

SCA 69

THE EFFECT OF THORACIC EPIDURAL ANALGESIA ON MYOCARDIAL DAMAGE DURING AND AFTER CORONARY ARTERY BYPASS GRAFT SURGERY, AS MEASURED BY TROPONIN T

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Troponin T has been used in a number of studies as a marker for myocardial tissue damage during cardiac surgery^(1,2). Levels have been shown to correlate with aortic cross clamp time and the degree of myocardial damage that occurs as a result of surgery⁽²⁾. Troponin T has also been shown to identify myocardial damage earlier than cardiac myosin light chain I⁽¹⁾. Thoracic epidural analgesia (TEA) has been shown to decrease the infarct size following coronary artery occlusion in animal models⁽³⁾. This study aimed to assess the effect of TEA on myocardial damage during and after coronary artery bypass grafting, as measured by Troponin T.

33 patients with left ventricular ejection fractions greater than 40% for elective coronary artery bypass grafting (CABG) were recruited to the study following ethics committee approval and informed consent. The patients were randomly allocated to receive TEA plus general anaesthesia (group TEA) or general anaesthesia alone (group GA). General anaesthesia was standardised for both groups using target controlled infusions of propofol and alfentanil. β blockers were withheld on the day of surgery and for the study period. In group TEA an epidural catheter was sited prior to induction of anaesthesia at T2/T3. A demonstrable epidural block was established using 0.5% bupivacaine and then an infusion of 0.125% bupivacaine plus clonidine 0.6 mcg/ml was commenced at 10 mls/hr. Group GA had a target controlled infusion of alfentanil continued into the post-operative period for analgesia.

During cardiopulmonary bypass, mean arterial pressure was maintained between 55 and 75 mmHg for all patients.

Patient characteristics were similar and there was no significant difference in aortic cross clamp times between the two groups. There was no statistically significant difference in inotrope usage or the rise in Troponin T between the epidural and non-epidural groups.

Troponin T levels rose significantly in all patients in both groups following surgery ($p < 0.001$). One patient in the epidural group and two patients in the non-epidural group had very large increases (> 8 ng/ml) in Troponin T, this did not correlate with prolonged aortic cross clamp time.

TEA does offer theoretical advantages for myocardial blood flow over conventional opiate techniques but this study would suggest that TEA does not protect against myocardial damage as assessed by a rise in Troponin T after CABG. Troponin T appears to be a non-specific and inaccurate marker for myocardial damage in patients undergoing CABG. In addition, the use of β blockers in these patients may mask the benefits of TEA and it is also possible that continuous and sustained analgesia using target controlled alfentanil minimizes the risk of myocardial ischaemia.

When assessing myocardial ischaemia post cardiac surgery, a raised Troponin T level should be interpreted with caution.

References:

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