Leading Article

Diuretic-induced hypokalaemia and surgery: much ado about nothing?

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There is physiological and clinical evidence that has given rise to concern about the risk of arrhythmias in patients with hypokalaemia. It is known that hypokalaemia, when induced experimentally, potentiates arrhythmias by altering the intracellular/extracellular ionic ratio.¹ The resulting increase in resting transmembrane potential² and the reduction in atrio-ventricular conduction³ facilitate both re-entrant and automatic arrhythmias.⁴ In clinical practice the characteristic electrocardiogram (ECG) changes of hypokalaemia⁵ are seen in up to 80% of patients with serum potassium less than 2.7 mmol/l⁶ and ectopic beats are common.⁷ Hypokalaemia causing significant arrhythmias is less well documented, and occurs more often when hypokalaemia is severe or acute.^{8,9}

The only well-documented risk of arrhythmias due to mild hypokalaemia is seen in hypokalaemic patients taking digoxin.¹⁰ There is also circumstantial evidence that hypokalaemia predisposes to arrhythmias during acute myocardial infarction.^{11–14} However, it has never been clearly demonstrated that diuretic-induced hypokalaemia, *per se*, increases the risk of clinically important arrhythmias.^{15–18} The Multiple Risk Factor Intervention Trial Research Group¹⁹ found no association between coronary artery disease mortality and hypokalaemia and the Medical Research Council mild hypertension trial²⁰ found no significant association between the number of ventricular extra-systoles and serum potassium in patients on long-term thiazides. There is only one small randomized crossover study which found more frequent ventricular extra-systoles and myocardial instability when patients were taking thiazides rather than potassium-sparing diuretics.²¹

The risk of chronic preoperative hypokalaemia due to diuretics remains controversial because of

differences in perception of attributable risk for intraoperative arrhythmias. Moreover, serum potassium is not static intraoperatively. Serum potassium is reduced by catecholamines,²² by hyperventilation which results in a respiratory alkalosis^{23,24} and by the action of some anaesthetic agents.²⁵ Other anaesthetic drugs such as suxamethonium can also cause hyperkalaemia.²⁶

Arrhythmias during anaesthesia and surgery, even in normokalaemic patients, are common,^{27,28} although only a small proportion of these arrhythmias are serious.²⁹ Certain patient characteristics, such as cardiac disease, poor general medical condition and age over 70 years, are associated with increased risk of arrhythmia and cardiac complications in patients undergoing non-cardiac operations under general anaesthesia.³⁰ Laryngoscopy, intubation, anaesthetic agents, metabolic changes produced by controlled ventilation and surgical manoeuvres may also potentiate arrhythmias.³¹ As a consequence of these other risk factors, and the frequency with which intraoperative arrhythmias are seen, it has been calculated that a controlled study of 8,600 patients would be needed to detect a 50% increase in mortality due to intraoperative arrhythmias attributable to hypokalaemia.³²

There are only two controlled studies which address the issue of the risk of preoperative hypokalaemia for intraoperative arrhythmia and compare outcome and events in anaesthetized hypokalaemic patients with normokalaemic patients. In the first study published in 1985 from Las Vegas,³³ there was no significant difference in incidence of arrhythmia between 88 normokalaemic (serum potassium; 3.5–5 mmol/l) and 62 chronically hypokalaemic patients (serum potassium; 2.6–3.4 mmol/l). The only risk factor associated with intraoperative dysrhythmia was the presence of preoperative dysrhythmia ($P < 0.0082$) and there were no intraoperative arrhythmias requiring treatment. The second larger study reported a series of patients undergoing cardiac or vascular surgery,³⁴ and hence considered to be at

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greater risk of intraoperative arrhythmia than the patients in the Las Vegas study. There was, however, no significant difference in either severity or frequency of intraoperative arrhythmias between the 255 normokalaemic patients (serum potassium greater than 3.6 mmol/l), the 152 hypokalaemic patients (serum potassium; 3.1–3.5 mmol/l) and the 40 markedly hypokalaemic patients (serum potassium less than 3.0 mmol/l). Hypokalaemia did not even increase the incidence or severity of ectopy in this study, although frequent and complex ventricular ectopics were more common in patients in congestive cardiac failure or taking digoxin. In both studies the groups were matched for age, cardiac disease and treatment, operative factors and hypoxaemia.

The conclusions from this data were that preoperative hypokalaemia was not a risk factor for intraoperative arrhythmias, which were related to other patient characteristics and preoperative factors. Whilst a low serum potassium may be a

marker of increased sympathetic activity or metabolic alkalosis, chronic asymptomatic preoperative hypokalaemia is not a risk factor for intraoperative arrhythmias.

The consensus minimum recommended preoperative serum potassium, at which routine or emergency anaesthesia should be safely undertaken in the absence of cardiac disease or treatment with digoxin, has been 3.5 mmol/l.³⁵ In the light of these two controlled studies^{33,34} this is overcautious. A chronic serum potassium 3 mmol/l or above should not alter preoperative and anaesthetic management in patients at low risk of cardiac complications of surgery and anaesthesia and chronic asymptomatic hypokalaemia as low as 2.5 mmol/l may even be acceptable in these patients. McGovern³⁶ in 1988 stated that hypokalaemia in the range 3–3.5 mmol/l was not an indication for potassium supplementation or postponement of operation and we strongly endorse this view.

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