

## Research

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**Severe electrolyte disorders following cardiac surgery: a prospective controlled observational study**Kees H Polderman<sup>1</sup> and Armand RJ Girbes<sup>2</sup><sup>1</sup>Senior Consultant in Intensive Care Medicine, Department of Intensive Care, VU University Medical Center, Amsterdam, The Netherlands<sup>2</sup>Professor of Intensive Care Medicine, Department of Intensive Care, VU University Medical Center, Amsterdam, The NetherlandsCorresponding author: Kees H Polderman, [k.polderman@tip.nl](mailto:k.polderman@tip.nl)

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*Critical Care* 2004, **8**:R459-R466 (DOI 10.1186/cc2973)This article is online at: <http://ccforum.com/content/8/6/R459>© 2004 Polderman *et al.*, licensee BioMed Central Ltd. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/2.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is cited.**Abstract**

**Introduction** Electrolyte disorders are an important cause of ventricular and supraventricular arrhythmias as well as various other complications in the intensive care unit. Patients undergoing cardiac surgery are at risk for development of tachyarrhythmias, especially in the period during and immediately after surgical intervention. Preventing electrolyte disorders is thus an important goal of therapy in such patients. However, although levels of potassium are usually measured regularly in these patients, other electrolytes such as magnesium, phosphate and calcium are measured far less frequently. We hypothesized that patients undergoing cardiac surgical procedures might be at risk for electrolyte depletion, and we therefore conducted the present study to assess electrolyte levels in such patients.

**Methods** Levels of magnesium, phosphate, potassium, calcium and sodium were measured in 500 consecutive patients undergoing various cardiac surgical procedures who required extracorporeal circulation (group 1). A total of 250 patients admitted to the intensive care unit following other major surgical procedures served as control individuals (group 2). Urine electrolyte excretion was measured in a subgroup of 50 patients in both groups.

**Results** All cardiac patients received 1 l cardioplegia solution containing 16 mmol potassium and 16 mmol magnesium. In addition, intravenous potassium supplementation was greater in cardiac surgery patients (mean  $\pm$  standard error:  $10.2 \pm 4.8$  mmol/hour in cardiac surgery patients versus  $1.3 \pm 1.0$  in control individuals;  $P < 0.01$ ), and most (76% versus 2%;  $P < 0.01$ ) received one or more doses of magnesium (on average 2.1 g) for clinical reasons, mostly intraoperative arrhythmia. Despite these differences in supplementation, electrolyte levels decreased significantly in cardiac surgery patients, most of whom (88% of cardiac surgery patients versus 20% of control individuals;  $P < 0.001$ ) met criteria for clinical deficiency in one or more electrolytes. Electrolyte levels were as follows (mmol/l [mean  $\pm$  standard error]; cardiac patients versus control individuals): phosphate  $0.43 \pm 0.22$  versus  $0.92 \pm 0.32$  ( $P < 0.001$ ); magnesium  $0.62 \pm 0.24$  versus  $0.95 \pm 0.27$  ( $P < 0.001$ ); calcium  $1.96 \pm 0.41$  versus  $2.12 \pm 0.33$  ( $P < 0.001$ ); and potassium  $3.6 \pm 0.70$  versus  $3.9 \pm 0.63$  ( $P < 0.01$ ). Magnesium levels in patients who had not received supplementation were  $0.47 \pm 0.16$  mmol/l in group 1 and  $0.95 \pm 0.26$  mmol/l in group 2 ( $P < 0.001$ ). Urinary excretion of potassium, magnesium and phosphate was high in group 1 (data not shown), but this alone could not completely account for the observed electrolyte depletion.

**Conclusion** Patients undergoing cardiac surgery with extracorporeal circulation are at high risk for electrolyte depletion, despite supplementation of some electrolytes, such as potassium. The probable mechanism is a combination of increased urinary excretion and intracellular shift induced by a combination of extracorporeal circulation and decreased body temperature during surgery (hypothermia induced diuresis). Our findings may partly explain the high risk of tachyarrhythmia in patients who have undergone cardiac surgery. Prophylactic supplementation of potassium, magnesium and phosphate should be seriously considered in all patients undergoing cardiac surgical procedures, both during surgery and in the immediate postoperative period. Levels of these electrolytes should be monitored frequently in such patients.

**Keywords:** cardiac surgery, electrolyte disorders, extracorporeal circulation, hypokalaemia, hypomagnesaemia, hypophosphataemia, hypothermia, magnesium, potassium, phosphate

## Introduction

Electrolytes such as potassium, magnesium, calcium and phosphate play important roles in cellular metabolism and energy transformation, and in the regulation of cellular membrane potentials, especially those of muscle and nerve cells. Depletion of these electrolytes can induce a wide range of clinical disorders, including neuromuscular dysfunction and severe arrhythmias. The risk for these disorders increases significantly when more than one electrolyte is deficient, and increases still further in the presence of ischaemic heart disease [1].

It is well known that hypokalaemia can induce cardiac arrhythmias (especially in patients with ischaemic heart disease and left ventricular hypertrophy), and that it is associated with other adverse effects such as muscle weakness, rhabdomyolysis, renal failure and hyperglycaemia. Thus, the importance of regulating potassium levels is well recognized in most intensive care units (ICUs) and potassium levels are measured frequently, especially in patients with cardiovascular disease. In contrast, electrolytes such as magnesium, calcium and phosphate are measured far less frequently. However, a large number of clinical and *in vitro* studies have provided strong evidence that depletion of magnesium, phosphate and calcium can adversely affect outcome, especially in patients with cardiovascular disease. Several studies have been published that link hypomagnesaemia to increased mortality in the ICU [2,3] and in the general ward [3]. Hypomagnesaemia is also associated with adverse outcomes in patients with unstable angina or myocardial infarction [4-7], and administering magnesium has been shown to reduce mortality and infarction size in these patients [8-11]. Hypomagnesaemia can cause cardiac arrhythmias, neuromuscular irritability, hypertension and vasoconstriction (including constriction of coronary arteries), as well as metabolic effects including decreased insulin sensitivity [12,13], all of which are extremely undesirable, especially in patients who have undergone cardiac surgery. In addition, magnesium appears to play a role in the scavenging of free radicals and in the prevention of reperfusion injury [14,15]. Because reperfusion injury is thought to play a key role in the development of myocardial injury during and after coronary bypass surgery [16-18], the occurrence of hypomagnesaemia may contribute to this complication.

Low levels of other electrolytes such as phosphate and calcium can also have highly undesirable effects in patients with cardiovascular disease. Low phosphate levels can affect numerous intracellular enzymes and energy metabolism, leading to low levels of intracellular ATP [19]. Clinical symptoms include muscle weakness, respiratory failure, increased risk for respiratory infections, impaired myocardial function and a decrease in cardiac output [13,20-24]. Hypophosphataemia can lead to ventricular tachycardia in patients with recent myocardial infarction [25]. Low serum calcium levels can also induce arrhythmias (specifically shortening of the electrocardiographic QT interval).

Hypocalcaemia can lead to severe cardiovascular depression [26,27] and congestive heart failure that is unresponsive to inotropic agents, especially in patients with underlying cardiomyopathies [28,29]. These cardiovascular effects may occur in the absence of specific electrocardiographic changes. Thus, low electrolyte levels can have severe adverse effects on the clinical course of patients with cardiovascular disease. Moreover, when more than one electrolyte is deficient the effects may be cumulative, especially in the case of magnesium and potassium deficiencies. These impacts of electrolyte disorders may be more pronounced in patients undergoing cardiac surgery, who are already at increased risk for tachyarrhythmia and other haemodynamic complications during the perioperative and postoperative periods [1,30,31]. Preventing electrolyte disorders is thus an important goal of therapy in this category of patients.

During surgery, patients undergoing cardiac surgery are usually cooled to temperatures between 32°C and 34°C, in order to reduce tissue oxygen demand. At the end of the procedure patients are rewarmed to 36°C. We previously reported that induction of similar degrees of hypothermia induced electrolyte loss in patients with severe head injury [32]. Although hypothermia was maintained much longer in this category of patients, electrolyte disorders occurred mainly during the phase when body temperature decreased. This led us to hypothesize that other groups of patients treated with moderate hypothermia (such as patients undergoing major surgical interventions) might also be at risk for electrolyte depletion during the perioperative and postoperative periods. We therefore conducted the present study to assess the incidence of electrolyte depletion in patients who have undergone cardiac surgery.

## Methods

We measured serum levels of magnesium, phosphate, potassium, calcium and sodium at ICU admission in 500 consecutive patients undergoing cardiac surgical interventions (group 1).

The normal reference values for these electrolytes in our laboratory were as follows (all in mmol/l): magnesium 0.8–1.1, phosphate 0.7–1.2, potassium 3.8–4.8, calcium 2.20–2.60 and sodium 135–145. We used slightly lower levels as cutoff points for clinically significant electrolyte depletion. These values were as follows (all in mmol/l): magnesium 0.7, phosphate 0.6, potassium 3.6, calcium 2.0 and sodium 129. We also measured levels of ionized magnesium and ionized calcium in a subgroup of 40 patients in each group in order to determine whether the total serum levels of these electrolytes corresponded with levels of the ionized (i.e. active) form.

Surgical procedures included coronary bypass graft ( $n = 352$ ), valve replacement ( $n = 54$ ), combinations of these ( $n = 68$ ) and Bentall procedure for dissection of the ascending

aorta (usually in combination with aortic valve replacement;  $n = 26$ ). Extracorporeal circulation was employed in all patients in group 1. Cardioplegic arrest was accomplished using cold crystalloid solution (average amount 1000 ml). The cardioplegic solution contained the following concentrations of electrolytes (all mmol/l): sodium 120, potassium 16, magnesium 16, calcium 1.2 and chlorine 172.

A total of 250 patients who had undergone other major elective surgical procedures (noncardiac thoracic surgery [i.e. lung surgery] and repair of abdominal aortic aneurysms) served as control individuals (group 2). Neurosurgical patients were not included in the control group because we previously observed that such patients are at risk for developing low electrolyte levels for various reasons [32-34]. Patients undergoing nonelective (emergency) surgical procedures were not included in the present study.

Patients in group 1 were treated with low doses of dopamine (between 2 and 4 mg/hour; average dose 2.4 mg/hour) and nitroglycerine (0.1 mg/hour), according to protocol. Upon admission, a fluid infusion containing  $MgSO_4$  and phosphate was initiated in all patients in group 1 after blood sample aspiration. Potassium administration was initiated in all group 1 patients during surgery; this was continued and, in most patients, increased at ICU admission. Urine production was measured in all patients. Urinary electrolyte excretion was measured in 50 patients in each group. Measurements in urine produced during surgery were obtained (because extra electrolyte administration was initiated soon after ICU admission, which might have affected urinary excretion).

Where applicable, values are expressed as mean  $\pm$  standard error.

## Results

The results are summarized in Tables 1 and 2. Electrolyte levels (measured 1–4 days before surgery) were normal before surgical intervention. Severe electrolyte depletion was observed in group 1 (cardiac surgery patients). The differences between group 1 and group 2 (control individuals) were significant for potassium ( $P < 0.001$ ), magnesium ( $P < 0.001$ ), phosphate ( $P < 0.001$ ) and calcium ( $P < 0.001$ ; Table 1). Potassium levels were significantly lower in group 1 despite considerable potassium supplementation ( $10.4 \pm 4.6$  mmol/hour in group 1 versus  $1.6 \pm 1.4$  mmol/hour in group 2;  $P < 0.001$ ). Similarly, magnesium levels were significantly lower despite the fact that 380 patients in group 1 received magnesium during surgery (average amount: 2.1 g) because of arrhythmias (as compared with only five patients in group 2;  $P < 0.001$ ). Calcium was given to 84 patients in group 1 and two patients in group 2 ( $P < 0.001$ ) for clinical reasons, mostly hypotension, prevention or treatment of arrhythmias, and prevention or treatment of excessive blood loss.

Levels of ionized magnesium and ionized calcium were measured in a subgroup of 40 patients in each group; these levels corresponded with the corrected levels of non-ionized electrolyte levels in these patients. Average levels of ionized magnesium were  $0.27 \pm 0.23$  mmol/l in group 1 and  $0.48 \pm 0.36$  mmol/l in group 2 ( $P < 0.01$ ). Average levels of ionized calcium were  $0.91 \pm 0.55$  mmol/l and  $0.98 \pm 0.54$  mmol/l in groups 1 and 2, respectively ( $P < 0.05$ ).

**Table 1**

Parameter	Group 1	Group 2	<i>P</i>
Number of patients	500	250	
Age (years)	$59.9 \pm 23.2$	$60.3 \pm 28.6$	NS
ICU mortality	5.8%	4.0%	$P < 0.01$
Preoperative serum creatinine levels ( $\mu\text{mol/l}$ )	89 (range 53–131)	104 (range 64–153)	$P < 0.01$
Patients treated with dopamine	100%	15% ( $n = 37$ )	$P < 0.001$
Average dose of dopamine	5.7 $\mu\text{g/kg per min}$	6.9 $\mu\text{g/kg per min}$	$P < 0.02$
Patients treated with furosemide during surgery and/or in the 12-hour period preceding surgery	35% ( $n = 176$ )	25% ( $n = 63$ )	$P < 0.01$
Average dose of furosemide	16.7 mg	17.2 mg	NS
Rectal temperature at ICU admission	34.6°C	36.0°C	$P < 0.001$
Patients requiring antiarrhythmic medication (amiodarone, sotalol or other $\beta$ blocker) <sup>a</sup>	189 (38%)	26 (10%)	$P < 0.001$

<sup>a</sup>Prescribed in the intensive care unit (ICU) for treatment of arrhythmias. Patients using antiarrhythmic medication before surgery not included; prescription of  $\beta$  blocker for hypertension not included. Where applicable, values are expressed as mean  $\pm$  standard error. NS, not significant.

**Table 2****Electrolyte levels at intensive care unit admission**

Parameter	Group 1	Group 2	P
<b>Magnesium</b>			
Number of patients treated with magnesium before ICU admission	76% (n = 380)	2% (n = 5)	<i>P</i> < 0.001
Magnesium levels (mmol/l)	0.62 ± 0.24	0.95 ± 0.27	<i>P</i> < 0.001
Magnesium levels in patients without magnesium supplementation during surgery (mmol/l)	0.47 ± 0.16	0.95 ± 0.26	<i>P</i> < 0.001
Patients with hypomagnesaemia	46% (n = 228)	16% (n = 40)	<i>P</i> < 0.001
Patients with hypomagnesaemia in group with no magnesium supplementation	80% (n = 96)	1% (n = 3)	<i>P</i> < 0.001
<b>Phosphate</b>			
Phosphate levels (mmol/l)	0.43 ± 0.22	0.92 ± 0.32	<i>P</i> < 0.001
Patients with hypophosphataemia	83% (n = 415)	12% (n = 29)	<i>P</i> < 0.001
<b>Potassium</b>			
Potassium supplementation during surgery (mmol/hour)	10.1 ± 4.7	1.3 ± 1.0	<i>P</i> < 0.001
Potassium levels (mmol/l)	3.6 ± 0.70	3.9 ± 0.63	<i>P</i> < 0.01
Patients with hypokalaemia	34% (n = 170)	12% (n = 29)	<i>P</i> < 0.001
<b>Calcium</b>			
Calcium levels <sup>a</sup> (mmol/l)	1.96 ± 0.41	2.12 ± 0.33	<i>P</i> < 0.01
Patients with hypocalcaemia <sup>a</sup>	7.8% (n = 39)	5.6% (n = 15)	<i>P</i> < 0.01
<b>Sodium</b>			
Sodium levels (mmol/l)	134 ± 8	141 ± 9	<i>P</i> < 0.01
Patients with hyponatraemia	6% (n = 31)	5% (n = 12)	NS

<sup>a</sup>Corrected to serum albumin levels of 4 g/dl. Where applicable, values expressed as mean ± standard error. ICU, intensive care unit; NS, not significant.

Urinary electrolyte excretion, measured in 40 patients in each group, was as follows (group 1 versus group 2; values expressed in mmol/hour): magnesium 0.6 ± 0.33 versus 0.20 ± 0.32 (*P* < 0.01); phosphate 5.1 ± 3.0 versus 2.2 ± 2.0 (*P* < 0.01); potassium 11.0 ± 4.8 versus 7.2 ± 4.8 (*P* < 0.01); and calcium 1.2 ± 0.7 versus 0.4 ± 0.2 (*P* < 0.01).

Significant differences were also observed in the number of patients with clinically significant electrolyte depletion (i.e. levels below which deleterious effects are likely to occur). In group 1, 228 patients (44%) had magnesium levels below 0.70 mmol/l, as compared with 40 patients (16%) in group 2 (*P* < 0.001). As stated above, many patients in group 1 had been given magnesium during the surgical procedure for clinical reasons (mostly occurrence of brief ventricular or supraventricular arrhythmias). Of the 120 patients who had not been given magnesium, 96 (80%) had magnesium levels below 0.70 mmol/l.

In group 1, 415 patients (83%) had phosphate levels below 0.60 mmol, as compared with 29 patients (8%) in group 2 (*P* < 0.001). Moderate hypokalaemia (potassium < 3.6 mmol/l)

was present in 170 patients (34%) in group 1 (despite potassium supplementation and frequent measurements), as compared with 20 patients (8%) in group 2 (*P* < 0.001). Severe hypokalaemia (potassium = 3.0 mmol/l) was present in 60 patients (12%) in group 1, as compared with eight patients (3%) in group 2 (*P* < 0.01).

Overall, of patients in group 1, 438 (88%) had clinical deficiency in at least one electrolyte, as compared with 50 (20%) in group 2 (*P* < 0.001).

## Discussion

Our findings clearly demonstrate that patients undergoing cardiac surgical procedures with extracorporeal circulation are at high risk for electrolyte depletion. This phenomenon occurred despite the facts that our cardioplegia solution contained high doses of potassium and magnesium, and that potassium supplementation was given throughout the surgical procedure. The mechanism for this appears to be a combination of increased urinary excretion and intracellular shift, induced by a combination of extracorporeal circulation and decreased body temperature during surgery (hypothermia induced diuresis and

intracellular shift). We previously reported induction of electrolyte depletion induced by hypothermia in patients with severe head injury [32]; in these patients the responsible mechanism was a combination of increased urinary loss and intracellular shift. Indeed, high urinary excretion of magnesium, potassium and phosphate was documented in our patients, although urinary excretion alone could not account for our observations.

That urinary electrolyte excretion was high whereas serum electrolyte levels were low indicates a degree of tubular dysfunction in our patients, despite the fact that serum creatinine levels were normal; if tubular dysfunction were not present then the kidney would have reabsorbed most of the excreted electrolytes. The cause of the tubular dysfunction in our patients is unknown. It seems likely that some of the medications used in their treatment played a role. All were treated with low doses of dopamine, which can enhance renal excretion of sodium and other electrolytes [34], and with catecholamines, which can contribute significantly to the development of hypophosphataemia [35]. About one-third of patients were given diuretics before and/or during surgery; however, high electrolyte excretion also occurred in patients not given diuretics, and so the effect cannot be explained by diuretics alone.

A potential limitation of the present study is that we did not measure levels of ionized magnesium and ionized calcium in all patients. However, we did measure ionized magnesium and calcium in a subgroup of 40 patients in each group and found similar differences; moreover, these differences were statistically significant. There is no reason to assume that protein binding in the other patients was likely to be significantly different between the two groups; in addition, albumin levels in groups 1 and 2 were similar. We therefore feel that our observations of differences between the two subgroups are likely to reflect real differences between the two groups overall.

A number of potential mechanisms could account for the intracellular shifts that probably explain part of our findings. The most common reason for electrolyte shifts is the occurrence of changes in acid-base status (intracellular shift induced by alkalosis). However, this did not occur in our patients because acid-base status was regularly monitored and no major changes were noted. However, a number of other metabolic interactions might have taken place. For example, one of the causes for intracellular shift of phosphate is an increase in insulin levels. Although we did not measure insulin, a degree of insulin resistance may have been induced in our patients following preoperative administration of corticosteroids. Another reason may be loss of potassium and magnesium, each of which can cause insulin resistance. The effects of extracorporeal circulation are difficult to assess. Hypomagnesaemia in patients undergoing open heart surgery was described in a number of papers published about 30 years ago [36-39], and attributed to haemodilution [38,39]. Urinary excretion of magnesium or serum levels of other electrolytes were not meas-

ured in those studies; in retrospect, intraoperative hypothermia might also have played a role in these observations. However, it is not possible to separate the effects of extracorporeal circulation from those of hypothermia either in those studies or in the present one.

Remarkably, although the differences for cardiac surgery patients were clear, low electrolyte levels (especially magnesium) were also observed quite frequently in control individuals. Of control individuals, 20% had at least one clinical electrolyte deficiency (as compared with 88% in the study group). Although our study was not designed to address this issue, we strongly suspect that this phenomenon is related to intraoperative hypothermia. Although all patients undergoing cardiac surgery with extracorporeal circulation were intentionally cooled to approximately 32°C during surgery, mild accidental hypothermia with body temperatures between 35°C and 36°C occurred in many control patients during the (lengthy) surgical procedures. We suspect that this might have led to moderate electrolyte loss in these patients.

Low levels of magnesium, phosphate and, to a lesser degree, calcium and potassium were observed despite the fact that all patients were given substantial amounts of potassium during surgery, and most patients received at least one bolus of magnesium during the surgical procedure. A large number of these patients met clinical criteria for hypomagnesaemia or hypophosphataemia, or both; even hypokalaemia was found relatively frequently, even though patients received considerable potassium supplementation during the surgical procedure. This latter observation might have been caused by the concomitant presence of hypomagnesaemia, which can lead to significant renal losses of potassium [12,13].

Low levels of magnesium cause not only cardiac arrhythmias but also hypertension and vasoconstriction, including constriction of coronary arteries [12,13,40-42]. Magnesium appears to act as a physiological calcium channel blocking agent, albeit without the associated negative inotropic effects [40,43]. Moreover, the susceptibility of blood vessels (including coronary arteries and presumably the mammalian arteries, which are frequently used in bypass surgery) to vasoconstrictive agents is increased by hypomagnesaemia [42,44]. A number of studies have documented low magnesium levels in patients presenting with acute myocardial infarction [4-6] and unstable angina [6,7]. Various animal experiments [45,46] and clinical studies [8-11] have suggested that supplementing magnesium in patients with unstable angina or suspected myocardial infarction may prevent infarction or limit infarct size, and reduce mortality. Although these observations do not apply directly to patients undergoing open heart surgery, they do indicate that hypomagnesaemia can be detrimental in situations in which coronary blood flow is threatened or impaired. Another potentially important mechanism is the possible role played by magnesium as a free radical scavenger in the pre-

vention of reperfusion injury [14,15], which may play a key role in the development of postoperative complications in this category of patients. Furthermore, a number of *in vitro* and animal studies have shown that magnesium can prevent intracellular sodium overload and excess mitochondrial calcium uptake during ischaemic injury. These two developments are key elements in the progression of ischaemic injury to cell death, and both are directly linked to the extent of ischaemic injury [40,47,48]. A number of studies have reported decreases in intraoperative and postoperative arrhythmias induced by addition of magnesium to warm blood cardioplegia or by intermittent magnesium administration in patients undergoing coronary artery bypass grafting [49-51].

In addition, magnesium may be linked to prevention of neurological injury after ischaemia or trauma [52,53]. Coronary bypass surgery has been associated with transient or even permanent neuropsychological deficits in up to 30% of patients undergoing cardiac surgical procedures [54,55]; these injuries may be due to small thrombotic emboli occurring during surgery. Prevention of hypomagnesaemia may help to mitigate these neurological injuries. All in all, there is a large body of evidence suggesting that magnesium plays an important role in preventing (additional) injury to the ischaemic or injured heart, and perhaps also the brain. The effects of magnesium depletion can be greatly enhanced in the presence of other electrolyte disorders, especially hypokalaemia. Conversely, the effects of hypokalaemia may become manifest or be enhanced in the presence of hypomagnesaemia; in addition, hypomagnesaemia can induce hypokalaemia through increased renal potassium excretion, and hypokalaemia in turn can cause hypomagnesaemia. These and other interactions of potassium and magnesium are discussed more extensively in various reviews [12,13]. As is the case for hypomagnesaemia, hypokalaemia can induce cardiac arrhythmias (especially in patients with ischemic heart disease and left ventricular hypertrophy). It is also associated with muscle weakness, rhabdomyolysis, renal failure and hyperglycaemia.

Phosphate levels were low in the overall majority of cardiac surgery patients we evaluated. The reasons why the depletion of phosphate was more marked than for other electrolytes are probably that phosphate was not supplemented during surgery and perhaps that greater intracellular shift occurred due to the mechanisms outlined above. Hypophosphataemia after cardiac surgery was previously reported [56]; in the present study hypophosphataemia was mitigated by blood transfusions, because anticoagulant solutions used in stored blood may contain relatively large amounts of phosphate. Various adverse effects of hypophosphataemia on myocardial and respiratory function are described in the introduction section above. On the basis of the studies cited, it appears plausible that outcome in cardiac surgery may be adversely affected by hypophosphataemia.

Clinical problems associated with hypocalcaemia are also briefly outlined in the introduction section above. Mild hypocalcaemia is frequently asymptomatic, although this depends partly on the presence of other electrolyte disorders and on the speed with which hypocalcaemia develops. Hypocalcaemia in our patients was generally mild, and might have been caused in part by magnesium deficiency (which is a frequent cause of hypocalcaemia). No visible symptoms of hypocalcaemia, such as tetany, were observed.

Arrhythmias occurred frequently in our cardiac surgery patients, and antiarrhythmic medication (mostly amiodarone or sotalol) was required in a substantial minority. It seems highly likely that electrolyte disorders in our patients either caused these arrhythmias or contributed to their development. Electrolytes were measured at ICU admission, and disorders were corrected immediately; it seems highly likely that, if left untreated, the disorders could have had a negative impact on outcome.

## Conclusion

We observed that patients undergoing cardiac surgery with extracorporeal circulation are at high risk for electrolyte depletion. The mechanism is probably a combination of increased urinary excretion and intracellular shift, induced by a combination of intraoperative hypothermia and extracorporeal circulation. Our findings may partly explain the high risk for tachyarrhythmia in patients who have undergone cardiac surgery. Alternatively, electrolyte depletion may increase the risk for this complication. On the basis of our findings we recommend that magnesium, potassium, phosphate and calcium be frequently measured during and after cardiac surgery. Prophylactic supplementation of potassium, magnesium and phosphate should be seriously considered in all cardiac surgery patients during surgery and in the perioperative period. Although mild hypocalcaemia usually does not require treatment, it should be kept in mind that intravenous replacement of phosphate and, to a lesser degree, magnesium can aggravate existing hypocalcaemia (due to calcium binding to phosphate, or to sulphate when  $MgSO_4$  is administered). Therefore, if mild hypocalcaemia is present while high doses of magnesium or phosphate are administered, calcium levels should also be corrected. In our opinion, careful monitoring and prompt correction of electrolytes will contribute to the prevention of postoperative tachyarrhythmia and help to improve outcomes in patients undergoing cardiac surgical procedures.

**Key messages**

- Patients undergoing cardiac surgical procedures with extracorporeal circulation and mild intraoperative hypothermia are at high risk for developing severe electrolyte depletion.
- Causative mechanisms include increased urinary excretion and intracellular shift, possibly linked to perioperative induction of hypothermia.
- Such electrolyte disorders may contribute significantly to the occurrence of perioperative tachyarrhythmia, which is an important cause of morbidity and mortality in patients undergoing cardiac surgery.
- Electrolyte levels should be frequently monitored in all patients undergoing cardiac surgical procedures; prophylactic supplementation of potassium, magnesium and phosphate should be strongly considered in these patients during surgery and in the immediate postoperative period.

**Competing interests**

The author(s) declare that they have no competing interests.

**Author's contributions**

Collection and analysis of the data was performed by KHP. The manuscript was jointly written by KHP and ARJG. All authors have read and approved the final manuscript.

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