TABLE Distribution according to intensity of pain

	Pain score			
	0	*1*	*2*	*3*
Group I (n = 30)	4 (13.3%)*	8 (26.6%)	10 (33.3%)	8 (26.6%)
#				
Group II (n = 30)	5 (16.6%)*	10 (33.3%)	10 (33.3%)	5 (16.6%)
Group III (n = 30)	18 (60%)	7 (23.3%)	4 (13.3%)	1 (3.3%)

Group I (saline), Group II (dexmedetomidine 0.1 $\mu g \cdot kg^{-1}$), Group III (dexmedetomidine 0.2 $\mu g \cdot kg^{-1}$). *$P < 0.001$ when compared with Group III. #$P < 0.05$ when compared with Group III.

Following Ethics Committee approval and written informed consent, 90 patients were randomly divided into three groups. Patients had two peripheral *iv*, with one dedicated for rocuronium. Changes in mean arterial pressure, oxygen saturation, and heart rate were measured. Patients received saline 1 mL (Group I; n = 30), dexmedetomidine 0.1 $\mu g \cdot kg^{-1}$ (Group II; n = 30), dexmedetomidine 0.2 $\mu g \cdot kg^{-1}$ (Group III; n = 30), diluted into 1 mL saline. Five minutes later, a priming dose of rocuronium 0.05 $mg \cdot kg^{-1}$ was injected over 10–15 sec. The patients were observed and asked immediately if they had pain in the arm. Three minutes later a sleep dose of thiopental and rocuronium 450 $\mu g \cdot kg^{-1}$ *iv* were administered. Reactions such as discomfort and pain, withdrawal of the hand, screaming etc., after the administration of rocuronium were recorded as side effects up to 24 hr.

Demographic characteristics were similar in the three groups. The number of patients experiencing no pain were four, five, and 18 in groups I to III respectively. The distribution of pain scores, according to group, are shown in the Table. No side effects were observed.

Yoshikawa *et al.*[2] examined the analgesic effects of orally administered clonidine on pain induced by injection of propofol. They found that with injection of propofol, pain was significantly lower with oral clonidine (5.5 $\mu g \cdot kg^{-1}$). Dexmedetomidine is approximately eight times more α_2 selective than clonidine.[1] The mechanisms of the peripheral analgesic effect of dexmedetomidine have not yet been clearly elucidated. However, there are studies suggesting a novel role for inwardly rectifying hyperpolarization-activated conductances in peripherally mediated antinociception.[3] Clonidine inhibits noradrenaline release at terminal nerve fibre endings, inducing analgesia when administered at peripheral sites, producing analgesia at intra-articular application.[4] Peripheral antinociception produced by clonidine-like drugs, mediated local release of encephalin-like substances is also possible.[5]

While the mechanism remains uncertain, we conclude that dexmedetomidine 0.2 $\mu g \cdot kg^{-1}$ may be useful in prevention of rocuronium injection pain.

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Percutaneous transhepatic biliary dilatation under thoracic epidural analgesia in a patient with a recent myocardial infarction

To the Editor:

Percutaneous transhepatic biliary drainage (PTBD) is one of the non-surgical modalities for treatment of obstructive jaundice.[1] Dilatation of the biliary tracts is extremely painful and may be repeated a number of times over a few days.[2] PTBD is generally performed under local anesthesia with sedation or general anesthesia. Epidural analgesia has been proposed as a method of choice for PTBD.[2] We report the case of a 71-yr-old male, hypertensive patient, with a history of coronary artery disease and chest pain for the last 15 to 16 yr; progressive, painless jaundice for the last 45 days; and myocardial infarction during endoscopic retrograde cholangio pancreaticography 15 days earlier.

In view of the progressive jaundice, urgent decompression of the biliary tract by PTBD was planned.

The patient's chief complaints were pruritus, decreased appetite and mild fever for the last 45 days. The patient was taking sorbitrate 5 mg qid, atenolol 50 mg, and aspirin 100 mg die. His cardiac specific troponin T levels were 1.28 $ng \cdot mL^{-1}$ confirming recent myocardial infarction. Total serum bilirubin was 38.2 mg %, the direct component being 16.0 mg %, alanine aminotransferase was 108 $U \cdot L^{-1}$, aspartate aminotransferase was 98 $U \cdot L^{-1}$ and the alkaline phosphatase was 143 $U \cdot L^{-1}$. The coagulation parameters, platelet count and electrocardiography (ECG) were within normal limits. Chest *x-ray* revealed cardiomegaly. Concentric left ventricular hypertrophy with diastolic dysfunction was observed by echocardiography. Contrast enhanced computer tomography was suggestive of cholangiocarcinoma.

We placed an epidural catheter in the thoracic 5–6 inter vertebral space. Thereafter 6.0 mL ± 4.0 mL (0.1% bupivacaine along with 5 $\mu g \cdot mL^{-1}$ fentanyl) were administered via the catheter. Sensory block extended from dermatomes T2–T10. Aspirin was continued for the prevention of ischemic complications associated with unstable angina.[3] Monitoring consisted of heart rate, invasive blood pressure, ECG and SpO_2. During PTBD (completed in two sessions) and thereafter, the patient was without any pain or hemodynamic instability.

Thoracic epidural analgesia (TEA) in patients with coronary artery disease reduces myocardial oxygen consumption by decreasing heart rate, cardiac output and systemic vascular resistance without jeopardizing coronary perfusion pressure by blocking thoracic 1–5 sympathetic fibres. TEA favourably alters the oxygen supply/demand ratio within ischemic myocardial area.[4] TEA is also effective in controlling pain in angina pectoris patients resistant to conventional medical therapy, including nitroglycerin infusion.[5]

We feel that TEA is a useful analgesic modality in these patients.

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Decrease of the inhibitory effect of lidocaine on trigeminal nerve response by the inflammatory oxidant peroxynitrite

To the Editor:

Although failure to achieve satisfactory anesthesia following the administration of local anesthetics in acute inflammatory tissues is a recognized clinical phenomenon,[1] the precise mechanisms of the failure are still under investigation.[2] To investigate whether peroxynitrite ($ONOO^{-1}$), known to be produced in inflammatory tissues, affects the nerve blocking action of the local anesthetic lidocaine, functional examinations with electrical transmural stimulation (ETS; 7.5 V, 10 Hz, 10 sec) were performed in an isolated rabbit iris sphincter muscle (n = 16). This muscle is innervated primarily by a sensory nerve (trigeminal nerve) other than a sympathetic nerve, and a parasympathetic nerve (oculomotor nerve). ETS releases tachykinin from primary sensory nerve endings and evokes tachykinergic muscular contractions after atropine (1 μM) treatment.[3] The effects of lidocaine (0.2, 6 or 20 mM), lidocaine pre-treated with $ONOO^{-1}$ (1 mM); lidocaine pre-treated with decomposed ONOO-1 (1 mM); lidocaine (3 mM) pre-incubated with 3-morpholinosynonimine-N-ethyl-carbamine (SIN-1, 1 mM; a continuous producer of submicromolar $ONOO^{-1}$);[4] or a solution without lidocaine pre-incubated with SIN-1; on the muscle-contraction evoked