bally to the patient, and stated clearly in the written consent form. It is only common sense that if the investigator has doubts about the patient's ability to comprehend, recruitment for study should not proceed.

My letter was intended not so much as to what information is required in an informed consent, but rather how the informed consent is obtained, especially from day-care surgery patients and in situations where there is a language barrier. In my case, the surgeon was a participating investigator who was in a good position to obtain informed consent. However, in other situations, attending surgeons, who are not participating investigators, may not be suitable for explaining the nature and risks involved. The move by many hospitals to preadmission clinics and same-day surgery creates a situation in which the investigator-anaesthetist may not be the one who sees the patient in the preadmission clinic. The non-investigator-anaesthetist who sees the patient in the preadmission clinic, and may not know all the details of the study, is not the appropriate person to obtain informed consent. Is it adequate for him/her to warn the patients regarding the study, as he/she may not be able to answer all the questions raised by the patient? Is it fair to the patient if the consent is obtained on arrival at the clinic, or minutes before surgery, even though the information includes the fact that refusal to participate would not jeopardize the quality of care? When informed consent is obtained through an interpreter, how can one be sure that none of the essential information is lost through the translation?

Clinical reports often simply state that "the study has been approved by the ethics committee and informed consent obtained from patients." However, it is the manner in which the informed consent is obtained in these situations that has not been addressed.

David H.W. Wong MB BS FRCPC
Department of Anaesthesia
University of British Columbia
Vancouver

PCA in burn injuries: the subcutaneous route

To the Editor:

Adequate pain control in burn injuries can be problematic due to wide variations in analgesic requirements.[1] In a retrospective study, 35 hospitalized patients suffering from acute burn injuries were assessed for three days after their injuries. A winged needle was inserted subcutaneously (*sc*) at a site distant from the burns. Morphine was given as an on-demand bolus dose of one milligram (mg) with a lockout time of six minutes. A background infusion of morphine (1 $mg \cdot hr^{-1}$) was used routinely, but was discontinued if morphine requirements decreased below 20 $mg \cdot day^{-1}$.

Every four hours, the respiratory rate, sedation score, pulse rate, and blood pressure were recorded. Each day, an assessment was made of each patient, a database form was filled in, and any complications were noted. The % body surface area burnt was recorded. Pain was assessed daily by the patient using a ten-point visual analogue pain scale. The quality of analgesia was also indirectly assessed by determining the ratio of successful to unsuccessful demands, the proviso being the higher the ratio the better the analgesia (or the less anxious the patient).[2,3]

Vomiting was seen in only one patient. No oversedation or respiratory depression occurred. Localized swelling and induration at the sc infusion site was found in two patients. The sc site was easily resited. A positive relationship between pain scores and body surface area burnt has previously been reported.[4] No such relationship occurred in this study.

However, sc PCA (with morphine) was found to be a safe and effective way of controlling burn pain. There is often a paucity of readily accessible veins in patients with burn injuries. The sc route has the advantage of reducing time taken to maintain a dedicated intravenous line, and is easy to initiate and maintain. To the best of our knowledge, it is the first time that the sc route has been used in this way.

Edward A. Shipton MBChB DA MMed FFA MD
Harold S. Minkowitz MBBCH Dip Data
Pieter J. Becker PhD
Hillbrow Hospital
Johannesburg, South Africa

REFERENCES

1 *Choiniere M, Grenier R, Paquette C.* Patient-Controlled analgesia: a double-blind study in burn patients. Anaesthesia 1992; 47: 467–72.

2 *Lehman KA.* Practical experience with demand analgesia for postoperative pain. *In*: Harmer M, Rosen M, Vickers MD (Eds.). Patient Controlled Analgesia. London: Blackwell, 1985; 134–9.

3 *De Kock M, Pinchon G, Scholtès JL.* Intraoperative clonidine enhances postoperative morphine patient-controlled analgesia. Can J Anaesth 1992; 39: 537–44.

4 *Atchison NE, Osgood PF, Carr DB, Szyfelbein SK.* Pain during burn dressing change in children: relationship to burn area, depth, and analgesic regimens. Pain 1991; 47: 41–5.

Hyperkalaemia after warm heart surgery

To the Editor:

We read with interest the case report of severe hyperkalaemia following warm heart surgery.[1] In our experience of over 900 cases of warm heart surgery we have

never encountered hyperkalaemia of this degree. We believe this to be due to several points. Firstly, the authors do not mention the *total* dose of potassium administered. In our initial experience we measured all potassium data, the total amount delivered being 37.12 ± 13.7 mmol · L^{-1} which produced a potassium concentration of 5.4 ± 0.8 mmol · L^{-1} at the end of cardiopulmonary bypass. Next, we question the choice of antegrade cardioplegia delivery for mitral valve replacement. Retraction of the left atrium for valve exposure leads to distortion and incompetence of the aortic valve and thus to inappropriate delivery of cardioplegia to the coronary arteries and a very "bloody" surgical field. Retrograde delivery techniques are now well established,[3-5] allowing uniform distribution of flow in an uninterrupted manner. Antegrade induction with retrograde delivery of cardioplegia is now our preferred delivery strategy even during coronary artery surgery.

It is not clear what type of electromechanical activity returned during the valve procedure. A pattern of "creeping" waves rather than rhythmic contractions is probably related to calcium overload related to potassium-induced membrane depolarisation,[6] in which case an increase in potassium load is counter-productive. Menasche[7] and Lessana[8] have used the natural calcium-antagonist magnesium in order to circumvent this problem and maintain effective, sustained asystole. Although we have not used magnesium in our perfusate we believe it may be useful in cases where sustained asystole is difficult to maintain, e.g., hypertrophied ventricles.

Although impaired renal function does reduce the effectiveness of the kidneys in clearing potassium during cardiopulmonary bypass, this can be improved with frusemide. Should there be a poor response to this manoeuvre, or in anephric patients, haemofiltration during bypass is effective in maintaining potassium in the physiological range.

Finally, the importance of regular monitoring of serum potassium concentration during the procedure should be stressed. Early intervention with either a diuretic or haemofiltration enables the anaesthetist to prevent hyperkalaemia of the degree reported without necessarily prolonging cardiopulmonary bypass.

C. Ip Yam
M.A. Fox
B.M. Fabri
The Cardiothoracic Centre
Liverpool, England

REFERENCES

1 *Kao Y, Mian T, Kleinman S, Raez GB.* Hyperkalaemia: a complication of warm heart surgery. Can J Anaesth 1993; 40: 67–70.

2 *Page R, Sharpe D, Bellamy C, Rashid A, Fabri B.* Normothermic arrest using continuous hyperkalaemic blood: initial experience. Eur J Cardio-thorac Surg 1992; 6: 461–8.

3 *Bolling S. Flaherty J, Bulkley B, Gott V, Gardner T.* Improved myocardial preservation during global ischaemia by continuous retrograde coronary sinus perfusion. J Thorac Cardiovasc Surg 1983; 86: 659–66.

4 *Partington M, Acar C, Buckberg G, Julia P, Kofsky E, Bugyi H.* Studies in retrograde cardioplegia. I. Capillary blood flow distribution to myocardium supplied by open and occluded arteries. J Thorac Cardiovasc Surg 1989; 97: 605–12.

5 *Solorzano J, Taitelbaum G, Chiu R.* Retrograde coronary sinus perfusion for myocardial protection during cardiopulmonary bypass. Ann Thorac Surg 1978; 25: 201–8.

6 *Steenbergen C, Murphy E, Watts J, London R.* Correlation between cytosolic free calcium, contracture, ATP, and irreversible ischemic injury in perfused rat heart. Circ Res 1990; 66: 135–46.

7 *Menasche P, Peynet J, Touchout B, et al.* Normothermic cardioplegia: is aortic cross-clamping still synonymous with myocardial ischaemia? Ann Thorac Surg 1992; 54: 472–8.

8 *Lessana A, Romano M, Singh A, et al.* Beyond cold cardioplegia. Ann Thorac Surg 1992; 53: 666–9.

REPLY

We appreciate the interest and comments expressed by Drs. Yam, Fox and Fabri. Their response made us realize that the practice of "Warm Heart Surgery" is indeed widespread and our experience in management of the hyperkalaemic patient might be helpful for other anaesthetists in their patient care.[1]

Since the introduction of warm heart surgery, the practice of retrograde cardioplegia infusion has gradually increased. Its use has been adopted widely, especially in valvular procedures. Indeed, antegrade cardioplegia administration for initial induction, followed by retrograde cardioplegia maintenance, has become the most common cardioplegia management technique in our hospital today.

In our original report, we mentioned that patients received a total dose of potassium chloride of 90 meq. Drs. Yam, Fox and Fabri reported that in their experience, patients received a total potassium dose of 37.12 ± 13.7 "mmol · L^{-1}" [sic], "mmol · L^{-1}" presumably means "meq," resulting in a post-cardiopulmonary bypass potassium concentration of 5.4 ± 0.8 mmol · L^{-1}. From their data, it appears that up to 50 meq of potassium infused for myocardial preservation can be administered safely.

In our patient, the electromechanical activity that returned during cardiopulmonary bypass was rhythmic in nature. The term "creeping" waves is not familiar to us. During hyperkalaemia cardioplegia induced diastolic standstill, intracellular calcium concentration slowly increased during ischaemia. Left ventricular contracture could also be demonstrated.[2] *However, this phenomenon is by no means unique to high potassium solution induced myocardial standstill. In both a high magnesium solution induced myocardiam arrest, or calcium channel blocker*