Thoracic Epidural Anesthesia in Patients with Unstable Angina Pectoris
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Activation of the Sympathetic Nervous System

During acute myocardial ischemia, the sympathetic nervous system is activated rapidly [17, 22]. In addition to chemical factors produced by the ischemic myocardium (e.g. bradykinin, prostaglandins, potassium, and lactic acid) separately [21, 25] or in concert [25], stretching of the ventricular wall during ischemia is also capable of stimulating sensory receptors in the ventricular wall [18]. It is generally agreed that the cardiac sympathetic afferent nerve fibers are the essential pathways for the conduction of anginal pain [26]. The afferent pathways pass through the cervical and the upper thoracic ganglia, project to the five most cranial sympathetic segments [15, 26] of the spinal cord (T1–T5), and eventually join with the neurons of the spinothalamic tract. The stimulus for activation of the sympathetic efferents, however, is not fully understood. Continuous hemodynamic monitoring in patients with unstable angina [5, 9] has demonstrated that, coincident to the onset of anginal pain (and even in the absence of pain), there is an “overshoot” increase in heart rate and in both systolic and diastolic arterial pressures (a “pressor response”), which most likely reflects a spinal cardiocardiac reflex mediated via sympathetic afferents and efferents activated by myocardial ischemia itself [4, 19]. Input from higher nervous centers such as during mental stress [16] or unloading of baroreceptors consequent to an initial reduction in arterial blood pressure, however, may also contribute. Since it has been speculated that sympathetic nerve activity may cause transient stenotic vasoconstriction [23] and thrombus formation [12, 20], it might be that myocardial ischemia itself will trigger a reflex mechanism, which further aggravates the ischemic process by increasing myocardial oxygen demand, coronary vasoconstriction, and platelet aggregation.

High Thoracic Epidural Anesthesia

High thoracic epidural anesthesia (TEA) with local anesthetics has the potential to block cardiac afferent and efferent sympathetic fibers. This technique could, therefore, have beneficial effects on cardiac pain and myocardial ischemia. In a series of studies, we investigated the effect of high TEA in patients with severe coronary artery disease, during both unstable and stable phases. The epidural catheter was inserted between the second and fifth thoracic interspaces. A mean of 4.4 ± 0.3 ml bupivacain (5 mg/ml) induced a blockage of Th1–7 with a duration of 98 ± 9 min.
The Effect of TEA on Cardiac Pain

In the first study, we investigated 28 patients with unstable angina [1] in whom signs or symptoms of myocardial ischemia at rest persisted in spite of maximal available medical therapy, including beta blockers, calcium antagonists, long-acting nitrates, anticoagulants, and nitroglycerin infusion for more than 24 h, which could not be discontinued. All patients but one had a history of previous myocardial infarctions and/or stable angina pectoris. Such patients are known to have a high risk of developing myocardial infarction with an increased mortality [10, 19]. The severity of the underlying coronary artery disease in the patients studied was further verified by the angiographic findings with 18 patients with two- or three-vessel disease, and 3 patients with main stem stenosis. Treatment with nitroglycerin infusion lasted on average 3–4 days (range 1–18). By grading the intensity of chest pain with a modification of the Visual Analogue Scale called the Numerical Rating Scale [13], it was found that the patients were not painfree but had a mean pain score of 2.3 (range 0–10), in spite of maximal infusion rate of nitroglycerin and addition of i.v. morphine (in 46%), indicating a need for supplementary treatment to control pain.

An epidural catheter was inserted followed by blood pressure and heart rate recordings before and 30 min after a bolus epidural injection of bupivacaine. TEA induced a significant decrease in heart rate (although all but one were on betablockers) from 70 ± 3 to 64 ± 3 beats/min, while no significant changes in systolic