Use of a stimulating catheter for femoral nerve block

Editor—In their paper, Jack and coworkers\(^1\) retrospectively analysed the effectiveness of femoral catheters for pain relief after knee replacement surgery and concluded that stimulating catheters are not advantageous in terms of pain levels and opioid consumption compared with conventional catheters.

The rationale for using stimulating catheters is based on the assumption that catheter tips are directed close to nerves. The question arises whether nerve proximity is really needed for the femoral nerve to be blocked effectively in routine clinical use. Several reasons argue against this necessity, particularly when larger volumes (40 ml) of local anaesthetics are used.

Firstly, anatomical review suggests that, once the iliac fascia is penetrated, there are no relevant diffusion barriers for local anaesthetics. Secondly, catheters threaded 16–20 cm from the inguinal level radiographically deviated in 77% of cases but were as effective in motor blockade of the femoral nerve, and only marginally less effective in sensory blockade of the femoral nerve, compared with radiographically well placed catheters.\(^2\) Thirdly, iliac fascia blocks performed without any nerve stimulation are as effective as femoral nerve blocks, in both children\(^3\) and adults,\(^4\) suggesting no clinically meaningful reason for placing catheter tips in close proximity to the femoral nerve. Assuming that the authors used the same insertion depth into a ‘femoral nerve sheath’ for both catheter types, that is 3–5 cm, we think that deviation rate would be much lower than those reported by Capdevila. Consequently, one would not expect a relevant difference whether an electrical proximity of the catheter tip is present or not. In this context, it is unclear why the authors accepted higher current thresholds for the stimulating catheters (\(\leq 1\) mA) compared with stimulating needles (\(\leq 0.5\) mA).

When using ultrasound, the tip position can suggest proximity even though sufficient nerve stimulation is not achieved, injection of local anaesthetic usually produces a clinically effective block.

Following this reasoning, the hypothesis that lower morphin consumption and pain scores could be expected in their study is questionable.

Pham-Dang and colleagues\(^5\) demonstrated that brachial plexus block catheters, which could not be stimulated, were ineffective and radiographically misplaced. In three sciatic catheters no stimulation was possible, but catheters functioned well, and in the radiography a correct position was present. Hence, it was impossible to stimulate an apparently correct catheter. The inability to stimulate clinically effective catheters in the report by Pham-Dang mainly is a result of using saline for catheter placement.

We agree with the authors, that well designed studies should to be done to prove the superiority of stimulating catheters, but not for the femoral nerve.

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Editor—We would like to thank Drs Birnbaum and Volk for raising a number of interesting points. The first is that, considering the anatomy and the good results which may be obtained by a blind technique,\(^5\) the results of our study were to be expected. There is some logic to this assertion, and yet so many self-evident matters have not stood up to closer investigation that we consider our study to be useful in the evolving debate about stimulating catheters.

Our catheters were inserted, as they suggest, 3–5 cm, which we consider to be a good balance between preventing dislocation on the one hand and deviation on the other. We accepted a stimulating value through the catheter as high as 1 mA as this was recommended to us by the manufacturers as safe and effective. Placement of the stimulating catheter is not always easy, and repeated attempts are often necessary. In a recently published study, the catheter had to be redirected up to 20 times.\(^5\) Repeated attempts to get low values can be uncomfortable for the patient and theoretically could increase the chance of nerve damage. In most of our cases, we were able to use 0.3–0.5 mA of current, but we accepted values up to 1 mA. It is unclear whether the results from the stimulating catheter are improved by only accepting lower stimulating values.

There are still many uncertainties about the value of stimulating through the catheter. We have had cases with excellent stimulation through the catheter and no block, and there are reports of no stimulation but an excellent block.\(^5\) Such experiences undermine the need for meticulous catheter placement and of course raise question marks about the whole principle of stimulating catheters. However, it is too early to write off the stimulating catheter as a white elephant. There are studies showing that the placement of stimulating catheters is on average more accurate.\(^7\) The clinical effectiveness of the stimulating catheter is not yet clear but we do not agree with their assertion that there is no need for further investigation of stimulating catheters for the femoral nerve. Perhaps the most important comment by Birnbaum and Volk concerns the use of ultrasound to confirm the catheter position. Nerve location by ultrasound will surely become an important tool in regional anaesthesia and may eventually
make the whole discussion about the stimulating catheter academic.

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Hyperinsulinaemic normoglycaemic clamp in coronary artery surgery

Editor—We congratulate Visser and colleagues on applying glucose–insulin–potassium (GIK) therapy using a hyperinsulinaemic normoglycaemic clamp. Their study confirms our findings that the clamp technique is an effective, and to date the only, method of maintaining normoglycaemia in patients undergoing coronary artery bypass grafting surgery. In addition, they demonstrated, for the first time in this population, the attenuation of systemic inflammation with perioperative GIK therapy. More importantly, this effect was prolonged beyond the time of insulin administration providing a rationale for insulin therapy to be started before the stress of surgery and cardiopulmonary bypass (CPB).

Several issues merit further comment. It is unfortunate that when initiating the clamp the authors were unable to prevent hypoglycaemia that is, a blood glucose <3.0 mmol litre⁻¹. Perhaps, if there had been more frequent measuring of blood glucose this complication may not have occurred. In our protocol, using an insulin dose three times that has been used in the present study, we avoided hypoglycaemia in diabetic and non-diabetic patients by measuring the blood glucose every 5 min at the start of the clamp.

We agree with the authors in stressing the importance of iatrogenic hyperglycaemia resulting from exogenous glucose administration. This is true of the metabolically unsupported control group (D3W at 30 ml h⁻¹) and may also be true of the treatment or clamp group. Although not stated in the manuscript, we assume that the five patients excluded, due to ‘insufficient insulin therapy during CPB’, belonged to the treatment group. If so, we suspect these patients were rendered hyperglycaemic secondary to the administration of additional exogenous glucose, as for example by transfusing blood products. Packed red blood cells and fresh frozen plasma contain high concentrations of glucose that are typically >20 mmol litre⁻¹. In anticipation of administering additional glucose, using a higher dose of insulin and therefore a higher glucose infusion rate (GIR), as proposed in our protocol, provides a larger buffer for adjusting the GIR and maintaining normoglycaemia.

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Editor—We thank Drs Carvalho, Leung and Schricker for their comment on our study. It is true that we did not succeed in preventing hypoglycaemia, (blood glucose <3 mmol litre⁻¹) in all our patients at all times. Glucose levels between 2.2 and 3.0 mmol litre⁻¹ were found in 7 out of 175 blood samples (4%) obtained during the first 5 h of the study. Their comment that more frequent glucose measurements taken at the start of the clamp may have prevented these individual episodes of hypoglycaemia is, therefore, gratefully acknowledged. However, measuring plasma glucose at 5 min intervals, as suggested, implies the continuous use of a blood gas analyzer, equipped with a glucose electrode. This is not realistic for the routine use of GIK, as each measurement takes ~5 min. The use of a faster ‘finger-stick’ glucose measuring device, as used by Carvalho and colleagues, is, in our opinion, not acceptable as an alternative, due to the well-known inaccuracy of those devices. The future availability of continuous glucose measuring sensors can potentially solve those problems. None of our patients showed effects after operation, that could be attributed to the short episodes of hypoglycaemia. Regarding the importance of iatrogenic hyperglycaemia, there appears to be a consensus among us. In addition, we agree that glucose, which is present in packed blood cells, may contribute to elevated plasma glucose levels. However, their assumption that five GIK patients were excluded from final analysis in our study because of hyperglycaemia following CPB is not correct. Those patients were excluded due to protocol violations when the insulin and glucose infusions were discontinued by mistake at the onset of CPB.