Thoracic epidural anaesthesia and analgesia and outcome

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ABSTRACT
Thoracic – but not lumbar – epidural anaesthesia provides relevant clinical advantages compared with general anaesthesia, improving patients' morbidity and mortality after major surgical procedures.[1] These advantages are excellent perioperative neuraxial analgesia and effective attenuation of the perioperative stress response by a reversible blockade of sympathetic afferents and efferents. In particular the attenuation of sympathetic tone prevents perioperative myocardial ischaemia and improves global and regional left ventricular function, pulmonary function and gastrointestinal perfusion. Thus, thoracic epidural anaesthesia is more than simply an anaesthetic regimen; it also has therapeutic options, especially in high-risk patients who underwent major cardiac, thoracic or abdominal surgical procedures.

Myocardial function

Major surgical procedures are associated with exaggerated perioperative adrenergic stimulation resulting in hormonal response and systemic inflammation. These pathophysiological changes can lead to perioperative myocardial ischaemia or other life-threatening cardiac events. The incidence of perioperative myocardial infarction is 0.2% in patients without and 4.0% in patients with pre-existing coronary artery disease, and severe myocardial ischaemia appears most frequently during the first 48 hours after major surgery.[2] However, in patients with major surgery and pre-existing coronary artery disease the incidence of perioperative myocardial ischaemia is 18%.[3] Thus, effective cardiomyocyte functional protection during a perioperative insult stress requires a balanced preservation versus blunting of ß-adrenergic signalling. Adrenergic sensitisation optimises post-ischaemic functional recovery while desensitisation protects against intraoperative oxygen supply/demand imbalance. The harmful effects of adrenergic stimulation on ß-adrenergic receptor density and ß-adrenergic receptor coupling and the positive effects of thoracic epidural anaesthesia have been well documented.[4] Therefore, attenuation of the stress response via blockade and/or selective anaesthetic regimens, such as thoracic epidural anaesthesia, has the potential to prevent adverse events. Consequently, a multimodal approach to reduce patients' stress response and to improve recovery, consisting of intraoperative general anaesthesia, thoracic epidural anaesthesia, postoperative patient-controlled epidural anaesthesia, early extubation, early oral nutrition and enforced mobilisation is the most appropriate regimen, in particular for patients undergoing thoracic and cardiac surgical procedures.[5]

An activation of myocardial sympathetic nerves in cardiac surgery can result in myocardial ischaemia, especially in patients with coronary artery disease. Subsequently, a reversible cardiac sympathectomy by high thoracic epidural anaesthesia has anti-ischaemic effects owing to the blockade of efferent sympathetic fibres.[6] Clinical studies of high thoracic epidural anaesthesia imply beneficial effects for the perioperative management of patients who underwent coronary artery bypass grafting. The sympathetic blockade by high thoracic epidural anaesthesia results in depression of the endocrine perioperative stress response, an improvement in global systolic and diastolic left ventricular function, and a reduction of new wall motion abnormalities during and after coronary artery bypass grafting.[7,8] These effects of high thoracic epidural anaesthesia may improve the long-term outcome after myocardial revascularisation. Several studies reported the advantages of an additional high thoracic epidural anaesthesia, such as earlier weaning from mechanical ventilation, decreased catecholamine response, reduction of perioperative myocardial ischaemia, improved renal and pulmonary outcome and better pain control. However, there are some problems limiting the routine use of high thoracic epidural anaesthesia for coronary artery bypass grafting. Since the neurological risk of high thoracic epidural anaesthesia for this procedure remains inadequately defined, a placement of the epidural catheter a day prior to surgery is mandatory to minimise the risk of epidural haematoma because of intra-operative systemic anticoagulation. Recently, the use of antiplatelet drugs such as aspirin and clopidogrel in patients undergoing coronary artery surgery has come under the spotlight. Perioperative treatment with these agents totally limits the use of high thoracic epidural anaesthesia for patients undergoing coronary artery bypass grafting.[9] Moreover, in many cases pain control may not be effective for chest tubes and venous graft preparation. Thus, an additional systemic analgesia with opioids and/or non-opioid analgesics is also routinely necessary.

Pulmonary function

Major surgical procedures are known to impair pulmonary function. For example, sternotomy and in particular lateral thoracotomy result in postoperative pain and impair respiratory mechanics and coughing. Both affect the retraction forces of the chest wall. The following major problems result in impaired pulmonary function:

1. Decreased lung volume (e.g. atelectasis, resection of lung tissue, pleural effusion, thoracic restriction)
2. Impaired ventilation (decreased functional residual capacity, dysfunction of the diaphragm, dysfunction of intercostal

muscles, increased airway resistance)
3. Impaired gas exchange (atelectasis, lung oedema, decreased cardiac output, decreased minute ventilation)[10]

The effects of thoracic epidural anaesthesia on lung volume, respiratory mechanics and pulmonary gas exchange depend on the extent of segmental regional blockade and sympathicolysis. Theoretically, thoracic epidural anaesthesia may lead to an alteration of intrathoracic blood volume, lung volume and pulmonary vasotone by sympathicolysis, and to paralysis of intercostals muscles, decreased volume of thoracic cavity and increased thoracic and abdominal compliance. However, the results of prospective, randomised clinical trials postulate beneficial effects of thoracic epidural anaesthesia, and no study reported a deterioration of pulmonary function by thoracic epidural anaesthesia during and after thoracic surgery. The excellent analgesia after thoracic surgery, by contrast, results in lower incidence of respiratory morbidity.[11]

Gastrointestinal perfusion

Gastrointestinal hypoperfusion following low systemic perfusion can also occur during major surgical procedures due to haemorrhage. At the level of microcirculation, hypoperfusion may result either from redirection of blood flow away from the splanchnic organs, mediated by increased sympathetic activity, or from impaired blood flow distribution within the microvascular networks. Because splanchnic hypoperfusion is considered to be important in the development of increased mucosal permeability, endotoxaemia and organ failure, the adequacy of gastrointestinal perfusion has become a major concern in high-risk surgical and critically ill patients.[12]

Thoracic epidural anaesthesia protects the gut from decreased microvascular perfusion and from increased leukocyte-endothelium interaction associated with insults due to haemorrhage/retransfusion. The greatest benefit of thoracic epidural anaesthesia on microvascular perfusion was observed in the muscularis layer.[13] Since thoracic epidural anaesthesia does not increase cardiac output, the question is whether the effects of sympathetic block on splanchnic blood flow are due to a redistribution of blood flow within splanchnic organs, or to an effect of thoracic epidural anaesthesia to increase the proportion of flow directed to these organs. The exact mechanisms underlying the protection, and the potential therapeutic uses of thoracic epidural anaesthesia on gastrointestinal perfusion beyond its use as an anaesthetic or analgesic technique, are matters for further investigation.[14] One important issue in the effect of thoracic epidural anaesthesia on splanchnic perfusion, however, is the location of the epidural block. A complete sympathetic block in the splanchnic region is achieved only if the spread of the local anaesthetic includes the thoracic sympathetic nerve fibres, which extend from T5 to T10. On the other hand, the epidural blockade of lumbar segments results in increased sympathetic activity in the splanchnic nerves due to a barometer drive.[15] For the clinician it is important that thoracic epidural anaesthesia offer advantages after operations on both the upper and the lower gastrointestinal tract, such as a reduced rate of anatomical insufficiency, less frequent vomiting and earlier resumption of gastrointestinal motility, and that these positive effects of high thoracic epidural anaesthesia should be used for early enteral nutrition and mobilisation of patients.[16]

Risks of thoracic epidural anaesthesia

Any invasive medical procedure with an inherent risk mandates a thorough assessment of the risk/benefit ratio. The most common complication of epidural anaesthesia is accidental dural perforation. The incidence using the loss resistance method is approximately 0.6%.[17] The incidence of paraesthesia and neurological injuries is approximately 0.01%–0.001%.[18] The most disastrous complication is paraplegia after development of an epidural haematoma.[19] For this reason the German Society of Anaesthesiology and Intensive Care Medicine published guidelines for the safe use of thoracic epidural anaesthesia in patients treated with anticoagulants. These guidelines, which were published in 1997, were updated recently.[20] A recently published study describes relatively high incidences of spinal haematoma (1:18 000), cauda equina syndrome (1:37 500), meningitis (1:90 000) and epidural abscesses (1:37 500).[21] However, this retrospective analysis, performed for Sweden, addresses the period from 1990 to 1999, without existing rules for thromboembolic prophylaxis. Thus, management of perioperative anticoagulation as well as of the insertion and removal of epidural catheters are necessary to reduce these complications associated with thoracic epidural anaesthesia. SAJAA

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