Three episodes of ischemia were detected in the parenteral group (30%). All were silent and occurred at higher than base line heart rates and were unrelated to ectopy.

These results suggest epidural analgesia can attenuate the post-operative rise in heart rate; reduce post-operative ectopy and significant arrhythmia. Finally, epidural analgesia may reduce the incidence of post-operative ischemia. A large scale trial is needed to determine if these results translate into reductions in post-op MI and death.

FIG. 2



References
1. Br J Anaes, 61:38, 1988
2. Circulation, 79:733, 1989
3. JAMA, 261:3577, 1989

TITLE: CEREBRAL AUTOREGULATION DURING CARDIOPULMONARY BYPASS IS TEMPERATURE DEPENDENT IN INFANTS

AUTHORS: R Taylor MB,F Burrows MD,B Bissonnette MD
AFFILIATION: Anes Dept and Research Inst, Hospital for Sick Children, University of Toronto, Canada M5G 1X8

Recent reports indicate that cerebral autoregulation is not preserved,in infants during non-pulsatile cardiopulmonary bypass (CPB).[1-3] Others found definite cerebral autoregulation in mature dogs during profound hypothermic non-pulsatile CPB (PHCPB)[4].

With institutional approval cerebral hemodynamics were investigated in 8 infants during normothermic CPB (35-37°C) (NCPB), moderate hypothermic (<26°C) (MoHCPB) and PHCPB (<20°C), using α-stat methods of acid-base CPB management. A transcranial doppler (TCD) was used to record middle cerebral artery (MCA) flow. Mean systemic blood pressure (MAP) was measured using a radial or femoral arterial catheter. Anterior fontanel pressure (AFP), using a Ladd transducer, nasopharyngeal temperature (NPT), carbon dioxide tension ($PaCO_2$), and haematocrit (Hct) were recorded. Cerebral perfusion pressure (CPP) was calculated as MAP-AFP. Cerebral blood flow velocity (CBFV) was plotted against CPP for the three temperatures. Linear regression analysis and r^2 was used to compare CBFV versus CPP during for the 3 groups.

Five infants (9.2 ± 11.2) weeks of age, had CBFV measurements over a range of CPP from 10-80 mmHg during NCPB (fig 1) and MoHCPB (fig 2). PHCPB measurements were recorded in 3 infants (fig 3). $PaCO_2$ (36±7 Torr) and Hct (0.26 ± 0.01) were kept in a narrow range (mean±SD). Autoregulation was intact during normothermic CPB (r^2=-0.017). However, during MoHCPB and PHCPB there was a relationship, indicating that CBFV is pressure-passive.

Cerebral autoregulation is preserved in infants, during NCPB, but at <26°C it becomes pressure-passive.

We thank MEDASONICS Canada for providing the TCD.

1.Anesthesiology 71:1033,1989 2.Circulation ;80:I209-1,1989
3.Pediatr Cardiol 8:161,1987 4.J T CvS 95:124,1988

Figure1

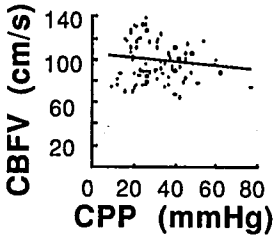


Figure 2

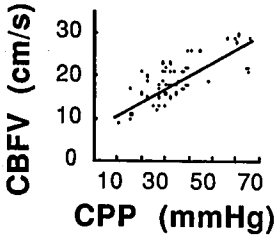


Figure 3