

Editorials

Errare humanum est - To err is human

“AIDS is not caused by the HIV” Thabo Mbeki
“Iraq has weapons of mass destruction” George W. Bush

Human error, particularly in the practice of medicine, is inescapable and ubiquitous. Error occurs when a planned activity fails to produce its planned outcome, and when the failure is not due to chance. The era of the submissive patient who blindly accepts the opinion and actions of the medical professional is long gone. The information era and the ease of access to information via libraries and the internet have enabled our patients to research our opinions and criticise our actions. Moreover, patients develop expectations of outcome. All this scrutiny of medical professional conduct is further fuelled by the legal profession and media instilling a sense of victimisation and “angst” in the public.

Human error can be viewed in two ways: the *person approach* and the *system approach*. The *person approach* focuses on unsafe and erroneous acts by the individual at “the coal-face”, and allows for the attribution of blame and the meeting out of punishment. This approach views these unsafe acts as arising primarily from aberrant mental processes such as forgetfulness, inattention, poor motivation, carelessness, negligence and recklessness. Countermeasures are mounted mainly at reducing unwanted variability in human behaviour. These measures include poster campaigns that appeal to people’s sense of fear, writing or amending yet another protocol, disciplinary measures, threats of litigation, retraining, naming, blaming and shaming. Blaming individuals, especially if you are the victim of error, is emotionally more satisfying than targeting institutions. Followers of this approach tend to treat errors as moral issues, assuming that “bad things happen to bad people”. This model is a relatively primitive and often counterproductive approach and may be seen as bullying or be interpreted as harassment. Seeking to uncouple a person’s unsafe acts from any institutional responsibility is obviously in the interest of administrators.^{1,2,3}

The *system approach* works on the premise that humans are fallible and errors are to be expected, even in the best organisations. Errors are seen as consequences rather than causes, having their origins not so much in the perversity of human nature as in “upstream”

systemic factors. These include recurrent error traps in the workplace and the organisational processes that give rise to them. Countermeasures are based on the assumption that though we cannot change the human condition, we can change the conditions under which humans work. A central idea is that of system defences. All hazardous technologies possess barriers and safeguards. When an adverse event occurs, the important issue is not who blundered, but how and why the defences failed (the analogy of lining up the holes in slices of Swiss cheese is often referred to).

The *system approach* is subdivided into active failures and latent failures. Active failures are unsafe actions by people in contact with the patient and due to lack of education or skills (or both). Protocolisation and simplification of activities have been employed to attempt to reduce active failures. “Everything should be made as simple as possible, but not simpler.” - Albert Einstein.

The other subdivision of the system approach is that of latent failures, which can best be described as resident factors in a system that may predispose to a set of unfavourable conditions culminating in an error (e.g. fatigue due to understaffing and poor rostering, inappropriately difficult casemix for the attending physician, progressive erosion of safety nets by poor maintenance of equipment, outdated equipment, workplace design inefficiencies or inappropriate alarm settings). Williamson *et al.*,⁴ reported a blood transfusion error that occurred after seven pairs of medical staff had checked the correctness of the procedure.

Donchin *et al.*,⁵ conducted a study to investigate the nature and causes of human errors in the intensive care unit (ICU). A human error was defined as a deviation from standard conduct, as well as addition or omission of actions relating to standard operational instructions or routines of the unit. During 4 months of data collection, a total of 554 human errors were reported. There was an average of 178 activities per patient per day and an estimated number of 1.7 errors per patient per day. The 1.7 errors per day indicate that hospital personnel were functioning at a 99% level of proficiency. However, a 1% failure rate is substantially higher than is tolerated in industry, particularly in hazardous fields such as aviation and nuclear power. Even 99.9% proficiency may not be good enough. If we had to live with 99.9% proficiency, this implies two unsafe plane landings per day at O’Hare airport, Chicago; 16 000 pieces of lost mail every hour and 32 000 bank cheques deducted from the wrong bank account every hour in the United States of America.² In the Donchin *et al* study, for the ICU as a whole, a severe or potentially detrimental error occurred on average twice a day. Physicians and nurses were approximately equal contributors to the number of errors, although nurses performed 84% of the activities.

The study highlights the critical importance of good communication and transfer of information between physicians and nurses. Nurses had error peaks at each shift change, which is another indication of problems in information exchange.

In this issue of the Journal, Einav *et al*, discuss the concept of the "Timeout" - a briefing in pre-operative surgical activity to confirm the correct patient, procedure and body part.⁶ The concept reiterates a team communication process to minimise the occurrence of a person error. For the solo player it is akin to "gathering your thoughts" but allows for cross-check by team members. I believe that most specialists are already making use of this concept in their clinical practice but may omit the "timeout" in situations of stress.

Prevention is better than cure. Risk management is a discipline for dealing with the possibility that some future error will cause harm. It provides strategies, techniques, and an approach to recognising and confronting any threat faced by an organisation in fulfilling its mission^{2,5,7}. The concept/cycle of identifying, measuring, analysing and evaluating, treating, monitoring and reviewing a potential risk is widely used. A risk management strategy familiar to most of us is the maintenance of professional standards (MOPS) program. Currently most MOPS programs are flawed in that they are not mandatory for many specialists (e.g. currently only 62% of registered Intensive Care specialists subscribe to the Joint Faculty of Intensive Care MOPS program), and they under-represent the maintenance of clinical skills. Hopefully MOPS membership will expand and the program will evolve to also encompass skill maintenance. A further concern is the pressure on specialist colleges, by government, to "create" more specialists in a shorter time-frame. Hopefully this will not occur at the expense of standards of training.

Needless to say, medical education and risk management to reduce error are costly exercises.

Medical indemnity societies should offer more support (as some already do) by sponsoring continued education, as they too benefit from the individuals' efforts to reduce error. Considering the financial and political leverage the insurance companies have to sway legislation that can for instance result in one being fined for not securing an unattended vehicle properly – why not assist in risk reduction in the medical arena?

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Elevation of plasma troponin in the critically ill patient

In cardiac and skeletal muscle, troponin I, troponin T and troponin C form a complex of proteins that regulate the calcium-mediated interaction of actin and myosin. An identical troponin C is expressed by both cardiac and skeletal muscle cells, whereas the amino acid sequences of troponin I and T in cardiac muscle differ from the sequences in skeletal muscle.¹ Monoclonal antibodies against cardiac troponin T (cTnT) and cardiac troponin I (cTnI) have been developed that have almost no cross-reactivity with their respective skeletal-muscle isoforms,^{2,3} and are currently the most sensitive and specific biochemical markers of myocardial damage.⁴ Currently, there are several cTnT and cTnI tests marketed which have different upper limits of normal. While the new third generation cTnT test is standardised using a single antibody assay with a clinically relevant cut-off point of about 0.1 ug/L (a level above which myocardial injury diagnosed), the newer cTnI tests have clinically relevant cut-off points varying between 0.1 ug/L and 2 ug/L depending upon the assay used.⁴

In patients with ischaemic myocardial damage, plasma cTnT and cTnI levels increase within 3.5 hr of the onset of chest pain (but are only diagnostically reliable after 6 hr) and remain elevated for at least 6 days (even up to 14 days).⁵ Both are the preferred biological markers for diagnosing myocardial infarction,⁶ and can stratify mortality risk,⁷⁻¹⁰ although an elevated level may not indicate irreversible myocyte damage with experimental¹¹ and clinical studies¹² indicating that troponin can be released in reversible cardiac ischaemia. While cTnI was believed to have a greater specificity

than cTnT in detecting cardiac muscle injury in patients with renal failure, polymyositis/dermatocytis or muscular dystrophy,¹³⁻¹⁵ two recent studies found that elevated levels of cTnT were associated with an accurate prognostication of myocardial ischaemic events or death in the presence of renal failure.^{16,17}

However, while cardiac troponins are useful markers of myocardial injury, they do not define the mechanism of injury,¹⁸ as there are numerous disorders that can cause their elevation in the presence of a normal coronary angiogram.^{15,19,20} For example, elevations in cTnT and cTnI have been reported in severe congestive heart failure,²¹⁻²³ pulmonary embolism,²⁴ myocardial trauma²⁵ (e.g. commido cordis, intracardiac ablation, cardiac surgery),²⁶ myocarditis,²⁷ pericarditis²⁸ (> 90% of whom have ST segment elevation²⁹) cerebrovascular accidents,³⁰ cardiac toxins,³¹ tachycardia (with and without haemodynamic compromise),¹⁹ strenuous exercise³² and with heterophile antibodies,³³ rheumatoid factor,³⁴ fibrin clots,³⁵ microparticles³⁵ and analyser malfunction.³⁶ Nonetheless, cardiac troponin levels do not increase following DC cardioversion of atrial flutter, atrial fibrillation,³⁷ ventricular tachycardia or ventricular fibrillation³⁸ unless one of the prementioned disorders is also present.

Of more relevance to the intensivist are the numerous studies recording elevated plasma levels of cTnI and cTnT levels in the absence of myocardial ischaemia in intensive care patients (with an incidence ranging between 35% - 80%)³⁹⁻⁴² due to hypotension (with a positive correlation to catecholamine administration),⁴³ sepsis,^{44,45} severe sepsis and septic shock.^{46,47} The levels also appear to be a marker of left ventricular dysfunction⁴⁶ severity of illness,⁴¹ and mortality.^{44,48,49} In one study of 20 patients with sepsis, septic shock and SIRS, an elevated plasma level of cTnI (median 0.57 µg/L; 0.17 - 15.4) was recorded in 17 patients (85%) in the absence of an acute coronary syndrome.⁵⁰ In another study of 46 patients with early septic shock, cTnI levels of 0.4 µg/L or more were recorded in 50% of patients and cTnT levels of > 0.1 µg/L or more were recorded in 36% of patients.⁴⁶ In a study of 19 patients with severe sepsis/septic shock cTnI levels of 0.5 µg/L or more were recorded in 11 patients (58%), all of whom received catecholamine infusions, and correlated with the degree and duration of hypotension,⁴³ and in a study of 58 consecutive critically ill patients admitted for reasons other than acute coronary syndrome (with relevant coronary artery disease excluded by subsequent stress echocardiography or autopsy) of the 55% who had elevated cTnT and cTnI levels, compared with patients with a normal troponin levels, there was a higher mortality (22.4% c.f. 5.2%), lower left ventricular ejection fraction, and higher mean TNF alpha and IL-6 levels.⁵¹

So while a raised cTnT or cTnI level is accepted for the diagnosis of an acute coronary syndrome,^{52,53} unless the characteristic clinical features of chest pain (e.g. central in location, radiating up to neck, jaw or down the outer side of the left arm and crushing or restrictive in nature) and ECG features of cardiac ischaemia (e.g. ST segment elevation, Q waves, deep ST depression or T inversion, recent LBBB) are also present, the diagnosis of an acute coronary syndrome based only on an elevated cTnT or cTnI level in the critically ill patient (who are then treated with antithrombin and antiplatelet therapy) could easily be wrong.

The appropriate therapeutic approach to patients with an elevated cardiac troponin level caused by sepsis or septic shock is unknown, although it would appear prudent to reduce the myotoxic effects of disease (e.g. infection, infarction or necrosis), metabolic abnormalities (e.g. hypokalaemia, hypomagnesaemia, hypercalcaemia, hypophosphataemia, anaemia, hypoxia, etc) and therapy (e.g. excessive use of vasoactive agents).

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A tribute to Dr. Geoff Clarke

The following is an excerpt from the citation delivered on the occasion of the award of the inaugural Joint Faculty of Intensive Care Medicine medal to Dr. Geoff Clarke in June 2005.

It is with great pleasure that I provide the following on the occasion of the award of the inaugural Joint Faculty of Intensive Care Medicine (JFICM) Medal to Dr. Geoffrey Malcolm Clarke. It is indeed a great privilege to honour an Intensivist who has not only seen the recognition of Intensive Care Medicine as a respected and valued specialty in Australasia, but has also been dedicated to its growth and development for over thirty years.

Geoff was born in Perth and graduated from the University of Western Australia in 1964. He was the recipient of several undergraduate awards. He then undertook specialist training in Anaesthesia, including a period as a research fellow, in the United Kingdom, attaining the DA in 1966 and the FFARCS in 1968. He returned to Royal Perth Hospital to take up a senior registrar position in the Intensive Care Unit (ICU) in 1969. In 1970, at the age of 29 years, Geoff was appointed Head of the Department of Intensive Care. He held this position until his retirement in 2003. From 1995 to 2002 he was also the Director of the Division of Critical Care. Geoff enjoyed the support and loyalty of his staff during his entire tenure as Head of Department. There are not many clinical leaders about whom this can be truthfully said. Similarly it would be difficult to find anyone who ever saw Geoff lose his temper, despite working a full clinical roster in addition to his administrative duties. There are those contend that if Geoff got angry with you, then it was definitely deserved.

Geoff Clarke achieved an extraordinary balance in his career, as well as his personal life (more about that later). He accumulated the required postgraduate qualifications – FFARCS in 1975, endorsed in Intensive Care in 1981, FANZCA in 1992, FFICANZCA in 1993 and FJFICM in 2002. He became known as and is respected as an excellent clinician. Taking very good care of his patients has been the centrepiece of his career. His sage advice and clinical opinion are sought after and remain an inspiration and example to those who have followed him in the practice of Intensive Care Medicine. It is not an understatement to say that he trained a generation of Intensivists in Perth. As a leader in the specialty, Geoff Clarke is an example and inspiration to a much wider circle than just Perth through his activities in national bodies. He was President of ANZICS from 1977-1978. He was a Member of Council of ANZCA from 1993 – 1997, Chairman of the Section of Intensive Care (ANZCA) in 1993, inaugural Dean of the Interim board, Faculty of Intensive Care in 1993-1994 and Elected Dean of the Faculty of Intensive Care from 1994 to 1997. There is some suspicion that it is no coincidence that the blue and gold colours in the ceremonial wear of the Faculty,

determined while Geoff was Dean, are identical to the colours of the West Coast Eagles AFL team!

Geoff is a sought-after teacher. He has taught at all levels (undergraduate, post-graduate and to nurses and allied health staff) and has been invited to lecture all around the world. His counsel has been sought in difficult clinical situations and in court cases in several countries. Related to the teaching aspect of his career, Geoff was also an examiner in the Intensive Care final examinations from 1980 – 1991, and was Chairman of the Panel of examiners from 1987 – 1991.

By now you have the picture of a superb clinician, a respected leader in his own department and hospital and an opinion-leader in the national Intensive Care Medicine bodies in Australasia. Geoff also found time to do a huge amount of committee work and contribute to many societies. If this was not enough, he managed to publish numerous papers and contributed chapters to many books. All of these aspects contributed to an excellent and balanced career.

Along the way Geoff Clarke has received significant recognition in the form of the Inaugural Royal Perth Hospital Outstanding Service Award in 1988, the Robert Orton Medal for outstanding service to Anaesthesia (awarded by ANZCA) in 1996, the Order of Australia (AM) for services to, patient care, teaching and training in intensive care in 1998 and the AMA(WA) award for outstanding services to Medicine in Western Australia in 2000. In 2003 his name was placed on the Honour Role of ANZICS for his

contribution to the development of Intensive Care Medicine in Australia and New Zealand.

You would be excused for thinking that Geoff had no time for anything besides his busy career. However Geoff has a very full private life. He has always been supported by his loving wife Susan, herself a nurse and a teacher who achieved a B.A. and Dip. Ed., whilst undertaking the lion's share of bringing up their four children; Kate, Ben, Kirsty and Felicity. Geoff's interests extend to farming at York, art history, painting in watercolours, home brewing, gardening and after-dinner speaking. More recently he has taken up deep-sea fishing, and is a crewmember for another illustrious Perth Intensivist, Dr. John Weekes.

As Fellows of the Joint faculty of Intensive Care Medicine we are indeed fortunate to belong to the first training and accreditation body for Intensive Care Medicine in the world. The Joint Faculty and its Fellows owe much to the giants who laid the foundations and continue to build this organisation – names such as Spence, Trubuhovich, Fisher, Wright, Duncan, Hawker and Matthews, among others. Geoff Clarke walks tall among these giants!

I present to you Dr. Geoffrey Malcolm Clarke, an extremely worthy recipient of the inaugural JFICM Medal.

Dr. P. V. van Heerden
Chief Editor