intubations. We also have used the Storz video laryngoscope in our institution and found it a very useful tool for difficult intubations and for teaching, not only for novice anaesthetists, but also for anaesthetic assistants, as they too are able to visualize the view of the glottis during external manipulation of the larynx and when applying cricoid pressure for rapid sequence inductions. We have found that the assistant is able to optimize the view of the glottis for the anaesthetist by directly visualizing the view on the portable screen, and not rely solely on feedback from the anaesthetist, as with conventional direct laryngoscopy.

In their study, Groeben and colleagues fail to say whether the assistant performing the external manipulation was allowed to see the view obtained on the screen in the video laryngoscope group. It would be interesting to know whether a subanalysis of this group of patients would show a significant difference in the grade of the view obtained and success of tracheal intubation, as the direct feedback obtained from the video laryngoscope allows the assistant to provide a much better and coordinated view for the anaesthetist during external laryngeal manipulation. Do the authors agree and did they consider a subanalysis of this group in their study?

In the study, a subanalysis of the patients with Cormack and Lehane grade III and IV was performed and this showed a significant difference in the intubation time, in favour of the video laryngoscope group and a significantly better rate of successful intubation. They also found that fewer manipulations were required in this group. The authors, however, failed to state how many of the cases in this subanalysis group required external laryngeal manipulation, although the need for optimizing manoeuvres was mentioned. These significant differences could be attributed to a poorer view of the glottis obtained as a result of ‘blind’ external manipulation in the direct laryngoscope group compared with the video laryngoscope group, where there is improved coordination between both the assistant and the anaesthetist as a result of the image seen on the monitor, which has been shown to result in a significant advantage over the conventional laryngoscope technique. Finally, do the authors feel that they could also conclude that the use of the video laryngoscope eases external laryngeal manipulation, especially in anticipated difficult intubation?

I. Ahmad*
C. Ong
Velliyottillom V. Parameswaran
London, UK
*E-mail: imran.ahmad@gstt.nhs.uk

Response entropy–state entropy difference and nociception: a matter of context

Editor—Aho and colleagues concluded that ‘response entropy (RE)−state entropy (SE) difference cannot reliably be used as an indicator of nociception in patients anaesthetized with propofol, nitrous oxide and remifentanil without neuromuscular blocking drugs’. This statement should be qualified by adding ‘when the values are averaged over 15 s and measured in a state of anaesthesia inadequate for the level of noxious stimulation’.

The authors reached their conclusion based on the observation that the entropy difference after intubation was not different between patients with and without remifentanil. This was because, in both groups, while the RE increased immediately with intubation, the SE also increased, and when the difference was averaged over 15 s, the groups were similar. Later, however, after trochar insertion, a weaker stimulation than intubation, the entropy difference was greater in the non-remifentanil group, a more expected result.

H. Groeben*
A. Jungbauer
M. Schumann
V. Brunkhorst
A. Börgers
Essen, Germany
*E-mail: h.groeben@kliniken-essen-mitte.de


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The issue: what is the implication of an SE increase after an RE increase? Nociception can cause facial EMG activity, which can increase the RE–SE difference, as demonstrated in Table 2 of the paper. Remifentanil decreases this response, also demonstrated by the authors. As EMG activity contains some frequencies below 32 Hz, SE can be affected, as the authors describe. SE also increases with cortical arousal as seen in inadequate hypnotic states. In real time, it is sometimes difficult to discern what is causing the SE to increase: either indicates an inadequate anaesthetic state. Appropriate treatment for a large increase in both SE and RE is often augmenting both hypnotic and anti-nociceptive medications.

In this paper, most of the increases in RE were associated with increases in SE. However in both previous observations, SE increases followed RE increases only in situations of overt clinical arousal with a clearly inadequate anaesthetic state. The authors here do not describe the patient motor response to intubation; one would suspect that an observer would find the overall anaesthetic state to be inadequate for the level of nociception.

The inability of a 15 s averaged RE–SE difference to discriminate between the groups during intubation should not lead to a blanket statement about the reliability of the RE–SE difference to judge the adequacy of anti-nociception during other levels of stimulation. As with all monitors, data must be interpreted in clinical context.

D. M. Mathews
New York, USA
E-mail: dmathews@svemcny.org

Declaration of interest
D.M.M.’s work involves developing a closed-loop system for opioid delivery based on the RE–SE difference, and he has a patent application filed with this concept.

Editor—We thank Dr Mathews for his interest and comments on our article.1 In the last sentence of his letter, he summarized perhaps the essence of all research in medicine: data must be interpreted in clinical context. He pointed two problems related to our study. The first question was about the implication of the increasing SE value after the increasing RE value. Dr Mathews argues that the interpretation of the increasing SE value is difficult: one cannot tell if the increasing SE value is caused by EMG contamination or by cortical arousal. This is true if the anaesthetist is depending solely on the numerical values of Entropy. However, if the anaesthetist has the skill to interpret the biosignal on the monitor, EMG contamination and cortical arousal can be discerned. This is clearly demonstrated in Figure 4 of our article, where EMG appears on top of EEG burst suppression, and no EEG arousal is present. The second question raised was the patient motor response to intubation. The motor response was not recorded in our study and, as one might expect, several patients showed a clear motor response. As explained in our article,1 the anaesthetic regimen in our study was unusual and does not reflect our routine clinical practice. Our aim was to study the characteristics of the Entropy biosignal without confounding factors, such as neuromuscular blocking agents (NMBAs). In clinical practice, patients often receive NMBAs as part of their general anaesthesia. Therefore, when encountering SE increase during general anaesthesia, the anaesthetist will be denied the luxury of detecting a motor response to noxious stimulus. To make the interpretation of RE, SE, and RE–SE even more complicated, NMBAs have been found to suppress the RE–SE response, and this suppression is dose-dependent.2, 3 The strength of our study lies in the analysis of the original biosignal. Our study showed that the power spectra of EEG and EMG overlap significantly; therefore, the EEG-based depth of anaesthesia monitors cannot accurately separate EEG and EMG. In the presence of EMG, the resulting numerical value is always a combination of EEG and EMG. The 15 s averaged RE–SE difference may not be optimal in clinical practice. On the other hand, a phenomenon that lasts for <15 s cannot, in our opinion, be considered as a practical clinical monitoring tool. However, this was not the main issue. Instead, we wanted to demonstrate the physiology behind weird behaviour of a numerical index in a non-optimal clinical situation. This was done in the first scientific paper, where ‘the raw biosignal’ was analysed in detail together with the numerical indices. We wanted to do this due to the apparent confusion among clinicians, who do not have detailed understanding of neurophysiology, but see the abnormal index levels on their monitor screen. Our main purpose was not to judge the quality of Entropy monitoring concept in general. The conclusions made in our article were based on the results and statistics of our study, which, naturally, are sensitive to many factors, like the number of patients participating in the study. We regret that we were unable to convey our thoughts clearly. We fully agree that the augmentation of both hypnotic and anti-nociceptive medications is often the best way to treat increasing SE and RE values. We hope that our article has shed some light in understanding the role and importance of EMG in the EEG-based depth of anaesthesia monitoring. The quest for a depth of anti-nociception monitor continues.

A. J. Aho*
A. Yli-Hankala
L.-P. Lytytikäinen
V. Jäntti
Tampere, Finland
*E-mail: antti.j.aho@uta.fi
Diagnosis of vertebral canal haematoma by myelography and spiral computer tomography in a patient with an implantable cardioverter-defibrillator contraindicating magnetic resonance imaging

Editor—We report a case of a large vertebral canal haematoma (VCH) after the insertion of a thoracic epidural catheter in a 78-yr-old man. The patient presented for a laparoscopic sigmoid colon resection at Luebeck University Hospital, Germany. The patient had suffered a myocardial infarction in 2004 after which he received an implantable cardioverter-defibrillator. In addition, he was being treated for hypertension and diabetes mellitus type 2. The evening before surgery deltaparin 5000 IE was administered s.c. at 20:00 h. Before induction of anaesthesia, a thoracic epidural catheter was inserted at the T9/10 interspace with the patient sitting. Sufentanil 20 μg was injected into the epidural catheter and on commencing the operation, an epidural pump was started administering ropivacaine 0.2% at 6 ml h⁻¹. After uneventful surgery, the patient had normal lower limb motor function. At midnight, the patient received his first postoperative dose of deltaparin 5000 IE s.c., and at 04:00 h (postop day 1), he complained that he was unable to move his legs. The anaesthetist on call stopped the patient-controlled epidural analgesia (PCEA) pump and 3 h after that the motor block disappeared. The PCEA pump was then restarted at a reduced rate of 4 ml h⁻¹. During the morning of the second postoperative day, he developed a motor weakness of the right thigh. At 15:00 h, a neurological consult ruled out peripheral nerve damage and at 18:00 h radiological investigation was started. Owing to the implanted automated cardioverter-defibrillator, magnetic resonance imaging (MRI) was withheld, but a high definition spiral computer tomography (CT) did not reveal any pathology. Hence, a conventional, ascending myelography was undertaken which showed a significant bilateral narrowing of the contrast dye at level T6 to T10. The post-myelographic CT confirmed the suspected VCH ranging from T5/6 to T10/11 with complete compression of the subarachnoid space at level T7 to T9 (Fig. 1). At 22:00 h, the patient underwent emergency decompression laminectomy. After 3 weeks, all neurological symptoms had subsided. The low incidence of major complications after central neuraxial block has just been confirmed by a large national audit project.¹ The major issue leading to permanent patient harm in the past has been delay in the diagnosis, drainage of a haematoma, or both, as recently reviewed.² Notably, at present, there is a debate whether or not implanted cardiac devices prove a contraindication for MRI scanning³ and a safety protocol for non-cardiac and cardiac MRI in these patients has been proposed.⁴ However, if it has been decided that MRI scanning is unsafe for the patient, even a high definition spiral CT may be unable to detect even large VCHs and only conventional myelography with consecutive CT scanning will establish a diagnosis. In our case, the reduction of the PCEA infusion rate on the first postoperative day primarily led to a significant reduction in the motor block, which reoccurred on the second postoperative day. The neurological consultation and the radiological imaging took a considerable amount of time, before the patient finally underwent emergency decompression laminectomy. Despite that time

![Fig 1](http://bja.oxfordjournals.org/)  
Post-myelography CT confirms a VCH (H) ranging from T5/6 to T10/11, the air bubble within the haematoma might derive from epidural catheter injections.