evidence is synthesised and packaged, barriers to making evidence based decisions still exist.[12] The creation of meta-search engines that integrate the main evidence resources (through collaboration among the various publishers) could allow effective and efficient searches and facilitate rapid completion of the search cascade.

Evidence based diagnosis needs more primary evidence on diagnosis, more systematic reviews, and appropriate tools to translate the evidence into action. The challenge to clinicians, educators, researchers, funders, journal editors, and publishers is to work together to make this happen. Doctors and other clinicians should demand action now.

Sharon E Straus *associate professor*

(sharon.straus@utoronto.ca)

Department of Medicine, University of Calgary, Calgary AB, T2N 4N1

Competing interests: SES is an associate editor of the journal *EBM* and ACP Journal Club. The BMJ Publishing Group publishes *Clinical Evidence*.

1 Bossuyt PM, Irwig L, Craig J, Glasziou P. Comparative accuracy: assessing new tests against existing diagnostic pathways. *BMJ* 2006;332:1089-92.
2 Bates SM, Ginsberg JS, Straus SE, Rekers H, Sackett DL. Criteria for evaluating evidence that lab abnormalities are associated with the development of venous thromboembolism. *CMAJ* 2000;163:1016-21.
3 McAlister FA, Straus SE, Sackett DL on behalf of the CARE-COAD1 Group. Why we need large, simple studies of the clinical examination: the problem and a proposed solution. *Lancet* 1999;354:1721-4.
4 Lijmer JG, Mol BW, Heisterkamp S, Bonsel GJ, Prins MH, van der Meulen JH, et al. Empirical evidence of design-related bias in studies of diagnostic tests. *JAMA* 1999;282:1061-6.
5 Mallett S, Deeks JJ, Halligan S, Hopewell S, Cornelius V, Altman DG. Systematic reviews of diagnostic tests in cancer: review of methods and reporting. *BMJ* 2006;333:413-6.
6 Haynes RB. Of studies, syntheses, synopses, and systems: the "4S" evolution of services for finding current best evidence. *ACP J Club* 2001;134:A11-3.
7 Haynes RB, Wilczynski NC for the Hedges Team. Optimal search strategies for retrieving scientifically strong studies of diagnosis from MEDLINE: analytical survey. *BMJ* 2004;328:1040-4.
8 Leefland MMG, Scholten RJPM, Rutjes AWS, Reitsma JB, Bossuyt PMM. Use of methodological search filters to identify diagnostic accuracy studies can lead to the omission of relevant studies. *J Clin Epidemiol* 2006;59:234-40.
9 Sackett DL, Straus SE. Finding and applying evidence during clinical rounds. The evidence cart. *JAMA* 1998;280:1336-8.
10 Cabana MD, Rand CS, Powe NR, Wu AW, Wilson MH, Abboud PA, et al. Why don't physicians follow clinical practice guidelines? A framework for improvement. *JAMA* 1999;282;1458-65.
11 Straus SE, Richardson WS, Glasziou P, Haynes RB. *Evidence-based medicine: how to practice and teach EBM*. London: Elsevier, 2005.
12 Guyatt G, Rennie D. *Users' guides to the medical literature*. Chicago: American Medical Association, 2001.

doi 10.1136/bmj.38945.464722.80

Early intervention in acute renal failure

Give intravenous fluids, not loop diuretics

Research p 420

Oliguria and a rise in the plasma urea concentration are normal physiological responses to the haemodynamic changes associated with hypovolaemia, cardiac failure, or sepsis. Clinical decision making during the ensuing hours may determine whether a patient makes a speedy recovery or develops the serious complication of established acute renal failure. This condition still has a mortality of about 50%, despite recent improvements in clinical practice.[1] A meta-analysis in this week's *BMJ* by Ho and Sheridan reviews the evidence on one commonly used and cheap intervention—the loop diuretic furosemide (frusemide)—but finds it to be of little use in preventing or treating acute renal failure.[2]

Acute renal failure occurs in a variety of different circumstances and can complicate pre-existing chronic renal failure. The traditional split into prerenal, renal, and postrenal causes of acute failure is useful, if only to remind doctors of the need for a systematic approach to diagnosis and management of the underlying cause. It is appropriate that prerenal causes come first, because the treatment of hypovolaemia to maintain renal perfusion is the only reliable means of renal protection,[3] and intravenous fluids will probably need to be tried for most patients. This includes elderly patients, in whom comorbidity may complicate the picture, and patients with oedema, who may none the less have depletion of intravascular fluids.

Typically, junior members of the medical staff are responsible for the initial management of these patients. These doctors need early induction into a method of rapid clinical assessment, such as the acute life threatening emergencies: recognition and treatment (ALERT) course.[4] Doctors should take a pragmatic and prompt approach to intravenous fluid replacement, based on the patient's blood pressure, capillary refill time, and venous filling. They must watch out for and treat life threatening complications, such as hyperkalaemia, and catheterise the patient to measure the volume of urine hourly.

If the patient remains oliguric after correction of hypovolaemia, the next step is to distinguish between intrinsic renal pathology and obstructive causes. Acute renal failure may be the result of obstruction of the urinary tract, particularly in patients with only one functioning kidney. While total anuria suggests obstruction, the presence of oliguria or even polyuria does not exclude it.[5]

An ultrasound examination of the renal tract is warranted, mainly to identify obstructive changes and also to measure the bipolar length of the kidneys. Small kidneys suggest chronic renal disease. Ultrasound examination can be misleading, however, and additional imaging may be required.[6] Obstruction should be identified promptly so that it can be treated with urinary catheterisation, ureteric stenting, or nephrostomy. Delay can result in permanent but avoidable renal injury.

The immediate cause of acute renal failure may seem obvious, but contributory factors such as nephrotoxic drugs, metabolic disturbance (including hypercalcaemia, hyperuricaemia, and paraproteinaemia), and occult sepsis should not be overlooked. Epidemics of acute renal failure may follow outbreaks of haemolytic uraemic syndrome associated with diarrhoea, and other infections such as malaria and leptospirosis may cause renal failure by diverse mechanisms.

The more common causes in any location may change over time, as was noted in one unit in the

BMJ 2006;333:406–7

United Kingdom between 1956 and 1988, when a relative reduction in acute renal failure due to obstetric and traumatic causes and an increase in cases in elderly patients with complicated medical and surgical conditions were seen.[7] When the cause of acute renal failure is not obvious, medical teams must consider unusual or rare explanations—such as allergic interstitial nephritis, acute glomerulonephritis, and vasculitis—conditions that require urgent assessment by a specialist and aggressive treatment.

Renal specialists are not necessary, however, for patients to receive continuous renal replacement therapy by pumped venovenous haemofiltration systems.[8] Continuous renal replacement allows prompt initiation of treatment at the earliest stages of renal dysfunction by anaesthetic staff in the intensive therapy unit. Patients with acute renal failure tend to fall under the care of either nephrologists or anaesthetists, and this split has made it hard to conduct clinical trials during the early stages of the illness.

The meta-analysis by Ho and Sheridan in this issue is therefore timely, and provides a valuable assessment of the role of loop diuretics in the prevention and treatment of acute renal failure.[2] The use of loop diuretics has a theoretical basis and some support from animal studies, but the evidence from clinical studies is not strong. The results of the meta-analysis show that furosemide has no clinical benefit in the prevention or treatment of established acute renal failure.

In normal practice many clinicians, including nephrologists, think that furosemide may help increase urine output in acute renal failure, ease the management of fluid balance, and reduce the degree of hyperkalaemia.[9] The evidence from Ho and Sheridan's paper—that furosemide does not improve mortality, does not reduce the need for renal replacement therapy or the number of dialysis sessions required, and may increase the risk of ototoxicity—must temper this opinion. The priorities in treating acute renal failure are to optimise fluid balance, treat underlying causes, and initiate renal replacement therapy at the appropriate time.

David N Bennett-Jones *consultant nephrologist*

(David.Bennett-Jones@ncumbria-acute.nhs.uk)

North Cumbria Acute Hospitals NHS Trust, West Cumberland Hospital, Whitehaven CA28 8JG

Competing interests: None declared.

1 Bagshaw SM, Laupland KB, Doig CJ, Mortis G, Fick GH, Mucenski M, et al. Prognosis for long-term survival and renal recovery in critically ill patients with severe acute renal failure: a population-based study. *Crit Care* 2005;9:R700-9. Epub 2005 Oct 25.
2 Ho KM, Sheridan DJ. Meta-analysis of frusemide to prevent or treat acute renal failure. *BMJ* 2006;333:420-3.
3 Duke GJ. Renal protective agents: a review. *Crit Care Resusc* 1999;1:265-75.
4 Smith GB. *ALERT—acute life-threatening events: recognition and treatment. A multiprofessional course in care of the acutely ill patient.* 2nd ed. University of Portsmouth, 2003.
5 Rose BD. Urine output in urinary tract obstruction. In: Rose BD, ed. *UpToDate.* Waltham, MA, 2006.
6 Amis ES Jr, Cronan JJ, Pfister RC, Yoder IC. Ultrasonic inaccuracies in diagnosing renal obstruction. *Urology* 1982;19:101-5.
7 Turney JH, Marshall DH, Brownjohn AM, Ellis CM, Parsons FM. The evolution of acute renal failure, 1956-1988. *Q J Med* 1990;74:83-104.
8 Canaud B, Garred LJ, Christol JP, Aubas S, Beraud JJ, Mion C. Pump assisted continuous venovenous hemofiltration for treating acute uremia. *Kidney Int Suppl* 1988;24:S154-6.
9 Firth J. Acute renal failure. In: Warrell DA, Cox TM, Firth J, eds. *Oxford textbook of medicine.* 4th ed. Oxford: Oxford University Press, 2003;3:255-6.

doi 10.1136/bmj.38945.596215.80

Exempting mental health units from smoke-free laws

Would worsen health inequalities for people with mental health problems

An estimated five million deaths worldwide will occur from tobacco consumption in 2006. This figure is projected to reach 10 million deaths annually by 2020.[1] Smoking is also the largest single cause of preventable illness and premature death in the United Kingdom, with 106 000 people dying of smoking related diseases in 2002[2] and more than 10 000 dying each year as a result of passive smoking.[3] The Health Act 2006 will make all enclosed public and work places in England and Wales smoke-free environments and represents an important step forward for public health.

Consultation on exemptions to the Health Act continues until October 2006. Among proposed exclusions are mental health units that provide long term accommodation (defined as not less than six months) as well as prisons, care homes, and hospices. We applaud the government for not exempting mental health settings in their entirety as has happened in some other countries that have introduced smoke-free legislation. Making mental health units smoke free ensures improved physical health of mental health patients and protection of staff and patients from the effects of environmental tobacco smoke.

The prevalence of smoking is high among people with mental health problems. Nearly three quarters of people with schizophrenia, affective psychosis, and other mental health disorders who live in mental health settings are smokers,[4] and they are more likely to be heavier and more dependent smokers than the general population.[5] The prevalence of smoking is also significantly higher among people with diagnosed mental health problems who live in private households than in those without such a diagnosis, and a clear relation exists between the prevalence of smoking and the number and severity of depressive or anxiety symptoms.[6]

The consequence of these higher levels of smoking is a substantially greater risk of premature death from smoking related diseases than is seen in the general population.[5] In a study from Finland, people with schizophrenia had an almost 10-fold greater risk of death from respiratory disease compared with the general population.[7] The high levels of smoking among

BMJ 2006;333:407–8