# Reducing patient time in the emergency department

*Most of the solutions lie beyond the emergency department*

HOSPITALS REPRESENT essential infrastructure. Engineers who run an essential community resource such as the water supply system at 100% of capacity might expect to lose their jobs the first time consumers had to queue to use a tap. In contrast, some hospital funding models include activity targets that reward administrators who run at 100% of capacity — a level that guarantees queuing in the emergency department for coronary care beds and other critical inpatient services. The community accepts the use of price and denial (eg, restrictions of hours or allowed uses) as a rationing mechanism for the water supply, but not for hospital beds — queuing is the only rationing method currently accepted in the hospital system. Rationing is an essential feature in modern medicine,[1] and queuing has long been used to ration elective services. But queuing is fundamentally an inefficient means of rationing care for time-critical illness.

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Access block — the inability of patients in the emergency department (ED) to access hospital beds — is the major issue currently facing emergency medicine in Australasia[2] and, indeed, the whole Western world. Given a fixed physical resource and a relatively fixed labour force, increased average total ED time,[3] also called ED length of stay (EDLOS), will decrease the resources available for providing care to acutely ill patients. Access block decreases access to emergency care (eg, measured as waiting time),[4] and the resultant overcrowding is associated with adverse outcomes for ED patients.[5,6] It is certainly in the interests of ED staff and patients to decrease EDLOS.

The article by Liew et al in this issue of the Journal *page 524*[7] adds to the growing evidence for an association between EDLOS and outcomes beyond the ED.[8] The authors used a multivariate approach to study the relationship between EDLOS, other confounding factors, and subsequent inpatient length of stay in three Melbourne metropolitan hospitals. They found a positive association between EDLOS and inpatient length of stay after adjusting for casemix, time of presentation, and patient age. Whether this relationship is causal is a subject for further study. The Australasian College for Emergency Medicine has committed resources to such research through the Emergency Medicine Research Foundation.

From an administrative perspective, the underlying mechanism is less important than the result. Increased inpatient length of stay after correction for casemix is financially undesirable: it is in the interest of hospital management to reduce both EDLOS and inpatient length of stay.

Emergency departments are specialist multidisciplinary units with expertise in managing acutely unwell patients for the first few hours in hospital. Neither the facilities (generally poor privacy, small trolleys, 24-hour lighting) nor the staff are appropriate for providing longer term inpatient care. Very few patients who require an inpatient bed benefit from staying in the ED longer than 4 hours, and no ED benefits by caring for patients beyond this time. If the 30% or so of patients who are admitted spend twice as long in the ED, this represents a 30% increase in workload for ED staff with no change in conventional measures of activity (presentations, admission rate).

Steadily worsening delays in accessing inpatient beds have been documented in many EDs over the past decade,[9] suggesting there might be an underlying incentive such as increased efficiency in a different part of the health system. This research demonstrates an association with increased opportunity costs rather than any benefits.

There is a clear need to reduce EDLOS for patients, but most of the solutions lie beyond the ED.[10] Changes within the ED can mitigate the effects of increased workload, but, because of access block, they cannot shorten EDLOS.

Much can be done to improve our hospital systems, including use of protocols for common conditions, transparent bed-management processes, and a focus on efficient use of the available beds, particularly through admission and discharge planning. Clinicians must be willing to trial different methods of management, such as treatment in the home and accelerated discharge, and to evaluate the outcomes rigorously.[10] The study by Liew et al identifies the elderly as a group with the greatest potential for effective intervention. There have been significant achievements and more can be expected, but process change will not completely address the underlying mismatch between demand for inpatient beds and resources available.

The primary problem is the lack of acutely available beds.[11,12] The scarcer those empty beds become, the more difficult they are to access.

Queuing for care at the entrance to ED is managed by triage, which stratifies patients by urgency — the most time-critical cases have the shortest queues. Queuing for a bed at the exit of ED is managed by bed allocation, which tends to stratify patients by their nursing load — the least intensive cases generally have the shortest queues. Elementary queuing theory predicts the accumulation of patients, but the daily variation in emergency medical activity has for too long allowed both emergency staff and others to assume the ED has "rubber walls" and that the marginal cost of the ED absorbing additional care to inpatients is low. The study by Liew et al[7] and other studies on the effects of overcrowding[5,6,13] now provide clear evidence that this is not the case.

Emergency departments are expert at triage to achieve "the greatest good for the greatest number", but, when prioritising, even emergency physicians are reluctant to consider denying care to patients with whom they have begun a therapeutic relationship. EDs are faced with the ridiculous situation of providing many hours of care to patients whose conditions were urgent on arrival but stable

after treatment, while potentially unstable patients of similar initial urgency languish in the waiting room or in an ambulance for want of an ED trolley and nurse. It is time that hospitals addressed this inequity: restricting the access and quality of initial care because of inability to provide timely later care is ethically dubious[14] and is likely to lead to adverse outcomes and medicolegal exposure.

These weighty issues cannot be addressed by EDs alone. Demand for emergency services has increased[9] while bed numbers have decreased,[15] and demographic projections indicate that these trends are unlikely to reverse. Unless practices change, our EDs will cease to function in their designated role, and will instead inappropriately spend most of their resources providing care to patients who should be in inpatient beds. Hospitals, communities, and government must debate and decide the allocation of resources to EDs and wards and agree on a sensible approach to providing appropriate care in both environments. The debate is no longer about the level of resources our EDs deserve, but rather about how to ensure that ED resources are directed to those who need them — the patients in the waiting room.

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# Population genetic screening for hereditary haemochromatosis

*Even for a simple genetic condition, screening the general population is not straightforward*

HEREDITARY HAEMOCHROMATOSIS has been touted as the "poster child" for public health genetics. Most cases of haemochromatosis are due to homozygosity for a single mutation leading to iron overload. It is considered to be an ideal candidate for population genetic screening because genetic susceptibility is common, testing is inexpensive, and iron studies can detect early stages of disease. Most importantly, venesection is a simple and effective way to both prevent and manage the potential sequelae of iron overload, which include severe fatigue, arthritis, impotence, cirrhosis, diabetes, and cardiomyopathy. However, even though most cases of haemochromatosis are due to a single mutation, it is still unclear how many people homozygous for this mutation will develop serious disease. Consequently, there is uncertainty as to the benefit of screening.

Following characterisation of the *HFE* gene in 1996,[1] genetic testing for hereditary haemochromatosis has become available. In Australia, Medicare claims for testing for genetic susceptibility to hereditary haemochromatosis have risen from 14 414 in 1999 to almost 30 000 in 2002.[2] It is predicted that about one in 200 Australians are homozygous for the C282Y mutation, which accounts for about 90% of cases of hereditary haemochromatosis identified to date in high-risk families.[3]

A person carrying two copies of the C282Y mutation is at risk of developing iron overload and subsequent disease. However, like all diseases, haemochromatosis is defined by pathology, and a person does not have hereditary haemochromatosis unless body iron stores, as reflected by abnormal iron indices (serum ferritin and fasting transferrin saturation), are elevated. There is a wide spectrum of potential consequences of iron overload, and while about 60% of C282Y homozygotes will eventually develop iron overload,[3,4] it is not known what proportion will progress to serious clinical disease. The answer is complicated by the long latency for development of disease (probably many years) and the possible modifying effects of sex, diet, environment and other genes.

Recent population-based studies have shed light on disease expression in people genetically susceptible to hereditary haemochromatosis. In the Busselton study, 16 homozygotes were identified from a sample of 3011 adults (1 in 188) with a median age of 52.7 years (range, 20–79 years).[3] Twelve were not previously aware of their genetic risk, and of these, seven had elevated serum ferritin levels and the four with normal iron studies were premenopausal women. Half of the original 16 had clinical features consistent with hereditary haemochromatosis, although the prevalence of symptoms in non-homozygotes of the same age was not presented for comparison.[3]

A recent study in California identified 152 homozygotes from a sample of 41 038 individuals (1 in 270) with mean age 57 years (SD, 14).[4] Among these homozygotes, 76% of men and 54% of women had raised serum ferritin levels.