BEST EVIDENCE TOPIC REPORTS

Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary

Edited by K Mackway-Jones

Best evidence topic reports (BETs) summarise the evidence pertaining to particular clinical questions. They are not systematic reviews, but rather contain the best (highest level) evidence that can be practically obtained by busy practising clinicians. The search strategies used to find the best evidence are reported in detail in order to allow clinicians to update searches whenever necessary.

The BETs published below were first reported at the Critical Appraisal Journal Club at the Manchester Royal Infirmary.[1] Each BET has been constructed in the four stages that have been described elsewhere.[2] The BETs shown here together with those published previously and those currently under construction can be seen at http://www.bestbets.org.[3] Six topics are covered in this issue of the journal

- Frusemide or nitrates in acute left ventricular failure
- Bypass or external rewarming after hypothermic cardiac arrest
- Using intravenous adenosine in asthmatics
- First ECG in chest pain
- Timing of aspirin administration in acute myocardial infarction
- CPAP in acute left ventricular failure

1 Carley SD, Mackway-Jones K, Jones A, *et al*. Moving towards evidence based emergency medicine: use of a structured critical appraisal journal club. *J Accid Emerg Med* 1998;**15**:220–2.
2 Mackway-Jones K, Carley SD, Morton RJ, *et al*. The best evidence topic report: A modified CAT for summarising the available evidence in emergency medicine. *J Accid Emerg Med* 1998;**15**:222–6.
3 Mackway-Jones K, Carley SD. bestbets.org: Odds on favourite for evidence in emergency medicine reaches the worldwide web. *J Accid Emerg Med* 2000;**17**:235–6.

Frusemide or nitrates in acute left ventricular failure

Report by Annette Johnson, *Specialist Registrar*
Search checked by Kevin Mackway-Jones, *Consultant*

Clinical scenario
An 80 year old man is brought into the emergency department in the early hours of the morning with acute shortness of breath. He is pale, clammy and very distressed. You diagnose acute left ventricular failure. You have heard that frusemide may increase vascular resistance and wonder whether nitrates should be used instead.

Three part question
In [patients with acute left ventricular failure] is [frusemide better than nitrates] at [reducing symptoms and avoiding the need for intubation]?

Search strategy
Medline 1966–09/00 using the OVID interface. ([exp heart failure, congestive OR exp ventricular dysfunction, left OR left ventricular failure.mp OR exp pulmonary edema OR pulmonary edema.mp OR pulmonary oedema.mp] AND [nitrate$.mp OR exp nitroglycerin OR nitroglycerin.mp OR gtn.mp OR glyceryl trinitrate.mp OR exp isosorbide dinitrate OR isosorbide dinitrate.mp OR isosorbide mononitrate.mp] AND [exp furosemide OR furosemide.mp OR frusemide.mp OR exp bumetanide OR bumetanide.mp OR exp diuretics OR loop diuretic$.mp]) LIMIT to human AND english.

Search outcome
Altogether 116 papers found of which 112 were irrelevant or of insufficient quality. The remaining four papers are shown in table 1.

Comments
There are still no large trials looking directly at this question. The majority of this work was carried out on patients with recent myocardial infarction.

Clinical bottom line
Nitrates have some benefit as the first line pharmacological treatment of acute pulmonary oedema.

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1 Nelson GI, Silke B, Ahuja RC, *et al*. Haemodynamic advantages of isosorbide dinitrate over frusemide in acute heart failure following myocardial infarction. *Lancet* 1983;i:730–3.
2 Verma SP, Silke B, Hussain M, *et al*. First-line treatment of left ventricular failure complicating acute myocardial infarction: a randomised evaluation of immediate effects of diuretic, venodilator, arteriodilator, and positive inotropic drugs on left ventricular function. *J Cardiovasc Pharmacol* 1987;**10**:38–46.
3 Cotter G, Metzkor E, Kaluski E, *et al*. Randomised trial of high-dose isosorbide dinitrate plus low-dose furosemide versus high-dose furosemide plus low-dose isosorbide dinitrate in severe pulmonary oedema. *Lancet* 1998;**355**:389–93.

Table 1

Author, date and country	*Patient group*	*Study type (level of evidence)*	*Outcomes*	*Key results*	*Study weaknesses*
Nelson GI *et al*, 1983	28 men with radiographic and haemodynamic evidence of left ventricular failure following acute myocardial infarction	PRCT	Left heart filling pressure	Fell with both	Small numbers
			Cardiac output	Fell with frusemide, maintained with isosorbide	
	Frusemide (1 mg/kg) *v* isosorbide dinitrate (50–200 µg/kg/h)		Systemic blood pressure	Transiently rose with frusemide, fell with isosorbide	
Verma SP *et al*, 1987	48 men with transmural myocardial infarction and a pulmonary artery wedge pressure over 20 mm Hg within 18 h of admission to CCU	PRCT	Pulmonary artery wedge pressure	Frusemide and ISDN reduced PAWP more than hydralazine and prenalterol	Small numbers
	Frusemide (12) *v* isosorbide dinitrate (ISDN) (12) *v* hydralazine (12) *v* prenalterol (12)		Cardiac index	Hydralazine and prenalterol increase cardiac index by increasing heart rate	
Cotter G *et al*, 1998, Israel	110 patients with acute severe pulmonary oedema. All treated with oxygen at 10 l/min and frusemide 40 mg. Isosorbide dinitrate 3 mg every 5 min (56) *v* frusemide 80 mg every 15 min and isosorbide dinitrate 1 mg/h (54).	PRCT	Need for mechanical ventilation	7/52 *v* 21/52 (p=0.0041)	Important group of patients were excluded
Beltrame JF *et al*, 1998, Australia	59 consecutive patients with acute pulmonary oedema.	PRCT	Change in Pao_2 and FIo_2 over the first 60 minutes	No significant difference	Small numbers
	iv morphine / frusemide (32) *v* iv nitroglycerin / N-acetylcysteine (37)		Need for mechanical ventilatory assistance	No significant difference	

4 Beltrame JF, Zeitz CJ, Unger SA, *et al*. Nitrate therapy is an alternative to furosemide/morphine therapy in the management of acute cardiogenic pulmonary edema. *Journal of Cardiac Failure* 1998;**4**:271–9.

Bypass or external rewarming after hypothermic cardiac arrest

Report by Claudia Webster-Smith, *Medical Student*
Search checked by Angaj Ghosh, *Senior Clinical Fellow*

Clinical scenario

A 24 year old woman is brought into the emergency department having fallen into a frozen lake. Passers by heard her cries for help and alerted the emergency services who rescued her 15 minutes later. On the way to hospital she suffered a cardiac arrest. Her core temperature on arrival is 25 degrees centigrade. You know that she needs rewarming but wonder whether her eventual outcome will be improved by cardiopulmonary bypass rather than external rewarming.

Three part question

In [severely hypothermic patients who have suffered cardiac arrest] is [core rewarming by cardiopulmonary bypass better than external rewarming] at [re-establishing spontaneous circulation and leading to eventual discharge]?

Search strategy

Medline 1966–09/00 using the OVID interface. ([exp heart arrest OR cardiac arrest.mp] AND [exp hypothermia OR hypothermia.mp OR hypothermic.mp OR exp body temperature] AND [exp heat OR exp heating OR exp rewarming OR warming.mp OR rewarming.mp]) LIMIT to human AND english.

Search outcome

Altogether 114 papers found of which 111 were irrelevant or of insufficient quality. The remaining three papers are shown in table 2.

Comments

None of the studies directly answer the question. It seems that there is a significant functional recovery after severe hypothermic cardiac arrest, and cardiopulmonary bypass seems to be an efficacious treatment. The number of patients treated by external rewarming is very small and more work will be needed before this can be recommended in preference to bypass.

Table 2

Author, date and country	*Patient group*	*Study type (level of evidence)*	*Outcomes*	*Key results*	*Study weaknesses*
Vretnar DF *et al*, 1994, Canada	68 hypothermic patients with a mean core temperature of 21°C of whom 61 were in cardiac arrest.	Review	Overall survival	60%	Publication bias likely as success more likely to be reported than failure.
			Survival if core temperature <15 degrees	0%	
	All patients placed on cardiopulmonary bypass		Return to previous function	60% of survivors	
	Data derived from 34 reports.				
Koller R *et al*, 1997, Switzerland	5 patients with core temperature below 30°C of whom 2 were in cardiac arrest	Cohort	Overall survival	100%	Small numbers.
			Return to previous function	100%	
Walpoth BH *et al*, 1997	32 of 46 patients in cardiac arrest with core temperature below 28°C	Prospective cohort	Overall survival	15/32 (45%)	Unclear why patients selected for bypass
	All patients placed on cardiopulmonary bypass				

Clinical bottom line
In severely hypothermic patients in cardiac arrest cardiopulmonary bypass should be considered.

1 Vretenar DF, Urschel JD, Parrot JC, *et al*. Cardiopulmonary bypass resuscitation for accidental hypothermia. *Ann Thorac Surg* 1994;**58**:895–8.
2 Koller R, Schnider TW, Neidhart P. Deep accidental hypothermia and cardiac arrest-rewarming with forced air. *Acta Anaesth Scand* 1997;**41**:1359–64.
3 Walpoth BH, Walpoth-Alsan BN, Mattle HP, *et al*. Outcome of survivors of accidental deep hypothermia and circulatory arrest treated with extracorporeal blood warming. *N Engl J Med* 1997;**337**:1500–5.

Using intravenous adenosine in asthmatics

Report by Polly Terry, *Specialist Registrar*
Search checked by Gail Lumsden, *Specialist Registrar*

Clinical scenario
A 32 year old woman with asthma presents to the emergency department with a 20 minute history of palpitations. On examination she is cardiovascularly stable, there is no bronchospasm and the ECG shows a supraventricular tachycardia (SVT) that fails to respond to vagal manoeuvres. You would like to use intravenous adenosine but you are aware that asthma is a contraindication treatment. You wonder what evidence there is that intravenous adenosine will cause bronchospasm.

Three part question
[In an asthmatic patient with a SVT] is [treatment with adenosine] associated with [an increased risk of bronchospasm]?

Search strategy
Medline 1966–09/00 using the OVID interface. ([exp tachycardia OR exp tachycardia, supraventricular OR narrow complex tachycardia.mp OR exp arrythmia OR exp tachycardia OR dysrhythmia.mp] AND [exp asthma OR asthma.mp OR exp respiratory sounds OR wheezing.mp OR exp bronchial spasm OR bronchospasm.mp] AND (exp adenosine OR adenosine.mp OR adenosine.ae.ct]) LIMIT to human AND english.

Search outcome
Altogether 16 papers were found of which 14 were irrelevant or of insufficient quality for inclusion. The remaining two papers are shown in table 3.

Comments
There is very little evidence recording the effect of intravenous adenosine on asthmatic airways. Many studies have documented that inhaled adenosine is a potent bronchoconstrictor in the asthmatic but not normal patients. In the literature there are four case reports of patients with asthma or COAD developing bronchospasm following treatment with intravenous adenosine. This level of evidence has many limitations. Similarly there are many studies looking at the efficacy of adenosine that report no "significant side effects" some specifically mention no patients reported bronchospasm.

Clinical bottom line
At worst adenosine is only relatively contraindicated in the treatment asthmatic patients with supraventricular tachycardia.

1 Larsson K, Sollevi A. Influence of infused adenosine on bronchial tone and bronchial reactivity in asthma. *Chest* 1988;**93**:280–4.
2 Losek JD, Endrom E, Dietrich A, *et al*. Adenosine and pediatric supraventricular tachycardia in the emergency department: multicenter study and review. *Ann Emerg Med* 1999;**33**:185–91.

Table 3

Author, date and country	*Patient group*	*Study type (level of evidence)*	*Outcomes*	*Key results*	*Study weaknesses*
Larsson K and Sollevi A, 1988, Sweden	5 well subjects with a previous diagnosis of asthma. Increasing doses of adenosine *v* placebo	Crossover placebo trial	HR BP Pulmonary function	No significant difference No significant difference No significant difference	Children only Retrospective chart review
Losek JD *et al*, 1999, USA	82 patients aged 18 yrs or less who received iv adenosine in the ED for the treatment of SVT. 13 had documented evidence of asthma	Survey	Successful cardioversion Adverse effects	72% cardioversion success rate 22 adverse patient events, no bronchospasm but 2 complaints of "dyspnoea" in non-asthmatic patients	Dose of adenosine very different to that used clinically Subjects well, and in normal sinus rhythm

First ECG in chest pain

Report by Doug Speake, *Medical Student*
Search checked by Polly Terry, *Specialist Registrar*

Clinical scenario
A 55 year old man with cardiac sounding chest pain presents to the emergency department. The first ECG is normal. Just before you

Table 4

Author, date and country	*Patient group*	*Study type (level of evidence)*	*Outcomes*	*Key results*	*Study weaknesses*
McGuinness JB *et al*, 1976, Scotland	898 patients admitted to CCU. 400 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	51%	
Starck M and Vacek JL, 1987, USA	221 ED chest pain patients. 39 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	62%	Air Force hospital, possible selection bias. No raw cardiac enzyme data confirming how AMI diagnosed.
Sharkey SW *et al*, 1988, USA	34 patients admitted to CCU. 34 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	61%	CCU population not ED. Small population size.
Fesmire F *et al*, 1989, USA	440 ED chest pain patients. 100 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	47%	No evidence of timing of the initial ECG.
Rouan G *et al*, 1989, USA	918 ED chest pain patients. 811 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	13%	Interrator agreement of ECG interpretation not measured from separate participating ED. Inclusion of AMI and ischaemic ECG changes.
Gibler B *et al*, 1992, USA	616 ED chest pain patients. 108 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	36%	Recruitment criteria unclear. Unclear if series or selection of patients recruited
Young P and Green T, 1993, USA	222 ED chest pain patients. 43 with AMI.	Retrospective survey	Sensitivity of initial ECG	28%	Retrospective study. Study population mostly elderly
Zalenski R *et al*, 1993, USA	149 ED chest pain patients. 34 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	47.1%	Small population
Fesmire F, 1998, USA	1000 ED chest pain patients. 204 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	55.4%	? 34 patients had AMI following admission to ED.
Kudenchuk PJ *et al*, 1998, USA	3027 ED chest pain patients. 1149 with AMI.	Prospective diagnostic cohort	Sensitivity of initial ECG	69%	Initial ECG taken by paramedics, not in ED.

discharge him you stop to wonder what the sensitivity of the initial 12 lead ECG is in predicting acute myocardial infarction.

Three part question
In [patients presenting to the ED with cardiac-sounding chest pain] what is the [sensitivity] of the [initial 12 lead ECG]?

Search strategy
Medline 1966–09/00 using the OVID interface. [(exp myocardial infarction OR myocardial infarction.mp OR AMI.mp OR MI.mp) AND (exp electrocardiography OR electrocardiogram.mp OR ECG.mp OR EKG.mp) AND (initial.mp OR first.mp OR single.mp or premier.mp)] AND maximally sensitive diagnostic study filter LIMIT to human AND english.

Search outcome
Altogether 543 papers found of which 533 were irrelevant or of insufficient quality for inclusion. The remaining 10 papers are shown in table 4.

Comments
At presentation history, clinical findings and ECG are all that are available to aid clinicians in the diagnosis of AMI. These studies have shown that the first ECG is between 13–69% sensitive for AMI.

Clinical bottom line
The first ECG is not sensitive enough to rule out AMI in the emergency department.

1 McGuinness JB, Begg TB, Semple T, *et al*. First electrocardiogram in recent myocardial infarction. *BMJ* 1976;2:449–51.
2 Starck M, Vacek JL. The initial electrocardiogram during admission for myocardial infarction. *Arch Intern Med* 1987;**147**:843–6.
3 Sharkey S, Apple F, Elsperger J, *et al*. Early peak of creatinine kinase in acute myocardial infarction with a non-diagnostic electrocardiogram. *Am Heart J* 1988;**116**:1207–11.
4 Fesmire F, Percy R, Wears R, *et al*. Initial ECG in Q wave and non-Q wave myocardial infarction. *Ann Emerg Med* 1989;**18**:741–6.
5 Rouan G, Lee T, Cook F, *et al*. Clinical characteristics and outcome of acute myocardial infarction in patients with initially normal or non-specific electrocardiograms. *Am J Cardiol* 1989;**64**:1087–91.
6 Gibler WB, Young G, Hedges J, *et al*. Acute myocardial infarction in chest pain patients with non-diagnostic ECGs: serial CK-MB sampling in the emergency department. *Ann Emerg Med* 1992;**21**:504–12.
7 Young P, Green T. The role of single ECG, creatinine kinase, and CKMB in diagnosing patients with acute chest pain. *Am J Emerg Med* 1993;**11**:444–9.
8 Zalenski R, Cooke D, Rydman R, *et al*. Assessing the diagnostic value of an ECG containing leads V4r, V8 and V9: the 15 lead ECG. *Ann Emerg Med* 1993;**22**:786–93.
9 Fesmire F. Usefulness of automated serial 12-lead ECG monitoring during the initial emergency department evaluation of patients with chest pain. *Ann Emerg Med* 1998;**31**:3–11.
10 Kudenchuk P, Maynard C, Cobb L, *et al*. Utility of the prehospital electrocardiogram in diagnosing acute coronary syndromes: the myocardial infarction triage and intervention (MITI) Project. *J Am Coll Cardiol* 1998;**32**:17–27.

Timing of aspirin administration in acute myocardial infarction

Report by Polly Terry, *Specialist Registrar*
Search checked by Mark Davies, *Senior Clinical Fellow*

Clinical scenario
A 49 year old man presents to the emergency department with a three hour history of central crushing chest pain. An ECG reveals an acute inferior myocardial infarction. You know that the administration of aspirin reduces future morbidity and mortality but wonder if the administration of aspirin is as time critical as thrombolysis.

Three part question
In [adults with an acute myocardial infarction] does [early administration of aspirin] decrease [mortality]?

Search strategy
Medline 1966–09/00 using the OVID interface. [(exp myocardial infarction OR myocardial infarction.mp OR heart attack.mp) AND

Table 5

Author, date and country	Patient group	Study type (level of evidence)	Outcomes	Key results	Study weaknesses
ISIS-2, 1988, multinational	17 187 patients within 24 hours of suspected MI. iv streptokinase or aspirin or both or neither	PRCT	Overall vascular mortality Odds of death at 5 weeks *v* placebo	4% relative risk reduction (0–4 h *v* 5–12 h *v* 13–24 h, p=NS)	Not the primary aim of the study, so very hard to extract data.
	Subgroup analysis mortality *v* time of aspirin administration from onset of symptoms at 0–4 h, 5–12 h, 5–24 h		0–4 h 5–12 h 13–24 h	0.75 (SD=0.07) 0.79 (SD=0.07) 0.79 (SD=0.12)	

(exp aspirin OR aspirin.mp OR salicylic acid.mp)]AND maximally sensitive RCT filter LIMIT to human AND english.

Search outcome
Altogether 295 papers found of which 294 were either irrelevant or of insufficient quality. The remaining paper is shown in table 5.

Comments
While this paper does not reach statistical significance it does show a trend in reduction of mortality with early aspirin administration. Taking this into account and the large standard deviations given a true difference may indeed exist. Other available data look at the combined effect of early thrombolysis and aspirin on the reduction of mortality and the data here are clear that the earlier the administration the greater the reduction in mortality and morbidity. Evidence exists to show that pharmacologically aspirin has maximal effect within one hour of oral administration, although whether this translates into the clinical setting is unclear. The availability of aspirin, its cost, ease of administration, and the minimal risks associated with a single dose make it an ideal immediate treatment to be given prehospital admission. The available data however suggest that this is not so time critical, that other factors cannot be taken into consideration, gor example, gastrointestinal upset, respiratory contraindications, etc.

Clinical bottom line
In an acute myocardial infarction, aspirin should be given as early as possible.

1 ISIS Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17 187 cases of acute myocardial infarction: ISIS-2. *Lancet* 1988;ii: 349–60.

CPAP in acute left ventricular failure

Report by Rupert Jackson, *Specialist Registrar*
Search checked by Simon Carley, *Specialist Registrar*

Clinical scenario
A 76 year old man is brought into accident and emergency in a collapsed state. He has a history of ischaemic heart disease. He is agitated, tachypnoeic and sweating profusely. His neck veins are distended and there are widespread coarse crepitations in his chest. He has a diminished oxygen saturation. You make a clinical diagnosis of acute cardiogenic pulmonary oedema. In addition to vasodilator treatment and opioids, you wonder whether you should administer non-invasive continuous positive airways pressure (CPAP).

Three part question
[In patients with acute LVF] is [CPAP better than O_2 via normal mask] at [avoiding intubation and improving mortality]?

Search strategy
Medline 1966–09/00 using the OVID interface. ([exp pulmonary edema OR pulmonary oedema.mp OR exp ventricular dysfunction, left OR exp heart failure, congestive OR exp myocardial infarction OR left ventricular failure.mp OR LVF.mp) AND (exp positive-pressure respiration OR CPAP.mp OR continuous positive airway pressure$.mp OR PEEP.mp OR positive end expiratory

Table 6

Author, date and country	Patient group	Study type (level of evidence)	Outcomes	Key results	Study weaknesses
Rasanen J *et al*, 1985, Finland	40 patients with acute cardiogenic pulmonary oedema. RR >25 and Pao_2 <200 mm Hg	PRCT	Need for intubation	6/20 *v* 12/20 (NS)	Small numbers. Unblinded
	CPAP (20) *v* control (20)		Hospital mortality	17/20 *v* 14/20 deaths in hospital (NS)	
Bersten A *et al*, 1991, Australia	39 patients with acute cardiogenic pulmonary oedema. Pao_2 <70 mm Hg and $Paco_2$ >45 mm Hg	PRCT	Need for intubation	0/19 *v* 7/20 (p<0.005)	Small numbers. Unblinded. Randomisation not concealed.
	CPAP (19) *v* control (20)		Hospital mortality	2/19 *v* 4/20 (NS)	
Lin M and Chiang HT, 1991, Taiwan	55 patients with acute cardiogenic pulmonary oedema. RR >20	PRCT	Need for intubation Hospital mortality Shunt size	7/25 *v* 17/30 (p<0.05) 2/25 *v* 4/30 (NS) Significantly improved in CPAP group	
	CPAP (25) *v* control (30)		Pao_2	Significantly improved in CPAP group	
Lin M *et al*, 1991, Taiwan	100 patients with a clinical diagnosis of acute cardiogenic pulmonary oedema	PRCT	Need for intubation	8/50 *v* 18/50 (p<0.01)	Unblinded
	CPAP (50) *v* control (50)		Hospital mortality	4/50 *v* 6/50 (NS)	
Takeda S *et al*, 1998, Japan	22 patients with acute cardiogenic pulmonary oedema. Pao_2 <80 mm Hg.	PRCT	Need for intubation	2/11 *v* 8/11 (p=0.03)	Small numbers. Unblinded
	CPAP (11) *v* control (11)		Hospital mortality	1/11 *v* 7/11 (p=0.02)	

pressure$.mp] AND maximally sensitive randomised controlled trial filter) LIMIT to human AND english language

Search outcome

Altogether 114 papers were found of which 109 were either irrelevant or of insufficient quality for inclusion. The remaining five papers are shown in table 6.

Comments

All of these trials have shown significant reductions in the need to intubate patients in acute pulmonary oedema. In these small trials a reduction in mortality could not be seen. The numbers in the trials are not large and there is not yet absolute evidence of benefit from CPAP. A large, well designed PRCT may provide this. In the meantime it would seem that patients with severe LVF will benefit from CPAP.

Clinical bottom line

Patients presenting with severe acute pulmonary oedema should be treated with CPAP.

1 Rasanen J, Heikkila J, Downs J, *et al*. Continuous positive airway pressure by facemask in acute cardiogenic pulmonary edema *Am J Cardiol* 1985;**55**:296–300.
2 Bersten AD, Holt AW, Vedig AE, *et al*. Treatment of severe cardiogenic pulmonary edema with continuous positive airway pressure delivered by face mask. *N Engl J Med* 1991; **325**:1825–30.
3 Lin M, Chiang HT. The efficacy of early continuous positive airway pressure therapy in patients with acute cardiogenic pulmonary edema. *J Formosan Med Assoc* 1991;**90**:736–43.
4 Lin M, Yang YF, Chiang HT, *et al* Reappraisal of continuous positive airway pressure therapy in acute cardiogenic pulmonary edema. Short-term results and long-term follow-up. *Chest* 1995;**107**:1379–86.
5 Takeda S, Nejima J, Takano T, *et al*. Effect of nasal continuous positive airway pressure on pulmonary edema complicating acute myocardial infarction. *Jpn Circ J* 1998;**62**:553–8.