

Capnometry in the prehospital setting: are we using its potential?

Dejan Kupnik, Pavel Skok

Emerg Med J 2007;**24**:614–617. doi: 10.1136/emj.2006.044081

Capnometry is a non-invasive monitoring technique which allows fast and reliable insight into ventilation, circulation, and metabolism. In the prehospital setting it is mainly used to confirm correct tracheal tube placement. In addition it is a useful indicator of efficient ongoing cardiopulmonary resuscitation due to its correlation with cardiac output, and successful resuscitation. It helps to confirm the diagnosis of pulmonary thromboembolism and to sustain adequate ventilation in mechanically ventilated patients. In patients with haemorrhage, capnometry provides improved continuous haemodynamic monitoring, insight into adequacy of tissue perfusion, optimisation within current hypotensive fluid resuscitation strategy, and prevention of shock progression through controlled fluid administration.

Managing the critically ill patient is one of the most challenging aspects of prehospital emergency care. The nature of work in such circumstances demands methods which rapidly recognise patients at risk. Capnometry, as a non-invasive method, offers fast and reliable insight into certain pathophysiological processes and aids in the clinical evaluation of critically ill patients, especially those in cardiac arrest and requiring cardiopulmonary resuscitation, and assists in the monitoring patients after endotracheal intubation and mechanical ventilation.

For many years aggressive fluid resuscitation has been a mainstay in the early treatment of the patient in haemorrhagic shock. But in recent years hypotensive fluid resuscitation before surgical repair of uncontrolled thoraco-abdominal haemorrhage has proven to be superior over aggressive fluid resuscitation. Methods like capnometry could allow better additional discrimination between therapeutic demands, leading to more precise fluid resuscitation in the prehospital setting. In fact, changes in end tidal carbon dioxide partial pressure (PETCO₂) promptly reflect circulatory compromise and as such help in timely recognition of patients at risk.

BASIC PHYSIOLOGY OF CARBON DIOXIDE AND END TIDAL CARBON DIOXIDE MEASUREMENTS

Carbon dioxide (CO₂) is the product of cellular aerobic metabolism. It diffuses easily from cells into blood and erythrocytes and is transported to the lungs by venous blood through the function of cardiac output. Under normal conditions of

circulation and ventilation the partial pressure of CO₂ approaches 50 mm Hg at the level of tissues, and 45 mm Hg in the venous blood. The difference between the latter and alveolar CO₂ partial pressure (PACO₂) which is around 40 mm Hg, is responsible for diffusion of CO₂ into the alveoli. There, CO₂ is eliminated from the body with minute ventilation. Arterial CO₂ partial pressure (PaCO₂) normally varies from 35–45 mm Hg.¹

Approximately 7% of CO₂ is dissolved in blood. The rest of it diffuses into erythrocytes where 23% of CO₂ reversibly bonds to haemoglobin (carbaminohaemoglobin), and the rest forms carbonic acid with water after carbonic anhydrase mediated enzymatic reaction. Carbonic acid dissolves into hydrogen and bicarbonate ion. The former is buffered by deoxygenated haemoglobin in tissues, and the latter can be exchanged with plasma chloride ions. In lung capillaries these reactions are reversed: bicarbonate enters erythrocytes, binds to hydrogen ions recently released from oxygenated haemoglobin, and dissolves into CO₂ and water.

Capnometry is a measurement of end tidal CO₂ partial pressure (PETCO₂). PETCO₂ closely approximates PACO₂ at the end of normal expiration in conditions with normal perfusion and ventilation,² and therefore makes the difference between PACO₂ and PETCO₂ minimal.³ In healthy individuals there is essentially no alveolar dead space which represents the volume of gases in non-perfused alveoli. This means that PETCO₂ equals PACO₂ and with correct sampling P(a–A)CO₂ difference equals P(a–ET)CO₂ difference, which makes PETCO₂ a good estimate of PACO₂.

Factors that influence PETCO₂ can be divided in four groups: metabolism, circulation, ventilation, and technical errors. When production (metabolism) and transfer (circulation) of CO₂ are constant, PETCO₂ values reflect changes in ventilation, and vice versa.^{1,2} Under normal conditions of ventilation and circulation in the awake individual, there is a 0–2 mm Hg gradient between PETCO₂ and PACO₂, and the gradient increases up to 6 mm Hg in intubated patients under general anaesthesia.⁴

CAPNOMETRY IN MONITORING AND DIAGNOSTICS

Along with ventilatory and circulatory monitoring, changes in PETCO₂ allow early diagnosis of

Abbreviations: CO₂, carbon dioxide; CPR, cardiopulmonary resuscitation; PACO₂, alveolar carbon dioxide partial pressure; PaCO₂, arterial carbon dioxide partial pressure; PETCO₂, end tidal carbon dioxide partial pressure; PvCO₂, venous carbon dioxide partial pressure

See end of article for authors' affiliations

Correspondence to: Dejan Kupnik, MD, Cesta proletarskih brigad 62, 2000 Maribor, Slovenia; dejan.kupnik@triera.net

Accepted 22 May 2007

hypermetabolic episodes like hyperthermia after general anaesthesia, since greater tissue production of CO₂ in such circumstances leads to increased delivery of CO₂ to the lungs.¹ Measurement of PETCO₂ during mechanical ventilation has been used traditionally in evaluating the adequacy of ventilation because of its relationship with PaCO₂. In the prehospital setting capnometry is also used to confirm correct tracheal tube placement,⁵ to monitor efficacy of cardiopulmonary resuscitation (CPR),⁶ and to confirm the return of spontaneous circulation after successful CPR.^{7, 8}

Confirmation of correct endotracheal tube placement is of paramount importance because oesophageal intubation increases morbidity and mortality if unrecognised. Capnometry, and capnography with typical waveform in particular, are the most reliable methods to confirm tracheal intubation.⁵

In cardiac arrest PETCO₂ values decrease abruptly almost to zero. They increase with the start of chest compressions during CPR, and as spontaneous circulation resumes they return to normal or above normal levels.⁹ As the cardiac arrest occurs, cardiac output no longer serves as a conduit between the large peripheral tissue compartment where production of CO₂ is generated, and the smaller central pulmonary compartment where elimination of CO₂ takes place. The result is accumulation of CO₂ in the tissues and a drastic fall in alveolar and consequently end tidal CO₂. Therefore initial values of PETCO₂ after endotracheal intubation and resumed ventilation are low. Initiation of chest compressions restores a fraction of normal cardiac output and begins to wash CO₂ out of the peripheral tissue compartment, where CO₂ is now produced in part by aerobic metabolism and in part by anaerobic metabolism. The latter is caused by buffering of metabolic acids by bicarbonate, which leads to carbonic acid production and its dissociation to CO₂ and water.^{10, 11}

Establishing cardiac output with CPR re-establishes CO₂ transport and causes alveolar CO₂ tension to increase which ultimately results in increased PETCO₂ values. But cardiac output produced by closed chest compressions is only a fraction of normal cardiac output, so the PETCO₂ values are usually lower than normal. Return of spontaneous circulation is usually indicated by a rapid increase in PETCO₂ since normal or near-normal cardiac output increases alveolar and therefore end tidal CO₂ partial pressure.⁸ However, initial and final values of PETCO₂ of ≤ 10 mm Hg during CPR proved to predict mortality,^{12, 13} which makes capnometry also an important tool in the prognostication of cardiac resuscitation.^{7, 14} Capnometry in CPR thus provides information about efficacy of chest compressions, return of spontaneous circulation, and prognosis of outcome after cardiac arrest.

Capnometry can also help to differentiate an asphyxic from primary cardiac arrest,^{10, 15} detect waning neuromuscular blockade, and serves as an additional diagnostic tool in pulmonary thromboembolism.^{16, 17} In patients with asphyxia as the cause of cardiac arrest, initial PETCO₂ values after endotracheal intubation are much higher than in patients with primary cardiac arrest due to ventricular fibrillation and/or ventricular tachycardia.¹⁵ An animal study by Bende *et al* reached the same conclusion, explaining that in asphyxial cardiac arrest it is the continued cellular production of CO₂ and continued cardiac output before cardiac arrest that allows delivery of CO₂ to the lungs which ultimately increases alveolar CO₂.¹⁸ This in turn is reflected as high PETCO₂ once ventilation is resumed during CPR. However, the difference in PETCO₂ between primary and secondary cardiac arrest is no longer present after 1 min of CPR, as described in the study by Grmec *et al*.¹⁵ The initial PETCO₂ difference thus serves as a good diagnostic tool for reassuring emergency physicians to take

measures appropriate for asphyxial cardiac arrest, since the hypoxia is a potentially reversible cause of cardiac arrest.¹⁵

In pulmonary thromboembolism PETCO₂ values are characteristically lower than normal because of diminished pulmonary perfusion and increased alveolar dead space, and consequently decreased CO₂ elimination capability. Retention of CO₂ leads to increased venous carbon dioxide partial pressure (PvCO₂) and PaCO₂, which ultimately increases arterial to end tidal CO₂ gradient.⁶ This helps further in reaching a correct diagnosis, especially in silent pulmonary embolism.¹⁷

In haemorrhage, changes in PETCO₂ reflect haemodynamic changes.¹⁹ This allows not only prompt recognition of circulatory compromise but also more precise fluid resuscitation. Capnometry is the only continuous non-invasive monitoring method in the prehospital setting which gives indirect information about tissue perfusion through its correlation with oxygen supply dependency during haemorrhagic shock.²⁰ It is also of value in predicting outcome from major trauma and trauma surgery where P(a-ET)CO₂ difference >10 mm Hg can predict mortality even if blood pressure is normalised.^{4, 21}

PETCO₂ values can be measured in intubated and non-intubated patients,²² even though hyperventilation can alter measurements. The latter is successfully achieved with a nasal cannula,²³ especially in children.²⁴ Measurements taken through a facial mask are less accurate compared to a nasal cannula because of increased dead space, which decreases the amount of CO₂ in expired air.²⁵ Oxygen supplementation can affect measurements of PETCO₂, but not significantly.²⁶

However, there are some limitations in the interpretation of PaCO₂ on the basis of PETCO₂ measurements in critically ill patients. In any cardiorespiratory dysfunction, including breathing patterns which cause incomplete alveolar emptying, and some pharmacological interventions which are used in treating low flow states, P(a-A)CO₂ and consequently P(a-ET)CO₂ difference increases due to ventilation-perfusion mismatch.^{1, 27, 28} These limitations do not support PETCO₂ measurements as a substitute for PaCO₂ measurements.³

Nevertheless, in the prehospital setting it is sudden changes of PETCO₂ that make capnometry an important tool for early detection of mainly circulatory and/or ventilatory compromise in the critically ill, and not the relationship between PETCO₂ and PaCO₂. A sudden decrease in PETCO₂ is caused by sudden hypotension, sudden hyperventilation, massive pulmonary embolism, occlusion of the endotracheal tube, and disconnection or leakage in the artificial ventilatory system, while decreased CO₂ production and hyperventilation cause a more gradual decline in PETCO₂.³ A sudden increase in PETCO₂ is caused by a sudden increase in cardiac output, such as when spontaneous circulation returns after CPR, and following an injection of sodium bicarbonate, while hypoventilation and increased CO₂ production cause more gradual increases in CO₂.³ Therefore, a final interpretation of PETCO₂ must be made after all parameters which affect measurements have been accounted for—that is, ventilation and circulatory parameters, metabolism, and technical errors.

Sublingual capnometry shows great promise for the early recognition of patients at risk of tissue hypoperfusion, hypoxia, and shock progression,²⁹ because it avoids disturbances of measurements caused by hyperventilation.

PETCO₂ as an indicator of haemorrhage and tissue perfusion adequacy

In uncontrolled haemorrhage, cardiac output and blood pressure decrease. In early stages of haemorrhage, cardiac output decreases before changes in blood pressure can be detected. The relationship between cardiac output and PETCO₂,^{7, 19, 30} even in low flow states,³¹ makes PETCO₂ an indicator of changes in cardiac output. Shibutani *et al* and Wahba *et al*

Methods of information retrieval

Data were recovered with the search of articles in online medical databases (Pubmed, Pubmed Central, Medline, Medscape, Cochrane library), and were later retrieved for information processing. Key words used in article searches were capnometry, cardiac output, cardiopulmonary resuscitation, haemorrhagic shock, fluid resuscitation, prehospital setting, and capnometry limitations.

showed that haemodynamic changes lead to immediate changes in $PETCO_2$; this makes $PETCO_2$ a good indicator of an early stage haemorrhage due to early detection of changes in cardiac output.^{32, 33}

According to Fick's principle, cardiac output equals the relationship between oxygen consumption and arteriovenous difference in oxygen concentration. Decreased cardiac output leads to decreased tissue oxygen consumption. Under physiological conditions oxygen consumption is constant because oxygen supply is greater than its consumption. A critical decrease in oxygen supply makes oxygen consumption supply-dependent. This marks the initiation of anaerobic metabolism. Increased lactate, hypercapnia in gastric mucosa blood vessels, and increased arteriovenous difference in CO_2 partial pressure all indicate tissue hypoperfusion and hypoxia at this point.³⁴ Guzman *et al* showed that with constant minute ventilation, changes in $PETCO_2$ correlate well with changes in oxygen consumption in haemorrhagic shock, and these changes also indicate the onset of oxygen supply dependency during haemorrhagic shock.²⁰ Fluid resuscitation increases oxygen consumption due to its increased delivery, which ultimately increases $PETCO_2$.

CAPNOMETRY GUIDED FLUID RESUSCITATION IN UNCONTROLLED INTERNAL HAEMORRHAGE

The strategy of fluid resuscitation in hypovolaemic shock caused by haemorrhage has been thoroughly debated in recent years. Aggressive fluid resuscitation dilutes clot factors, dislodges formed blood clots, and leads to further bleeding, especially in parenchymal organ trauma.³⁵ These factors are associated with increased blood loss and mortality,³⁶ so that aggressive fluid therapy may be harmful to patients with uncontrolled thoraco-abdominal haemorrhage. However, aggressive fluid resuscitation is still considered appropriate in controllable haemorrhage (head and isolated extremity trauma) and in trauma patients without a palpable pulse.³⁷ In patients with uncontrolled thoraco-abdominal haemorrhage, limited or hypotensive fluid resuscitation avoids detrimental effects of an early aggressive resuscitation, decreases mortality, and is currently considered more appropriate for short periods before surgical repair, even at the expense of tissue perfusion.^{36, 38-40}

Nevertheless, at an early stage of haemorrhage blood pressure can still be normal, even with substantial blood loss, and significant delay in fluid resuscitation, based on clinical signs and normal blood pressure measurement alone, can be detrimental because of decreased tissue perfusion and shock progression. Changes in $PETCO_2$ are an immediate reaction to haemorrhage, and any decrease in cardiac output would cause $PETCO_2$ to decrease, indicating haemorrhage in progress. It is an intriguing thought that capnometry guided fluid resuscitation could avoid delay in initiating fluid resuscitation due to prompt detection of these haemodynamic changes. It could also help to enhance tissue perfusion by adjusting further fluid administration, while keeping the blood pressure in appropriate ranges

at the same time. This could significantly improve not only the clinical evaluation but also the fluid administration strategy in patients with uncontrolled haemorrhage.

On the other hand, there is also a possibility of severe under-resuscitation as a potential complication of hypotensive fluid resuscitation strategy in haemorrhagic shock. Capnometry could prevent this complication through prompt detection of a decrease in $PETCO_2$. In order to maintain normal or near normal $PETCO_2$ values, and tissue perfusion as well, increased amounts of infused fluids help to overcome this problem. More research is needed to confirm the usefulness of capnometry in decision making in patients with uncontrolled internal haemorrhage.

CONCLUSION

In the prehospital setting capnometry is an important tool in the diagnosis, monitoring, and prediction of outcome. Even though there are some limitations of $PETCO_2$ monitoring in critically ill patients, sudden changes in $PETCO_2$ promptly reflect circulatory and/or ventilatory compromise. Prompt reaction to acute haemodynamic changes in patients with constant minute ventilation, together with other measures of clinical evaluation, helps in timely detection of patients at risk in the prehospital setting. For that reason capnometry, especially sublingual, can also be an important non-invasive monitoring method in patients with uncontrolled internal haemorrhage in the prehospital setting, together with the control of fluid resuscitation through indirect control over tissue perfusion, especially in limited fluid resuscitation. This could ultimately help to impede shock progression and further bleeding at the same time.

ACKNOWLEDGEMENTS

Authors wish to thank Štefek Grmec, MD, PhD, for constructive criticism and suggestions.

Authors' affiliations

Dejan Kupnik, Center for Emergency Medicine, Prehospital Unit, Ulica talcev 9, Maribor, Slovenia

Pavel Skok, General and Teaching Hospital Maribor, Clinical Department of Internal Medicine, Department of Gastroenterology and Endoscopy, Maribor, Slovenia

Funding: None.

Conflicts of interest: None declared.

REFERENCES

- 1 Trillo G, von Planta M, Kette F. $ETCO_2$ monitoring during low flow states: clinical aims and limits. *Resuscitation* 1994;**27**:1-8.
- 2 Sanders AB. Capnometry in emergency medicine. *Ann Emerg Med* 1989;**18**:1287-90.
- 3 Ontario Ministry of Health. Noninvasive blood gas monitoring: a review for use in the adult critical care unit. Technology Subcommittee of the Working Group on Critical Care, Ontario Ministry of Health. *Can Med Assoc J* 1992;**146**:703-12.
- 4 Tyburski JG, Collinge JD, Wilson RF, *et al*. End-tidal CO_2 -derived values during emergency trauma surgery correlated with outcome: a prospective study. *J Trauma* 2002;**53**:738-43.
- 5 Grmec Š. Comparison of three different methods to confirm tracheal tube placement in emergency intubation. *Intensive Care Med* 2002;**28**:701-4.
- 6 Anderson CT, Breen PH. Carbon dioxide kinetics and capnography during critical care. *Crit Care* 2000;**4**:207-15.
- 7 Gazmuri RJ, von Planta M, Weil MH, *et al*. Arterial PCO_2 as an indicator of systemic perfusion during cardiopulmonary resuscitation. *Crit Care Med* 1989;**17**:237-40.
- 8 Falk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. *N Engl J Med* 1988;**318**:607-11.
- 9 Hatlestad D. Capnography as a predictor of the return of spontaneous circulation. *Emerg Med Serv* 2004;**33**:75-80.
- 10 Gazmuri RJ, Kube E. Capnography during cardiac resuscitation: a clue on mechanisms and a guide to interventions. *Crit Care* 2003;**7**:411-2.
- 11 Johnson BA, Weil MH, Tang W, *et al*. Mechanisms of myocardial hypercarbic acidosis during cardiac arrest. *J Appl Physiol* 1995;**78**:1579-84.
- 12 Grmec Š, Kupnik D. Does the Mainz Emergency Evaluation Scoring (MEES) in combination with capnometry (MEESc) help in the prognosis of outcome from

- cardiopulmonary resuscitation in a prehospital setting? *Resuscitation* 2003;**58**:89–96.
- 13 **Levine LR**, Wayne MA, Miller CC. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. *N Engl J Med* 1997;**337**:301–6.
 - 14 **Mauer D**, Schneider T, Elich D, *et al.* Carbon dioxide levels during pre-hospital active compression-decompression versus standard cardiopulmonary resuscitation. *Resuscitation* 1998;**39**:67–74.
 - 15 **Grmec S**, Lah K, Tusek-Bunc K. Difference in end-tidal CO₂ between aphyxia cardiac arrest and ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest in the prehospital setting. *Crit Care* 2003;**7**:R139–44.
 - 16 **Courtney DM**, Watts JA, Kline JA. End tidal CO₂ is reduced during hypotension and cardiac arrest in a rat model of massive pulmonary embolism. *Resuscitation* 2002;**53**:83–91.
 - 17 **Taniguchi S**, Irita K, Sakaguchi Y, *et al.* Capnometry as a tool to unmask silent pulmonary embolism. *Tohoku J Exp Med* 1997;**183**:263–71.
 - 18 **Bhende MS**, Karasic DG, Karasic RB. End-tidal carbon dioxide changes during cardiopulmonary resuscitation after experimental asphyxial cardiac arrest. *Am J Emerg Med* 1996;**14**:349–50.
 - 19 **Isserles SA**, Breen PH. Can changes in end-tidal PCO₂ measure changes in cardiac output. *Anesth Analg* 1991;**73**:808–14.
 - 20 **Guzman JA**, Lacombe FJ, Najjar A, *et al.* End-tidal partial pressure of carbon dioxide as a noninvasive indicator of systemic oxygen supply dependency during hemorrhagic shock and resuscitation. *Shock* 1997;**8**:427–31.
 - 21 **Deakin CD**, Sado DM, Coats TJ, *et al.* Prehospital end-tidal carbon dioxide concentration and outcome in major trauma. *J Trauma* 2004;**57**:65–8.
 - 22 **Ward KR**, Yealy DM. End-tidal carbon dioxide monitoring in emergency medicine: Part 1: Basic principles. *Acad Emerg Med* 1998;**5**:628–36.
 - 23 **Fukuda K**, Ichinohe T, Kaneko Y. Is measurement of end-tidal CO₂ through a nasal cannula reliable. *Anesth Prog* 1997;**44**:23–6.
 - 24 **Friesen RH**, Alswang M. End-tidal PCO₂ monitoring via nasal cannulae in pediatric patients: accuracy and sources of error. *J Clin Monit* 1996;**12**:155–9.
 - 25 **Loughnan TE**, Monagle J, Copland JM, *et al.* A comparison of carbon dioxide monitoring and oxygenation between facemask and divided nasal cannula. *Anaesth Intensive Care* 2000;**28**:151–4.
 - 26 **McNulty SE**, Roy J, Torjman M, *et al.* Relationship between arterial carbon dioxide and end-tidal carbon dioxide when a nasal sampling port is used. *J Clin Monit* 1990;**6**:93–8.
 - 27 **Prause G**, Hetz H, Lauda P, *et al.* A comparison of the end-tidal-CO₂ documented by capnometry and the arterial pCO₂ in emergency patients. *Resuscitation* 1997;**35**:145–8.
 - 28 **Tang W**, Weil MH, Gazmuri RJ, *et al.* Pulmonary ventilation/perfusion defects induced by epinephrine during cardiopulmonary resuscitation. *Circulation* 1991;**84**:2101–7.
 - 29 **Creteur J**. Gastric and sublingual capnometry. *Curr Opin Crit Care* 2006;**12**:272–7.
 - 30 **Dubin A**, Murias G, Estenssoro E, *et al.* End-tidal CO₂ pressure determinants during hemorrhagic shock. *Intensive Care Med* 2000;**26**:1619–23.
 - 31 **Jin X**, Weil MH, Tang W, *et al.* End-tidal carbon dioxide as a noninvasive indicator of cardiac index during circulatory shock. *Crit Care Med* 2000;**28**:2415–9.
 - 32 **Wahba RW**, Tessler MJ, Beique F, *et al.* Changes in PCO₂ with acute changes in cardiac index. *Can J Anaesth* 1996;**43**:243–5.
 - 33 **Shibutani K**, Muraoka M, Shirasaki S, *et al.* Do changes in end-tidal PCO₂ quantitatively reflect changes in cardiac output? *Anesth Analg* 1994;**79**:829–33.
 - 34 **Guzman JA**, Kruse JA. Continuous assessment of gastric intramucosal PCO₂ and pH in hemorrhagic shock using capnometric recirculating gas tonometry. *Crit Care Med* 1997;**25**:533–7.
 - 35 **Krausz MM**, Bashenko Y, Hirsh M. Crystalloid and colloid resuscitation of uncontrolled hemorrhagic shock following massive splenic injury. *Shock* 2001;**16**:383–8.
 - 36 **Stern SA**. Low-volume fluid resuscitation for presumed hemorrhagic shock: helpful or harmful? *Curr Opin Crit Care* 2001;**7**:422–30.
 - 37 **Fowler R**, Pepe PE. Fluid resuscitation of the patient with major trauma. *Curr Opin Anaesthesiol* 2002;**15**:173–8.
 - 38 **Capone AC**, Safar P, Stezoski W, *et al.* Improved outcome with fluid restriction in treatment of uncontrolled hemorrhagic shock. *J Am Coll Surg* 1995;**180**:49–56.
 - 39 **Xiao N**, Wang XC, Diao YF, *et al.* Effect of initial fluid resuscitation on subsequent treatment of uncontrolled hemorrhagic shock in rats. *Shock* 2004;**21**:276–80.
 - 40 **Stern SA**, Wang X, Mertz M, *et al.* Under-resuscitation of near-lethal uncontrolled hemorrhage: effects on mortality and end-organ function at 72 hours. *Shock* 2001;**15**:16–23.

International Forum on Quality and Safety in Health Care

23–25 April 2008
Le Palais des Congrès de Paris

Call for Abstracts: Submission deadline 3 October 2007

Why submit an abstract?

- Your achievement showcased to key international opinion leaders
 - Communicate your organisation's work
 - Enhance your organisation's profile
 - Network with others in your area/field
 - Share your success, learn from your failures
- For more details and to submit your abstract online visit internationalforum.bmj.com