Editorials

Recent advances in cardio-pulmonary resuscitation (CPR)

The threat of a cardiac arrest always looms over our existence, be it in our clinical practice or in our personal lives. Our desire to gain successful outcomes from these potential disasters has lead to much discussion over the centuries. With the march of evidence-based practice, anecdote and opinion are slowly giving way to a more consistent approach. This is most evident in the recent American Heart Association's "Guidelines 2000".[1] These guidelines have been produced as a result of many years of toil by many American and international experts, who critically evaluated the world's literature on cardiopulmonary resuscitation. The critical appraisal of published (and unpublished) literature involves more than just assessing the validity of the claims of each study.[2] The particular outcome being assessed must be considered (e.g. haemodynamic improvement versus 1 year survival) as must the 'number needed to treat' to achieve this outcome.[2] The characteristics of the wider population must also be compared with those reported in the study.

Over the past decade many interesting topics relating to the practice of CPR have been studied. Unfortunately, many initially promising modifications, on animal or model based evaluations, have not been proven to be useful in human subjects (e.g. high dose adrenaline, barbiturates, corticosteroids). Some techniques with early positive "efficacy" studies have not been demonstrated to be as effective when evaluated more widely.[3] As with other areas of investigation, the gold standard for CPR research is of course an adequately powered prospective randomised double blind trial. Many of these have been performed, and some of these have had positive results.

Interest in vasopressin as an alternative vasopressor peaked when Lindner and colleagues published a small prospective, randomized double blind study.[4] Patients in refractory ventricular fibrillation (VF) were given either 40U of vasopressin or 1 mg of adrenaline intravenously. Significantly more patients in the vasopressin group survived 24 hr, though survival to hospital discharge was not significantly different. The value of vasopressin in cardiac arrest management will be clarified by the much-awaited publication of the results of larger trials from Ottawa and Europe.

Amiodarone was recently shown to improve survival to hospital admission in patients with shock-refractory VF when compared with placebo,[5] but again survival to hospital discharge was unchanged. Sack and colleagues compared Interposed Abdominal Compress-ion CPR (IAC CPR) with standard CPR and demon-strated a higher hospital discharge rate in the IAC CPR group.[6] Unfortunately these promising results have not been replicated.

Another modification of standard CPR, Active Compression Decompression CPR ("toilet plunger" CPR) has also been widely studied.[7] Despite a lack of benefit in most studies, a French group of investigators were able to demonstrate an improved 1-year survival with this technique.[8] This result was obtained by a well-trained and adequately staffed pre-hospital team who provided advanced life support after a mean duration of 20 minutes following the arrest.

Another area of controversy is the necessity to include ventilation in basic life support (BLS) training. Many individuals are unwilling to perform mouth-to-mouth ventilation, and perhaps a simplified protocol without mouth-to-mouth ventilation would encourage more people to perform bystander CPR. This approach has been supported, in part, by a recently published prospective randomised study.[9] This study compared telephone dispatcher instructions which either included or excluded mouth-to-mouth ventilation. The investigators found that in Seattle, with an average response time of 4 minutes, there was no outcome benefit with the additional instructions for ventilation. However, it is difficult to extrapolate this result to common CPR practice and thus change the teaching for BLS courses, although it certainly adds fuel to the debate.

This issue of Critical Care and Resuscitation contains evidence of another attempt to push the frontiers.[10] The author evaluated the effectiveness of cardiac compressions performed on a mannequin that had been placed in the prone position. Considering the potential complexity of rolling a prone patient supine in the event of a cardiac arrest, the desire to assess the effectiveness of CPR performed on a prone patient is understandable. However, it is very difficult to extrapolate from this artificial scenario to a patient in the prone position. This study adds to the body of literature available, although there is a long way to go before CPR in the prone position can be advocated as an evidence-based alternative to supine CPR.

There are some potentially valid newer approaches that are, as yet, based only on low levels of evidence. For example a recent observational study showed that hospital discharge rates were better for out-of-hospital arrests if 90 seconds of CPR preceded defibrillation.[11] This premise is now being evaluated in a prospective

randomised trial. In addition, there is now a greater focus on the systematic exclusion of causes of cardiac arrests, using mnemonics such as the 5 Hs and 5 Ts.[12,13] A simple recommendation to disconnect patients from mechanical ventilation should also be included, as auto-PEEP (i.e. 'gas trapping') has been recognised as a potentially reversible factor that is easily treated.[14,15]

This is an exciting time for investigating therapy for CPR, for the world's researchers are aware that by asking the right questions, and by carefully designing studies, many answers can now be obtained.

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Hyponatraemia and speed of correction. Why is there a dilemma?

Plasma sodium and its accompanying anions largely determine the plasma osmolality. In health, osmolality (and therefore plasma sodium concentration) is regulated by mechanisms that regulate body water, keeping the osmolality at 287 $\pm$ 7 mosmol/kg and plasma sodium at 140 $\pm$ 4 mmol/L.[1] While there are some clinical conditions where the serum sodium does not predict the plasma osmolality (e.g. hyperglycaemia, pseudohyponatraemia),[2] hypernatraemia is always associated with hyperosmolality and in general hyponatraemia is associated with hypoosmolality.[3]

Mild hypoosmolar hyponatraemia (i.e. 125 - 134 mmol/L) is not an uncommon finding in the critically ill patient and is often asymptomatic. However, severe hyponatraemia (i.e. < 125 mmol/L) can be life threatening. The management of severe hyponatraemia has tended to polarise clinicians to those who believe that the disturbance should be corrected slowly,[4] to avoid osmotic demyelination[5,6] and those who believe the disturbance should be treated rapidly, to reduce the risk of seizures and the development of brain death due to cerebral oedema.[7,8] Confusion concerning the correct treatment has occurred largely because of a misunderstanding of the associated changes in body fluid compartments.

Normally, acute hyponatraemia (i.e. developing in less than 3 days or present for < 48 hr) is associated with an increase in both intravascular and intracellular fluid (ICF) volumes, whereas chronic hyponatraemia (i.e. developing in three or more days), is associated with an increased, normal or decreased intravascular volume and an increased or normal ICF volume.[9] Rapid correction is necessary if there is cerebral oedema (i.e. increased ICF volume). However, if cerebral oedema is

mild or absent then slow correction is required.

Chronic hyponatraemia tends to occur in patients who are malnourished and sodium and potassium depleted (e.g. alcoholism, prolonged gastrointestinal loss, diuretic therapy) or who have chronic cardiac failure (where antidiuretic hormone stimulation is influenced more by a reduction in cardiac output than by hypoosmolality), or who have the syndrome of inappropriate secretion of antidiuretic hormone (SIADH).[10]

As the progression of hyponatraemia is slow (e.g. usually 14 days from the start of thiazide diuretic therapy[11]), the brain is able to adapt to the reduction in osmolality by reducing its intracellular osmolar component, inhibiting the development of cerebral oedema.[12,13] However, the process of osmotic 'adaptation' is faster than the process of osmotic 'de-adaptation' with experimental studies showing that rapid restoration of osmolality can cause dehydration of brain tissue[14] (an effect which is greater in the hyponatraemic animal than normonatraemic one),[15] and in certain cases leads to demyelination of white matter.[16]

The chronic hyponatraemic patient is either asymptomatic (e.g. the result is an incidental finding) or has only minor symptoms (e.g. confusion, disorientation, drowsiness). The urea and creatinine levels are often normal or elevated, compared with acute hyponatraemia where the urea and creatinine are markedly reduced. As cerebral oedema is not a feature of chronic hyponatraemia, rapid reversal is not required and a slow elevation of plasma sodium is sufficient to correct the disorder without increasing the likelihood of demyelination.

On the other hand, acute hyponatraemia develops rapidly in less than 3 days and usually from an excess of intravenous dextrose solutions administered to a post-operative patient. As cerebral tissue is unable to rapidly accommodate to the reduction in osmolality, cerebral oedema develops.[17] The patient often presents clinically with post-operative confusion and disorientation, progressing to somnolence, seizures, and even brain death. The hyponatraemia is severe but in some cases (e.g. menstruant women undergoing minor surgical procedures[7]) a severe encephalopathy can occur with a plasma sodium up to 128 mmol/L.[18]

As acute hypoosmolar hyponatraemia has a reported mortality of up to 50%,[13] rapid correction is required. Mannitol 1.0 - 1.5 g/kg (i.e. 375 - 550 mmol/70 kg to provide a blood-brain osmolar gradient of 10 - 30 mosmol needed to mobilise brain water[19]) administered over 10 minutes is often used to treat cerebral oedema associated with trauma, cerebral malignancy or cerebral abscess.[20] Similarly, hypertonic saline (125 - 250 mmol) has been used to manage resistant intracranial hypertension.[21] The standard approach to a patient who has acute hypoosmolar hyponatraemia is to elevate the serum sodium by 2 mmol/L/hr, up to 20 mmol/L/day (correcting to no greater than 130 mmol/L in 48 hours). However, if the patient is convulsing, up to 250 mmol of hypertonic saline (e.g. 50 mL of 29.2%, 60 mL of 4N, 70 mL of 20% or 500 mL 3% saline) may be infused over 10 minutes which rapidly reduces the cerebral oedema, controls the seizures and increases the serum sodium in an adult by an average of 7 mmol/L.[8,22] This is often followed by a spontaneous diuresis (i.e. > 200 mL/hr) allowing the excess body fluid (both ICF and intravascular fluid) to be excreted, although frusemide may also be required. While the standard recommendation for the rate of correction of hyponatraemia is by no more than 2 mmol/hr, this degree of control is often difficult to achieve. The initial correction of sodium by 5 - 8 mmol/L is often all that is required to reverse the life threatening manifestations of cerebral oedema with the following diuresis elevating the plasma sodium to 130 mmol/L over the next 48 hours. As an approximation, the elevation in serum sodium that will be achieved with hypertonic saline is 1 mmol/L for every 40 mmol of sodium administered without water (i.e. numerically the same amount of sodium in mmol as the patient's total body water in litres). The excess in free water may be calculated from the formula,

$$X = TBW \times \left(\frac{Na^{+}d}{Na^{+}m} - 1 \right)$$

Where,

X = water excess (L)
TBW = total body water (L)
$Na^{+}d$ = desired (or normal) plasma sodium (mmol/L)
$Na^{+}m$ = measured plasma sodium (mmol/L)

For the management of chronic hyponatraemia in the asymptomatic patient, all that is required is fluid restriction (< 500 mL/day) and reversal of any precipitating factor.[23] If chronic fluid deprivation is difficult to maintain then, in patients with chronic cardiac failure, an ACE inhibitor (to inhibit both angiotensin II stimulation of thirst and antidiuretic hormone release) and frusemide are often used,[24,25] although a direct antidiuretic hormone inhibitor may one day be of greater value.[26] In the resistant patient with cardiac or renal failure, dialysis may be required. However, in the chronic hyponatraemic patient who is sodium depleted (e.g. chronic gastrointestinal loss or inappropriate diuretic use) and who has normal or mild cardiac or renal dysfunction, then 0.9% saline to correct the extracellular fluid and plasma volume deficit will suppress antidiuretic hormone release, improve renal perfusion and allow the excretion of free water with

reestablishment of plasma sodium levels, without the need for hypertonic saline.[6] If the patient is symptomatic and hypertonic saline is deemed necessary then the rate of correction is recommended to be no greater than 12 mmol/L/day and continued only until the plasma sodium is 130 mmol/L.[5,23,27-29] While some believe that the rate of correction should be no greater than 0.5 mmol/L/hr, the initial rate of correction can be rapid,[30] (e.g. 125 mmol hypertonic saline over 10 minutes) provided that the final correction remains < 15 mmol/L/24 hours.[31] One review found that hypokalaemia was a risk factor of osmotic demyelination and suggested that, in patients without severe neurological symptoms, correction of hypokalaemia should precede the correction of hyponatraemia.[32] If a diuresis is established and the rate of correction is such that it may exceed 15 mmol/L/24 hr, intravenous DDAVP (or intravenous hypotonic fluids) should be used,[33] as the risk of cerebral oedema is low and the risk for osmotic demyelination is increased.

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Preoperative cardio-pulmonary risk assessment – more science and less art

For better outcomes in high-risk surgical patients, two basic requirements need to be addressed. The first is that a mechanism must be in place to ensure identification of all high-risk patients well prior to the day of surgery. The second is the need for validated effective risk-reduction strategies. Without one or both of these components, individual practitioners caring for such patients can do little to improve outcomes beyond the current level.

In this issue of Critical Care and Resuscitation, Dr Older and colleagues[1] outline their approach to the first requirement - the pre-operative identification of surgical patients at high risk of mortality and morbidity. They acknowledge that the American College of Cardiology/American Heart Association (ACC/AHA) guidelines[2] already allow identification of some high-risk surgical patients - for example those with certain clinical predictors such as unstable coronary syndromes, decompensated congestive cardiac failure, severe valvular disease, high grade atrioventricular block or significant arrhythmias. They also endorse the ACC/AHA concept of 'surgery-specific risk', where for example high-risk surgery is defined as major emergency surgery, major vascular surgery or prolonged procedures with large fluid shifts.[2] Where they differ is in their evaluation of other surgical patients, including those with intermediate or minor ACC/AHA clinical predictors of risk. Intermediate ACC/AHA predictors of cardiovascular risk include mild angina pectoris, prior myocardial infarction, compensated or prior congestive cardiac failure and diabetes mellitus. Minor predictors are advanced age, abnormal ECG, rhythm other than sinus, low functional capacity, history of stroke, and poorly controlled systemic hypertension.

Of course some patients in these lesser categories are actually high-risk, and this is where the determination of 'functional capacity' becomes important. The ACC/AHA guidelines quantify functional capacity purely from the history, using units called 'metabolic equivalents' (METS).[2] For example activities such as walking around the house, dressing and dishwashing are unlikely to exceed 4 METS, whereas playing football requires over 10 METS of energy expenditure. From the ACC/AHA standpoint, inability to achieve 4 METS is a marker of significant peri-operative risk. In contrast to this approach, which at best provides a mere estimate of functional capacity, Dr Older and colleagues advocate accurate measurement. To achieve this goal they recommend cardiopulmonary exercise (CPX) testing using a bicycle ergometer, 12 lead ECG and metabolic cart, ramping up to the estimated maximum work rate in 6 minutes.[1]

In particular the authors emphasise the accurate determination of the anaerobic threshold, which they regard as more reliable than maximal oxygen consumption since it is less dependent on patient motivation. The anaerobic threshold is the oxygen consumption at the point where the increase in CO_2 production during work escalation begins to exceed the increase in oxygen consumption. The onset of excess CO_2 production marks the titration of plasma and interstitial bicarbonate by the protons associated with lactic acidosis. Dr Older states that the fall in bicarbonate concentrations accompanying the rise in plasma lactate is stoichiometric. In fact, the major proportion of lactic acid added to blood is buffered by non-volatile buffer anions formed from haemoglobin, albumin and to a lesser extent phosphate.[3] Nevertheless, he is correct in that sufficient bicarbonate is titrated by the sudden appearance of lactic acid to cause a detectable increase in CO_2 production.

The logic underpinning CPX testing in surgical patients is based on a truism established many years ago by Shoemaker.[4-6] He showed that some patients fail to increase oxygen delivery and consumption sufficiently in the days following major surgery, and that these patients suffer a far higher mortality and morbidity than those who do. Essentially, what Dr Older does is establish the estimated peri-operative oxygen consumption requirement (a calculation based on the surgery-specific risk), determine whether the patient will be capable of sustaining this increase aerobically, and whether myocardial ischaemia is likely to be encountered during the requisite hyperdynamic response. In 1993, Dr Older and his colleagues applied the CPX testing criteria for cardiac failure devised by Weber and Janicki[7] to patients over the age of 60 years scheduled for major abdominal surgery. Mortality was 18% in those patients with an anaerobic threshold less than 11 mL/kg/min, and 0.8% in those with anaerobic thresholds exceeding this value.[8] They went on to obtain similar results in a prospective evaluation of CPX testing, with triage of higher risk patients to ICU or HDU, while sending lower risk patients back to the

surgical wards.[9] Interestingly, provided myocardial ischaemia did not develop before the anaerobic threshold, it was not a major risk factor.

So at least in Dr Older's hands, CPX testing seems to be a sensitive predictor of risk, and has the added advantage of availability before the surgical insult is inflicted. This puts it ahead of the POSSUM score for example,[10] which can only be determined in full post-operatively. It also enables refinement of the excessively broad and non-specific risk criteria developed by Shoemaker[11] and adapted subsequently by British researchers.[12,13] Presumably, many patients judged as high-risk by these criteria would be managed by Dr Older outside of ICU or even HDU provided CPX testing predicted low risk.

Most importantly, risk stratification by CPX testing might allow better evaluation of potential risk-reduction strategies. Dr Older himself advocates pre-operative ICU admission of high-risk patients for invasive monitoring and optimisation of oxygen-derived parameters, but provides no real evidence for the efficacy of this approach. Influential British researchers are quite insistent that the creation of a peri-operative hyperdynamic state is protective in high-risk surgical patients, including those with significant ischaemic heart disease, even to the extent of publishing didactic editorial statements.[14] However, for reasons outlined previously in this journal, the case for peri-operative goal-directed therapy is by no means established.[15] What makes the situation more confusing is the mounting evidence for establishing peri-operative beta-blockade in surgical patients at risk of myocardial ischaemia.[16,17] Acute beta-blockade works against the achievement of a peri-operative hyperdynamic state, at least conceptually. The benefit of this approach is particularly striking in patients with dobutamine-induced echocardiographic findings consistent with ischaemia at higher work-loads who then undergo major vascular surgery.[18] It would be interesting to find out how many of these patients also fall below the line on CPX testing. A direct comparison of goal-directed therapy versus simple beta-blockade might then be feasible.

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Lung oxygen consumption and its measurement

Oxygen consumption has been used in the critically ill patient to assess severity of illness,[1,2] caloric requirements,[3] and cardiovascular reserve,[4] and can be measured directly from inspired and expired respiratory gas measurements (i.e. metabolic cart)[5-7] or indirectly using the Fick principle.[8,9] The $\dot{V}O_2$ measured by the metabolic cart method is greater than that measured by the Fick method,[5,10,11] (by up to 30% in the presence of inflammatory lung disease) due largely to intrapulmonary $\dot{V}O_2$, which is excluded by the Fick method.[12] While this provides the potential for assessing pulmonary oxygen uptake at the bedside,[11] the Fick method with flow measured by the pulmonary artery catheter, does not include the left sided thebesian circulation (i.e. coronary blood flow returning directly to the left side of the heart through oxygen consuming myocardial tissue rather than through to the coronary sinus).[12] The Fick method is also more liable to variation compared with the metabolic cart method,[11] as it usually requires two more measurements to derive $\dot{V}O_2$ (e.g. CO, SaO_2, $S\bar{v}O_2$, PaO_2, $P\bar{v}O_2$, Hb) when compared with the metabolic cart method (e.g. $\dot{V}_I$, F_IO_2, $F\bar{E}O_2$, $F\bar{E}CO_2$)[13] and uses a constant (*k*) to represent the oxygen carrying capacity of haemoglobin in mL/g of Hb, which varies from 1.30 to 1.39.[14,15] While the theoretical oxygen carrying capacity in mL/g for haemoglobin A (STPD) is 1.3896, the oxygen combining capacity of 1 g of haemoglobin measured in human blood is often less, because some of the measured haemoglobin exists in a form that does not effectively transport oxygen (e.g. methaemoglobin, sulphaemoglobin, carboxyhaemoglobin). The *k* value of 1.31, as determined by Gregory, accounts for an average of the effective oxygen carrying capacity in mL/g when the haemoglobin is measured using the cyanmethaemoglobin standard (i.e. total haemoglobin).[16] However, the haemoglobin values from the current blood gas analysers (e.g. ABL 700 series Radiometer, Corning 800 series) are estimated using an optical method (e.g. a spectrophotometric multiple wavelength method detecting the haemoglobin species i.e. oxy, deoxy, met, carboxy and sulph haemoglobin). If the total oxy- and deoxyhaemoglobin concentration (i.e. active or effective haemoglobin) is used for the haemoglobin value, the value of *k* should be 1.3896,[17] although even then there may be small differences due to the 1 - 3% of haemoglobin A_2 and foetal haemoglobin present in the adult circulation. If the total haemoglobin concentration (when measured by the spectrophotometric method) is used, the value of *k* approaches that found by Gregory of 1.31, although this depends on the concentrations of methaemoglobin, sulphaemoglobin and carboxyhaemoglobin which may also vary widely in the critically ill patient.

Nevertheless, the difference in $\dot{V}O_2$ measured by the metabolic cart and Fick method may provide useful information concerning pulmonary metabolism,[11] and Opdam and Bellomo in this issue studied the correlation between the $\dot{V}O_2$ difference and the lung lactate release after cardiopulmonary bypass and in septic shock.[13] While they found an increase in the $\dot{V}O_2$ and lung lactate release in both groups, they found no correlation between the difference in $\dot{V}O_2$ measured by the metabolic cart and Fick method and lung lactate release. The lack of correlation may in part be due to the fact that the increase in oxygen consumption in inflamed lungs is due largely to the increase in oxygen consumption by neutrophils and macrophages during the 'oxygen bursts' associated with production of oxygen-derived free radicals.[18] These oxygen radicals are formed from NADPH and glutathione, utilising the hexose monophosphate shunt which does not directly involve the formation of lactate.[19]

As the study by Opdam and Bellomo did not demonstrate a direct link between lung lactate generation and lung oxygen consumption, this would tend to support the concept that inflammatory tissue oxygen utilization is predominantly aerobic and via the hexose monophosphate shunt, rather than the Embden-Meyerhof pathway. However, their statement that lung lactate release and lung oxygen consumption 'may not share a common pathogenesis' is probably the preferred conclusion, due to the inaccuracies in clinical measurement of lung oxygen uptake.

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With faltering steps still we come

Australasian critical care medicine has almost passed through its adolescent stage of development. For more than 20 years intensive care medical training has been under the watchful eye of both the Australian and New Zealand College of Anaesthetists (ANZCA) and the Royal Australasian College of Physicians. During this time there has only been sporadic expressions of a right to autonomy.[1,2] However, it appears that we are on the threshold of an independent and single training body in Critical Care Medicine – a separate College. This has been recently acknowledged by the dean of the Faculty of Intensive Care ANZCA in the July edition of the ANZCA bulletin in which he states "Two surveys overwhelmingly confirmed that this is an important matter for intensive care specialist and that an independent College is the ultimate goal".[3]

"This" (i.e a single body for training and certification in intensive care) is indeed an important matter for the intensivist, as a single Australasian intensive care medical training scheme has been the 'holy grail' for almost 20 years.[4]

To facilitate this move it seems that the formation of a joint Faculty of Intensive Care with the Royal Australasian College of Physicians is the first step. To provide financial independence, a separate subscription will apply for the Faculty of Intensive Care from the year 2002.[3] In a 'point of view' article in this issue of Critical Care and Resuscitation, Dr. Holt[5] reviews the implications of this and suggests that this may be the litmus test to indicate the number of totally committed intensivists (and perhaps the viability of the specialty).

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