Italy), which is a ‘hub’ centre in a hub-and-spoke regional trauma service of Tuscany.

In the population examined, the mortality rate was 11.5%, lower than the 18.9% in the German validating group of the original paper by Raum and colleagues, but EMTRAS calculations were still effective in predicting mortality (AUC 0.802). In our trauma population, EMTRAS lacked the efficacy shown in the German validating group. Our patients were similar to those in the German study with regard to characteristics of trauma, age, and gender, but our mortality rate was significantly lower, even considering that trauma severity inclusion criteria were similar in both groups (ISS $\geq 15$ in our population, ISS $\geq 16$ in German sample). We also found a slightly lower efficacy of EMTRAS in predicting mortality (AUC 0.802). In particular, we observed that when a score of 5 was reached, the mortality rate started to increase significantly. In fact, the mortality rate for the patients ($n=230$) included in the subgroup with an EMTRAS value between 0 and 4 was 4.78%, while in patients with EMTRAS $\geq 5$ ($n=94$), the mortality rate was 25.5%. The mortality rate for single EMTRAS score is shown in Table 1.

As previously mentioned, our trauma regional service is organized with hub-and-spoke centres. In this context, a cut-off value of EMTRAS could be useful in peripheral (spokes) hospitals, where a CT scan might not be immediately available, but where EMTRAS parameters normally are. This new characteristic of EMTRAS needs further validations, but it could be extremely useful in trauma care. EMTRAS could play an important role both to assess trauma severity and in helping clinicians in deciding for an early transfer of trauma patients to higher level hospitals.

### Table 1 Mortality rate for single EMTRAS score

<table>
<thead>
<tr>
<th>EMTRAS score</th>
<th>Patients</th>
<th>Mortality (%)</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>8</td>
<td>0</td>
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<tr>
<td>1</td>
<td>57</td>
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<tr>
<td>2</td>
<td>48</td>
<td>8.3</td>
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<tr>
<td>3</td>
<td>60</td>
<td>3.3</td>
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<tr>
<td>4</td>
<td>57</td>
<td>3.5</td>
</tr>
<tr>
<td>5</td>
<td>41</td>
<td>17</td>
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<td>2</td>
<td>50</td>
</tr>
<tr>
<td>11</td>
<td>1</td>
<td>100</td>
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</tbody>
</table>

### Declaration of interest

None declared.

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**Is sickle cell disease a possible risk factor for peripheral neuropathy after popliteal sciatic nerve block?**

Editor—Sickle cell disease (SCD) is a common form of clinically significant haemoglobinopathy. Although subclinical peripheral nerve involvement may be observed in SCD, no peripheral neurological impairment has been reported after nerve block in patients with SCD. Only one case of peripheral neuropathy has been reported after spinal anaesthesia. We report a case of peripheral neuropathy after a popliteal sciatic nerve block in a patient with SCD.

A 54-yr-old woman presented for right foot surgery. Her medical history included severe SCD (haemoglobin S-C). The results of clinical preoperative neurological examination were normal and a popliteal sciatic nerve block was planned for surgery. By mistake, a left popliteal ultrasound-guided block was performed. Ninety-five milligrams of ropivacaine with 75 $\mu$g of clonidine were injected incrementally, without report of pain or paraesthesia and no intraneural injection was detected. The patient was informed of this side error and a new block was administered on the right limb after the same protocol without any problem. Surgery was performed without tourniquet and intraoperative and immediate postoperative courses were uneventful.

On Day 1 after surgery, the patient reported persistence of sensory and motor block in the sciatic nerve area on both sides. She was discharged home at Day 4, without improvement at neurological examination. At Day 30, paraesthesia and weakness in both legs and feet persisted. At Day 40, electroneuromyography showed decreased bilateral amplitudes of potentially prolonged distal latencies, and slowing conduction velocities in the sciatic nerves at sensory and motor investigations (Fig. 1), consistent with a diagnosis of axonal loss and demyelinating neuropathy. At 10 weeks, electroneuromyography showed a significant improvement in these parameters, suggesting a diagnosis of moderate acute polyradiculoneuropathy, which was spontaneously regressive (Fig. 1). At 6 months, clinical examination showed significant improvement and 2 yr after surgery, neurological examination was almost normal.

Several mechanisms may explain the occurrence of neuropathy after single-shot peripheral nerve blocks. Intraneural injection of local anaesthetic, direct needle trauma, and
tourniquet are the main factors. In our patient, bilateral intraneural injection, direct nerve injuries, or both during the procedures seem unlikely. Indeed, pre-existing subclinical polyneuropathy has been reported as playing a role in the development of postoperative nerve injury.

To date, SCD has not been reported as a predisposing condition for neuropathy after peripheral nerve block. If central neuropathy is a common complication of SCD, symptomatic peripheral nerve damage is, in contrast, extremely rare. Neuropathy resulting from peripheral nerve infarction, such as complications of sickle cell vaso-occlusive crisis, seems uncommon. Recently, Okuyucu and colleagues conducted an electromyographic study in asymptomatic patients with the most severe form of SCD (SCD HbS/HbS). They found abnormalities in 19.6% of patients, with demyelination and axonopathy. It is possible that the severity of the vaso-occlusive crisis, associated with other factors such as hypoxaemia, acidosis, and dehydration, may play a role in the occurrence of peripheral neuropathy.

In our patient, the presence of latent preoperative neuropathy related to SCD may be hypothesized. A study in a rat model demonstrated that extraneural injection of ropivacaine in the nerve area results in substantial histological evidence of nerve injury with demyelination and Wallerian degeneration, with short-term regeneration. In our patient, injection of ropivacaine in close contact with popliteal nerves may therefore be responsible for the post-block demyelinating neuropathy.

In clinical practice, identifying undetected pre-existing subclinical neuropathy before peripheral nerve block administration in patients with SCD is mandatory. Thorough and well-documented preoperative neurological examination will help to define optimal anaesthetic protocol.

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Declaration of interest

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3 Tsen LC, Cherayil G. Sickle cell-induced peripheral neuropathy following spinal anesthesia for cesarean delivery. Anesthesiology 2001; 95: 1298–9
Accurate and easy to learn ultrasound sign to confirm correct tracheal intubation in cadaver model

Editor—The proper placement of a tracheal tube into the trachea should be systematically checked after each intubation to avoid morbidity related to hypoxaemia.1 No strategy is ideal,2 although ultrasound is promising. The study aimed to assess the performance of a new ultrasound sign for correct intubation, the increase in the antero-posterior diameter of the trachea during cuff inflation (Fig. 1), by an experienced sonographer and its learnability among novices after a short training course.

Freshly embalmed cadavers from people having donated their bodies to science were used in these studies, in accordance with French law (Collection number: DC-2008-137). Twenty freshly embalmed cadavers were randomly intubated correctly or not three times, leading to 60 assessments (30 tracheal intubations, 15 bronchial intubations, 15 oesophageal intubations). Correct tracheal intubation was defined as tube tip located into the trachea between the glottis and the carina. Incorrect intubation was defined as tube tip located either in the oesophagus or in a bronchus. All tube tip positions were checked with a laryngoscope and a disposable fibrescope (Ambu®, aScope™, Ballerup, Denmark). A piece of tissue was placed on the cadaver’s head to hide the tracheal tube so that the sonographer was blinded to the tube position. A second operator, with good experience in ultrasound and blinded to the position of the tube, determined if the tracheal tube was correctly positioned or not. Ultrasounds were performed using a 10 MHz ultrasound probe flat (Vivid-e™ General Electric, UK) according to the manufacturer’s instructions. The probe was placed on the longitudinal middle line on the neck between the sternum and cricoid cartilage as previously described.3 The operator looked for the better plan in this line to give a cross-section of the trachea. Then, the cuff of the tracheal tube was filled with an ultrasound contrast solution, a mixture of 1 ml of air and 15 ml of gelatin (Gelofusine 4%, B/BRAUN, Melsungen, Germany). Then, easiness to learn this method was assessed after a 20 min training course using 32 novices performing 64 assessments in eight cadavers in the same manner as in the observations made by experimented operator.

The trachea deformation during cuff inflation allowed identification of correct intubation by an experienced sonographer with a sensitivity of 90% (95% confidence interval (CI) 83–95), specificity of 97% (95% CI 92–99), positive predictive value of 97% (95% CI 91–99), and negative predictive value of 91% (95% CI 84–95). Using the ultrasound criteria, novices identified correct intubation with a sensitivity of 91% (95% CI 76–97), specificity of 94% (95% CI 80–98), positive predictive value of 94% (95% CI 79–98), and negative predictive value of 91% (95% CI 76–97). The performance of students was not significantly different from that of the experienced operator (P > 0.20 for all criteria). The method proposed to diagnose correct intubation was safe. Only one cuff rupture among 124 assessments (0.8%) was noted and all cuffs could be completely emptied at the end of the experiment. Large studies are needed.