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Monitoring Blood Gasses and Metabolic Parameters at the Bedside:

Clinically and Economically Justified?

Authors

Kees H. Polderman

Senior Consultant in Intensive Care Medicine

Armand R.J. Girbes

Professor of Intensive Care Medicine

Dept. of Intensive Care, VU, University Medical Centre

Amsterdam, The Netherlands

Correspondance

k.polderman@vumc.nl

Preventing hyperglycaemia and electrolyte disorders and optimizing ventilator settings can help prevent complications, thereby decreasing morbidity and length of stay in the ICU. On-site analysis may help in maintaining metabolic homeostasis, thereby improving outcome and reducing costs.

Introduction

On-site blood gas analyzers can be used to monitor blood gasses and guide ventilator settings, and also to quickly assess parameters such as hematocrit and serum levels of glucose and various electrolytes. This article focuses on clinical aspects of bedside blood gas analysis, and also touches on cost implications.

Electrolyte Disorders

Electrolyte disorders can be found with high frequency in critically ill patients. Clinical problems may arise both when electrolyte levels are excessively high or low. The most frequent cause of high electrolyte levels, especially hyperkalemia and hyperphosphataemia, is renal failure with oliguria or anuria. However, electrolyte depletion is a far more common finding in critically ill patients. For example, Wong and associates found hypomagnesaemia in 65% of patients admitted to an intensive care unit, compared to 12% of patients in a general ward (Wong et al. 1983). We observed significant electrolyte disorders in 20%-90% of our patients at ICU admission, depending on the category of patients (Polderman et al. 20001,2 Polderman and Girbes 2004). Apart from manifest renal dysfunction, electrolyte disorders were found most frequently in patients with various types of neurological injury (Polderman et al. 20001,2) and those admitted following cardiac surgery with use of extra-corporeal circulation (Polderman and Girbes 2004).

Electrolyte disorders in critically ill patients are often caused by subtle renal disorders such as tubular dysfunction, often occurring in patients admitted to the ICU. Tubular dysfunction often leads to excessive electrolyte loss. This problem can be significantly exacerbated by treatments commonly used in the ICU, such as administration of diuretics (including osmotic diuretics such as mannitol), vasoactive drugs such as norepinephrine and dopamine, antibiotics such as aminoglycosides and pipracillin, and many others (Polderman et al. 2002; Brown and Greenwood 1994; Smit et al. 1995; Weisinger and Bellorin-Font 1998). In addition, diverse interventions such as nasogastric suction, fluid administration and induction of hypothermia can further increase electrolyte loss (Polderman at al. 2001; Weisinger and Bellorin-Font 1998). Finally, initiation of continuous veno-venous haemodialysis for acute renal failure can also lead to severe electrolyte disorders.

There is convincing evidence that electrolyte disorders can have severe consequences in critically ill patients. The link between sodium

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disorders (both hypo- and hypernatraemia) and adverse outcome is well recognized (Kumar and Berl 1998) and has led to its inclusion in various severity of illness (risk adjustment) scores such as the APACHE-II score (Knaus et al. 1985). It is also well known that hypokalaemia can cause complications such as muscle weakness, rhabdomyolysis, renal failure and hyperglycaemia as well as cardiac arrhythmias, especially in patients with ischemic heart disease. For this reason levels of sodium and potassium are usually monitored carefully in severely ill patients. However, disorders of other electrolytes may have similarly negative consequences, although these are often monitored far less frequently.

For example, hypomagnesaemia has been linked to adverse outcome and increased mortality both in the intensive care (Chernow et al. 1989; Rubeiz et al. 1993) and general ward (Chernow et al. 1989). Although this does not necessarily imply a causal relationship (hypomagnesaemia could simply be a marker of more severe illness) there are various plausible mechanisms through which outcome could be directly influenced. Clinical studies have shown that hypomagnesaemia is linked to the occurrence of severe arrhythmias and adverse outcome in patients with unstable angina or myocardial infarction (Abraham et al. 1986; Kafka et al. 1987) and that administering magnesium can reduce mortality and infarction size in this category of patients (Teo et al. 1991; Woods et al. 1992). Hypomagnesaemia can also lead to insulin resistance and hyperglycaemia (Polderman et al. 2003; Weisinger and Bellorin-Font 1998) which in turn may adversely affect outcome; effective control of blood sugars has been shown to reduce mortality and length of stay at least in post-surgical patients (Van den Berghe et al. 2001). Another issue is the possible role of magnesium in neurological injuries; a range of animal studies has linked hypomagnesaemia to the development of additional and more severe neurological injuries, while administration of magnesium before or after the injury prevents or mitigates these injuries (Polderman et al. 2003; Vink and Cernak 2000). This issue has not yet been properly addressed in clinical studies.

Other electrolyte disorders may also adversely affect the clinical course of critically ill patients. Hypophosphataemia can induce muscle weakness including weakness of the diaphragm and respiratory muscles, leading to an increased rate of respiratory infections and failure to wean (Aubier et al. 1985; Fisher et al. 1983; Gravelyn et al. 1988; Varsano et al. 1983). In addition, hypophosphataemia can decrease myocardial output and may induce arrhythmias, especially

in patients with pre-existing myocardial ischemia (O'Çonnor et al. 1977; Ognibene et al. 1994). Hypocalcaemia can also lead to arrhythmias, as well as to muscle weakness with severe cardiovascular depression and congestive heart failure, gastro-intestinal disorders and renal failure (Bushinsky and Monk 1998; Connor et al. 1982; Drop 1985; Zaloga and Chernow 1986).

No studies specifically dealing with the financial consequences of electrolyte disorders have been performed. However, as electrolyte disorders have been linked to specific complications such as arrhythmias, increased infection rates and failure to wean, it seems highly likely that length of stay will be increased by electrolyte disorders. Although it has not been shown that correcting these disorders improves outcome and reduces length of stay this appears to be a reasonable assumption; moreover, such improvements have been reported in specific categories of patients such as those with myocardial infarction (Rasmussen et al. 1986; Shechter et al. 1990; Teo et al. 1991; Woods et al. 1992). Maintaining electrolyte levels within a narrow range is thus an important goal of therapy in the ICU, especially in neurocritical patients in whom the (injured) brain is more susceptible to the potential consequences of electrolyte disorders, including hypotension, cerebral vasospasms, cardiac arrhythmias and hyperglycaemia (Polderman et al. 2003).

Because electrolyte disorders often have the same causes there is a high likelihood that different disorders will occur simultaneously, especially in critically ill patients. This significantly increases the risk for clinical manifestations. Moreover, the risk of complications like arrhythmias increases in patients with pre-existing myocardial disease. An additional problem is that electrolyte disorders can develop very swiftly; we observed a 50% decrease in serum electrolyte levels 6 hours after induction of hypothermia in patients with severe traumatic head injury (Polderman et al. 2001).

All this implies that frequent measurements and supplementation of different electrolytes may be required to maintain electrolyte levels in the normal range in particular categories of patients at specific times.

Glucose Levels

Tight regulation of blood glucose levels has become an important goal of therapy in critically ill patients, as intensive insulin therapy to prevent hyperglycaemia has been shown to reduce mortality and improve outcome in various categories of ICU patients (Finney et al. 2003; Van den Berghe et al. 2001). This has led to a much more aggressive treatment of hyperglycaemia in many ICUs worldwide, implying that much more frequent assessments of blood sugar levels will be needed. Some studies have reported improved glycaemic control through use of strict protocols and standardization of intensive insulin therapy (Finney et al. 2003). On site measurements may help implement such protocols.

Blood Gasses

Blood gasses are used to guide ventilator settings in mechanically ventilated patients. In most patients peripheral saturation measurements can be used as an additional tool to assess oxygen levels and prevent hypoxia. On-site blood gas analysis will provide additional benefits in patients in whom peripheral saturation is difficult to measure and/or in whom this measurement does not correlate well with actual oxygenation. In addition, prevention of hypocapnia may be an important goal of therapy, especially in patients with neurological injuries; severe hypocapnia (usually defined by CO_2 levels below 30 mmHg) can lead to constriction of cerebral arteries and local ischemia, especially of injured areas. Note also that blood gas measurements are temperature dependent; levels of oxygen decrease by ±4mmHg/oC, and carbon dioxide levels by ±2mmHg/oC when temperature decreases. These differences are particularly important when, for example, mixed venous saturation is measured; a small difference in measured pO_2 due to temperature or if the patient has high fever or a very low temperature, will significantly effect the measurement. It is easier to correct for these temperature effects properly when measurements are performed on site.

Conclusion

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Critically ill patients have a very high risk of quickly developing electrolyte disorders and hyperglycaemia. These disorders can lead to complications which adversely affect outcome and increase length of stay. The evidence suggests that these adverse consequences can be prevented or mitigated by preventing or promptly correcting these disorders. It therefore makes sense to measure electrolyte levels and glucose levels on site in the ICU, to allow more frequent measurements, decrease errors due to incorrect temperature input, and allow quicker therapeutic interventions, guided by these measurements in high-risk patients.

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