steroidal anti-inflammatory drugs, which he stopped seven days back and paracetamol for his backache. His routine blood tests were unremarkable and no electrocardiogram was done. He did not receive any premedication.

An *iv* induction with propofol and fentanyl and orotracheal intubation with atracurium was uneventful. Anesthesia was maintained with 33% oxygen in nitrous oxide and sevoflurane. The patient was placed in the prone position. He remained very stable throughout and the operation began. As surgery progressed, one episode of sudden bradycardia was noted and the heart rate dropped from 69·min^{-1} to 30·min^{-1}. This episode lasted for only a few seconds and no change in blood pressure, saturation or end-tidal carbon dioxide was recorded. Ten minutes after, a second episode of relatively prolonged bradycardia with a heart rate of 20·min^{-1} necessitated treatment with 600 µg of *iv* atropine. The heart rate went up to 94·min^{-1} after atropine treatment. However, no significant alteration in blood pressure, saturation or end-tidal carbon dioxide was noted. During this second episode the surgeon was informed about the incidence and informed me that a tear in the dura had occurred about ten minutes before (coinciding with the first episode of bradycardia) and that he was repairing the dural hole (coinciding with the second episode of bradycardia). The patient recovered uneventfully.

The correlation in time suggests that the episodes of bradycardia were due to traction on the dura. This may have resulted in afferent parasympathetic stimulation causing a reflex-celiac (vasovagal) reaction. I fully agree with the authors that close vigilance and readily prepared vagolytic and sympathomimetic drugs should be available at all times for quick treatment of this potentially life-threatening situation. This is especially important when patients are undergoing an operation in the prone position when a cardiac arrest would be disastrous.

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Neuroendocrine stress response in laparoscopic surgery for benign ovarian cyst

To the Editor:

Stress-induced changes in postoperative organ function may be implicated in the development of perioperative complications. Modulation of stress response to surgery might favourably influence postoperative morbidity.

We compared the perisurgical stress response using total ***iv*** anesthesia (TIVA) or sevoflurane anesthesia in patients undergoing laparoscopic surgery for benign ovarian cysts.[1–3] This model allowed us to investigate the effect of anesthesia without interference from factors known to affect hormonal response (extensive tissue trauma, high blood loss, high level of postoperative pain).

After obtaining approval by the local Ethic Committee and written patient consent, 20 patients underwent laparoscopic surgery using TIVA (group A = 10) or sevoflurane anesthesia (B = 10).

Randomization was performed using a computer-generated random allocation. Both groups were similar with respect to age, weight, operation time and ASA physical status I. All patients were operated by the same surgeon using a standard technique.[4] Surgery started between 8:30 and 9 a.m. after premedication with oral diazepam 0.2 mL·kg^{-1}.

In group A, a remifentanil infusion of 1 µg·kg^{-1}·min^{-1} was administered for one minute then decreased to 0.5 µg·kg^{-1}·min^{-1}. Anesthesia was induced with propofol 1.5 mg·kg^{-1} followed by an infusion of 150 µg·kg^{-1}·min^{-1}until laparoscopy. Propofol was then reduced to 100 µg·kg^{-1}·min^{-1}and stopped with skin closure. Remifentanil was reduced to 0.3 to 0.25 µg·kg^{-1}·min^{-1} after pneumoperitoneum and stopped when surgeons removed the surgical instrumentation.

Patients in group B received fentanyl 3 µg·kg^{-1} and thiopental 5 mg·kg^{-1}. After loss of consciousness, patients in both groups received vecuronium 0.1 mg·kg^{-1} and the trachea was intubated. Sevoflurane was administered at end-tidal concentrations of 1.8 to 2%. Sevoflurane was discontinued with the end of skin closure. Ketorolac 30 mg was administered for postoperative analgesia in both groups.

Venous samples were collected at 8:00; 30 min after the beginning of surgery; after extubation and two and four hours after the end of surgery (time 0, 1, 2, 3, 4).

Concentrations of norepinephrine, epinephrine ACTH, cortisol, and GH were measured. Differences were analyzed by one way ANOVA and unpaired Student's t test.

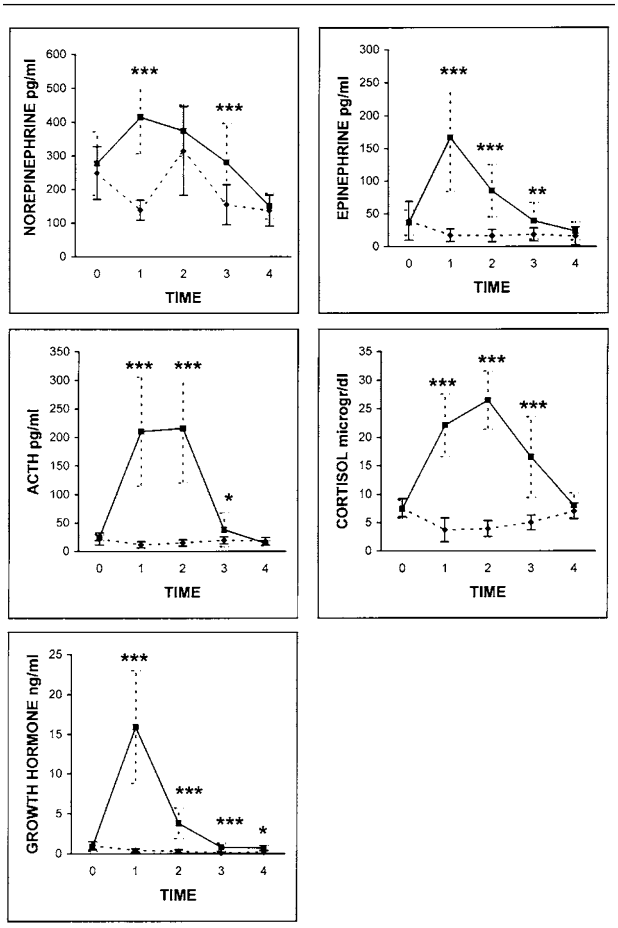

FIGURE Plasma levels (mean ± SD) of stress related hormones in patients undergoing laparoscopy. Total *iv* anesthesia group (dotted line) *vs* sevoflurane anesthesia group (continuous line). *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

Norepinephrine (time 1-3), epinephrine, ACTH and cortisol (time 1-2-3), and GH levels (time 1-2-3-4) were higher in group B (Figure). Inhibition of the ACTH-cortisol response and of catecholamine release in group A was most likely due to remifentanil but propofol could have partially contributed via the GABA receptor.[5]

The results show that TIVA, but not sevoflurane anesthesia, suppressed the stress response in low stress surgery.

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Early blood-patch for spontaneous intracranial hypotension

To the Editor:

Spontaneous intracranial hypotension (SIH) is a rare syndrome, described for the first time in 1938 by Shaltenbrand.[1] Spontaneous cerebral spinal fluid (CSF) leaks are the main etiology of SIH.[2] It is a syndrome of low CSF pressure characterized by postural headaches in patients without any history of dural puncture or penetrating trauma. Neurologic symptoms are sometimes associated with vomiting, vertigo, photophobia and worsened by orthostatism. A CSF opening pressure less than 6 cm H_2O is measured and there is a moderate increase in CSF protein level. Computed tomography (CT) is typically negative, sometimes revealing characteristic small ventricles, brain descent or subdural fluid collection.[3] Brain magnetic resonance imaging (MRI) frequently objectivates a local or a diffuse pachymeningeal enhancement.[3] Medullar MRI, radioisotope cisternography and CT-myelogram allow localization of the CSF leak.[3] The development of MRI technology, explains the increasing number of SIH recognized by neurologists.