and critical illness.\textsuperscript{4–6} Secondly, cortisol levels are rapidly dynamic and pulsatile by over 21 μg dl\textsuperscript{-1} (600 nmol litre\textsuperscript{-1}) within an hour.\textsuperscript{7} Therefore, a point measurement of 8 μg dl\textsuperscript{-1} alone does not tell one anything about what the cortisol level might be 1 h later. The measurement in this patient may simply be at the nadir of a pulse. There are numerous other gaps in our understanding of the physiology of the HPA axis during critical illness that makes diagnosis of corticosteroid insufficiency around this time almost impossible.

No test has been shown to accurately identify cortisol insufficiency, primarily as there is no accurate model for what constitutes ‘normality’. The inflammatory response is dynamic and it is likely that the glucocorticoid requirements vary throughout a patient’s illness. Glucocorticoid needs on day 1 may not be the same as day 3 or day 5, and indeed, this is one of the major gaps in our knowledge. The question of whether glucocorticoids in supra-physiological doses merely function as a non-catecholamine vasopressor is to be answered separately from trying to diagnose those with CIRCI, although much of the literature confuses these.

There are too many confounders in this case report to state that the patient’s poor haemodynamic state was as a result of glucocorticoid deficiency. Starting glucocorticoids on the third day post-insult may simply correlate with the resolution of the inflammatory response. Sterile, point insults causing a systemic inflammatory response appear to reach their peak level at around day 3 and then improve. This can be well seen after cardiac surgery, where the peak CRP and white cell count and the nadir in renal function all occur on or around day 3. The inflammation of a sterile systemic insult should not be confused with the ongoing inflammation of sepsis as can be seen from their vastly different outcomes. Point, sterile insults such as anaphylaxis\textsuperscript{8} and cardiac surgery\textsuperscript{9} have critical care survival rates of around 95%, whereas all-cause sepsis on UK dynamic and pulsatile by over 21

The bottom line is that attempts to identify, diagnose, and treat CIRCI will fail without high-quality studies of HPA axis pathophysiology and fortunately, the recent year has begun to see a move towards this.\textsuperscript{11} Until, we truly understand the nature and mechanisms of HPA axis function in critical illness, we are unlikely to be able to design tools to improve it.

**Declaration of interest**

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**It is 3 a.m. . . . do you know where your catheter tip is?**

Editor—Marhofer and colleagues\textsuperscript{5} have recently reported a high rate of perineural catheter dislocation (25% for femoral catheters and 5% for interscalene catheters). While their results should be confirmed in a clinical setting, the reported data are consistent with previous literature on secondary block failure.\textsuperscript{2–4} The finding that only 75% of femoral catheters placed under ultrasound guidance would be expected to provide effective analgesia at 6 h should raise substantial concerns, especially since they were performed in fit volunteers by expert practitioners. Actual patients may have a higher BMI and much longer indwelling time (typically 48–72 h or longer), both of which are likely to predispose to higher dislodgement rates. Since many institutions rely on femoral nerve catheters as the primary analgesic method for patients after total knee replacement, the data by Marhofer and colleagues\textsuperscript{5} suggest that a substantial proportion of these patients may not receive the presumed analgesic benefit from their catheters. Of note, no standards with regard to techniques of catheter insertion, their pharmacological management and securing to prevent dislocation have been established despite two decades of widespread use.\textsuperscript{5} There is no doubt that the technique used by this expert group is just one of many, and while the relative merits of various technical aspects could (and probably will) be debated, this is besides the more pressing point: if efforts to reduce secondary failure
are not successful, we may have to critically examine the expense and effort involved in using perineural catheters. Alternatively, perhaps it is time to re-focus our efforts to other means of delivering long-acting analgesia with nerve blocks, rather than continuing to deliberate on technical and equipment considerations. If current studies on the use of sustained-release local anaesthetics (e.g. liposomal bupivacaine) for nerve blocks yield positive results, a precise, ultrasound-guided single injection of such agents may be the future. 5 7

Declaration of interest

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Factors affecting perineural catheter dislocation rates

Editor—We read with interest the article by Marhofer and colleagues investigating dislocation rates of perineural catheters (PNCs) in a volunteer study. As described, PNCs are often positioned in clinical practice to facilitate perioperative anaesthesia and analgesia and thus, studies into their efficacy are often designed to assess perioperative pain. We agree that PNC dislocation is an important, yet poorly investigated, reason for failure of PNC anaesthesia or analgesia. We are however disappointed to find that, in our opinion, important information was omitted in the methodology of this otherwise well-designed study.

The technique described for insertion of the PNCs is an out-of-plane technique aiming to achieve passage under the fascia covering nerve structures as the needle tip entered the US beam. Catheters were advanced 3 cm beyond the tip of the needle and then retracted until appropriate spread of LA was seen around the nerve. The authors do not provide detail as to how far the catheter was in fact under the fascia in each case. We would be interested to see whether in the dislocated cases, the actual catheter length under the fascia was shorter than in the cases where dislocation did not occur. We would hypothesize that a catheter placed parallel to the femoral nerve with <2–3 cm under the fascia iliaca is likely to dislodge with full hip flexion, being pulled out of the fascial plane to lie superficial to the fascia as the authors describe. We would further suggest that a catheter inserted perpendicular to the nerve would be less likely to be dislodged with exercise because less pull is being exerted on the catheter itself. In 2010, Wang and colleagues showed that catheters positioned parallel to the nerve have a higher insertion failure rate than those positioned perpendicular to the nerve; interestingly, they also showed significantly quicker insertion times for perpendicular catheters.

The relatively low rate of dislocation reported for interscalene catheters may be because shoulder movement itself has less direct anatomical influence on neck structures; if subjects had been instructed to perform lateral neck flexion, thus exerting a direct pull on the catheter, the dislocation rate may well have been higher. Again we hypothesize that with an in-plane technique at the level of the supraclavicular trunks, positioning a catheter from posterior to the upper trunk to lie under the investing fascia may help to prevent dislocation. This topic would make for further interesting studies and we thank the authors for undertaking this novel work.

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