haemorrhage, a large air bubble at the apex of the left ventricle, and bubbles in the right coronary artery. Transthoracic echocardiography showed numerous hyper-refringency areas in the cardiac left chambers, the left ventricular outflow tract, and in the ascending aorta consistent with a pulmonary venous air embolism which entered the left side of the heart (Fig. 1). No neurological changes were to be noted and brain CT scan and the subsequent brain MRI showed no abnormality. Within 1 h, the patient was put on hyperbaric oxygen therapy (HBOT). The HBOT session included a period of compression at 4 atmospheres absolute (ATA) for 10 min, followed by a treatment period at 100% oxygen and 1.9 ATA for 60 min, and then a decompression period of 15 min. Immediately after HBOT, the patient experienced a complete cardiac recovery with the normalization of CT scan, electric, and echocardiographic parameters. Troponin I was undetectable initially but had increased to 5 ng ml$^{-1}$ 6 h later, confirming an acute ST-elevation myocardial infarction (STEMI). The intensive care unit stay was uneventful and the patient was discharged home within 48 h.

Pulmonary venous air embolism is a very rare event during transthoracic biopsy. This complication may happen when the needle punctures an airway and a pulmonary vein inducing a parenchymal haemorrhage which enters the left side of the heart. No neurological changes were to be noted and brain CT scan and the subsequent brain MRI showed no abnormality. Within 1 h, the patient was put on hyperbaric oxygen therapy (HBOT). The HBOT session included a period of compression at 4 atmospheres absolute (ATA) for 10 min, followed by a treatment period at 100% oxygen and 1.9 ATA for 60 min, and then a decompression period of 15 min. Immediately after HBOT, the patient experienced a complete cardiac recovery with the normalization of CT scan, electric, and echocardiographic parameters. Troponin I was undetectable initially but had increased to 5 ng ml$^{-1}$ 6 h later, confirming an acute ST-elevation myocardial infarction (STEMI). The intensive care unit stay was uneventful and the patient was discharged home within 48 h.

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Conflict of interest
None declared.

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doii:10.1093/bja/aer214

Preliminary results of a new ultrasound-guided approach to block the sacral plexus: the parasacral parallel shift

Editor—Surgical anaesthesia of the hip joint can be accomplished by a sacral plexus block combined with a lumbar plexus block in patients with severe cardiac morbidity who are not suitable for general or spinal anaesthesia. A parasacral approach to block the sacral plexus was originally described by Mansour and later improved by nerve stimulation guidance. Recently, an ultrasound-guided technique to block the sacral plexus has been described by Ben-Ari and colleagues. We describe a new ultrasound-guided approach to block the sacral plexus.

The transducer is aligned between the posterior superior iliac spine (PSIS) and the midpoint of the line connecting the PSIS and the greater trochanter (Fig. 1a) and the iliac bone line identified (Fig. 1a). The transducer is moved inferomedially with a parasacral parallel shift (PSPS) (Fig. 1c). When the transducer beam arrives at the sciatic notch, the ultrasonographic continuity of the iliac bone line is interrupted...
(Fig. 1D). This is exactly where the sacral plexus exits the pelvis. The transducer is tilted slightly caudad and the hyperechoic sacral plexus is visualized between the sacrum and the ischial bone and beneath the triangular piriformis muscle. The needle is advanced in-plane from the lateral end of the transducer until the needle tip touches the sacral plexus (Fig. 1D). The identity of the sacral plexus is confirmed by nerve stimulation with a sciatic motor response in the range of 0.3–0.5 mA. Then 20 ml of ropivacaine 0.5% is injected with sonographic observation of perineural spread as the endpoint with reposition of the needle tip if necessary using solely ultrasonographic guidance.

Successful surgical anaesthesia was defined as no need for conversion to general or spinal anaesthesia but allowing awake sedation with eye opening and oriented verbal response to speech and spontaneous respiration.

Seven consecutive patients with severe cardiac comorbidity (ASA III and IV) undergoing hip fracture surgery were included in the pilot study. The piriformis muscle and the sacral plexus could be identified ultrasonographically in all seven patients without reposition of the needle tip to obtain an appropriate motor response. Successful surgical anaesthesia was accomplished in all seven patients. Three patients required peroperative awake sedation with propofol for anxiolysis. No patients needed peroperative vasopressors to maintain haemodynamic stability. No nerve injuries, haematomas, or rectal perforation were observed.

For lumbar plexus block, 20 ml of ropivacaine 0.5% is injected as previously described. The ultrasound scanning was performed with an M-Turbo ultrasound machine (Sonosite, Bothell, WA, USA) using a 2–5 MHz curved array transducer (Sonosite).
We recognize that an ultrasound-guided parasacral approach to block the sacral plexus has been described. However, that approach relies on searching the target sacral plexus below the PSIS between the sacrum and the ischial bone. With our PSPS approach, it is easy to identify the characteristic iliococcygeal bone contour ultrasonographically and ‘walk off’ the ischiatic bone with the ultrasound beam to identify the target sacral plexus exactly at the pelvic exit accurately. Whether the PSPS technique can compare favourably with the success rate of using nerve stimulation alone remains to be investigated in a prospective randomized clinical trial.

In conclusion, the PSPS approach indicates an effective and easy roadmap to target the sacral plexus. When combined with a paravertebral lumbar plexus block, the PSPS allows various types of hip fracture surgery in elderly ASA class III–IV patients with good success.

**Conflict of interest**

None declared.

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**Early primary cardiac graft failure and combined heart–liver transplantation: need for an uncommon double bypass**

Editor—A 32-yr-old man was referred for combined heart and liver transplantation (CHLT) because of recurrent severe right heart failure and refractory ascites with biopsy-proven cirrhosis. Heart transplantation was first performed using the standard method of our cardiac surgery centre. Ischaemic time was 135 min. Despite the use of inotropes (epinephrine) and inhaled vasodilators (nitric oxide), mean arterial pressure (MAP) remained below 50 mm Hg because of failed cardiac contraction. A central extracorporeal membrane oxygenation (ECMO) (Carmedics®, Medtronic France SA, Rueil Malmaison, France) support was used. A 130 beats min⁻¹ sinus rhythm and left atrial pressure above 10 mm Hg could thus be maintained. Epinephrine and fluid loading were adjusted to maintain MAP and central venous pressure (CVP) at 70–80 and 8–15 mm Hg, respectively.

The liver procedure started after closure of the sternum. Once the liver was completely freed, flushed heparin-coated tubes were inserted in the inferior mesenteric vein, inferior vena cava, and axillary vein and connected to a double-limb heparin-coated venovenous bypass (VVB) (Bio-Medicus®, Medtronic France SA). After caval and portal cross-clamping, the liver together with the retrohepatic cava was explanted. The graft with its caval vein was then implanted and end-to-end caval anastomoses were performed. VVB flow was set to keep the ECMO output between 3 and 4 litre min⁻¹. VVB was withdrawn after portal revascularization.

Fluid, 2000 ml 4% human albumin, and epinephrine administration were set to maintain both CVP above 8 mm Hg and MAP above 70 mm Hg. International normalized ratio (INR) ranged between 2.4 and 4. Anticoagulants and blood products were not required. During the hepatic artery and bile duct anastomoses, MAP decreased and CVP increased leading to liver congestion. An epinephrine bolus restored MAP but had no effect on CVP and liver congestion. An increment in the ECMO outflow corrected this immediately. Cold and warm ischaemic times of the liver were 11 h and 52 min, respectively. Overall transfusion amounts during CHLT were: 8 red blood pack (RBP), 4 recovered autologous unit (RAU), 23 fresh frozen plasma (FFP), 22 units of platelet (PLT), and 3 g fibrinogen. Twelve thousand units per day of heparin were administered after POD2, while INR spontaneously decreased below 2.5. ECMO was discontinued on POD15. The patient was discharged to home at POD45 with normal liver and renal function, and a 45% left ventricular ejection fraction.

CHLT is a rare multidisciplinary challenge. This case illustrates that sequential CHLT can be achieved despite early primary cardiac graft failure, using two different bypass circuits. ECMO provided the equivalent haemodynamic and metabolic stability to that obtained with cardiopulmonary bypass described for simultaneous implantation of both organs, but without heparin requirement. In our case, despite transient heart graft failure, ECMO allowed continuous and prolonged support of hepatic and renal functions (Table 1). Since the first goal of VVB was to ensure the ECMO outflow, attendant management of VVB and ECMO is simple: any decrease in the ECMO outflow has to be corrected first by an increase in the VVB outflow, and, if ineffective, by fluid loading. If MAP decreases despite adequate ECMO outflow, vasoconstrictor drugs should be given.