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Out of hours primary care

Variable service provision means inequalities in access and care

See pp 182, 187, 190, 193, 198, 199

The six papers on out of hours care published in this week's *BMJ* highlight the increasing variability in primary care services available to patients outside normal surgery hours. Variations in the quality and acceptability of care provided by deputising services and general practitioner rotas have long caused concern,[1] but a third provider group has now entered the arena. General practitioner cooperatives have mushroomed, fuelled by general practitioners' dissatisfaction with rota commitments and financial support from the government.[2 3] Unlike commercial services,[4-6] cooperatives do not face external controls and, as Jessopp and colleagues point out (p 199),[7] they vary widely in their composition and patterns of service delivery.

Giving telephone advice alone is increasingly common. Cragg and colleagues' data from 1994-5 (p 187) show that less than 1% of callers to four deputising services and 20% of callers to general practitioner rotas received telephone advice,[8] which contrasts sharply with Salisbury's data for 1996, with rates of 19% for deputising service contacts and 58% for a general practitioner cooperative (p 182).[9] While it would be unwise to place too much reliance on figures from one metropolitan cooperative, Jessopp and colleagues report rates of telephone advice between 10% and 65% across 67 cooperatives, with a median of 38%.[7]

Is this a cause for concern? It is clear that clinical criteria alone do not govern the nature of the response to patients' calls and that widely differing standards of access to a doctor now obtain. In 1987, when Marsh and colleagues reported handling 59% of out of hours calls to their own practice rota by telephone advice alone,[10] this was considered sufficiently alarming to generate columns of correspondence in subsequent issues of the *BMJ*, questioning the safety and standard of care provided. In a 1992 survey of telephone use in general practice, a substantial proportion of general practitioners expressed personal disquiet with this form of care, particularly when they did not know the patient.[11] Yet telephone advice is now being offered increasingly often by deputising services and by large groups of general practitioners in cooperatives with consequently less personal knowledge of the patients they are advising and the communities in which they are working. Previously expressed concerns seem to have evaporated, and the potential role of practice nurses in providing telephone triage is now being studied (p 198).[12]

A new and reliable measure of patient satisfaction with out of hours care, developed by McKinley and colleagues (p 193),[13] has shown higher levels of dissatisfaction than ever previously reported (p 190).[14] The authors are right to point out that direct comparisons between these findings and those of earlier studies using less well designed and tested measures are not possible, but the levels are striking in themselves, and once again deputising services score less well than general practitioner rotas. Patient satisfaction has been shown in the past to be related to speed of response,[15 16] and this remains the aspect of care with which patients are least satisfied. Yet response times are slowing generally and seem to be poorest in the cooperative studied by Salisbury.[9] Cragg *et al*'s median response times of 35 minutes for rota general practitioners and 52 minutes for deputising doctors[8] contrast with Salisbury's 65 minutes for deputising doctors and 75 minutes for the cooperative.[9]

More research is needed on the ability of cooperatives to respond rapidly in cases of urgent need. Many rural cooperatives cover large geographical areas. Outside the periods of peak demand a single general practitioner may be responsible for providing telephone advice, centre based consultations, and home visits. In some cases, call handling services introduce an added delay between patients' calls and a general practitioner's response. This issue does not simply relate to patient satisfaction but also to patient safety.

In 1992 a stage had been reached at which the demands and expectations of patients for out of hours care had outstripped general practitioners' willingness and ability to meet them. Increased reliance on deputising services and the growth of the cooperatives has averted an immediate crisis. However, out of hours services are now more variable, and it is by no means clear to what extent that variability represents inequality in access, quality of care, and hence satisfaction with services. McKinley and colleagues' measure of patient satisfaction will be a valuable tool in assessing the impact of new methods of organising and delivering services on patient satisfaction.[12]

We have not yet accumulated a sufficient body of evidence to judge the quality of services offered by cooperatives. As Jessopp and colleagues show,[7] their enthusiasm for centre based care (5-70% of contacts) in preference to home visiting is as variable as their reliance on telephone advice and equally unevaluated. The benefits they offer their general practitioner members have been widely quoted,[17] but they have spread

without reference to patients' views, with no attempt to involve users in their planning and operation, and with limited efforts to assess patient satisfaction. Without evidence to support a need for uniform standards of service and care, and without a clear idea of what those standards should be, they are likely to maintain their independence of action, as practice rotas have always done.

Uniformity is not necessarily a virtue in circumstances where needs and demands for care differ. The importance of flexibility in order to address local needs and circumstances, particularly where services are poor, lies at the heart of the recent government white paper on primary care.[18] However, equality of access to uniformly high standards of care is an important goal for primary health care, and increasing variability in the organisation and delivery of out of hours services should not lead to increasing inequality.

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Epilepsy: getting the diagnosis right

All that convulses is not epilepsy

See p 171

Epilepsy may present with a variety of symptoms, and other conditions may mimic its manifestations. The diagnosis is almost always based solely on the clinical history. It is therefore not surprising that diagnostic accuracy remains a major problem.[1] About a fifth of patients referred to specialist units with "intractable epilepsy" are found, on further assessment, not to have epilepsy.[2] It is also common for patients to have symptoms for months or even years before epilepsy is diagnosed. Thus, it is important to be aware of both the heterogeneous and sometimes subtle forms of epilepsy and of the alternative diagnoses.[3]

The differential diagnosis of epilepsy includes all causes of transient loss of awareness, falls, paroxysmal sensory-motor phenomena, and generalised convulsive movements which are the most common presenting symptom of epilepsy.[3] Tonic-clonic seizures ("a convulsion") start with sudden loss of awareness, a guttural cry, generalised stiffening of the body and limbs, followed by rhythmic jerking of the limbs, often associated with tongue biting and urinary incontinence. The convulsive movements usually last for at most one to two minutes, and, as the attack proceeds, the jerking slows in frequency and increases in amplitude. There is often cyanosis and irregular breathing followed by confusion, headache, and drowsiness. Tonic-clonic seizures may sometimes be preceded by myoclonic jerks in idiopathic generalized epilepsy or by a simple partial seizure ("aura") in partial epilepsy. When all or most of these features are reported there is little room for diagnostic confusion.[3]

However, other conditions may present with similar phenomena. If misinterpreted, these can lead to unnecessary treatment and social and occupational handicap. The most common sources of confusion are vasovagal syncope and non-epileptic attacks of a psychological origin. Syncope is often misdiagnosed, as it may be accompanied by brief stiffening or jerking of the extremities, and consequently is liable to be reported as a convulsion by witnesses. A video study of syncope induced in healthy volunteers has shown that multifocal and generalised myoclonic jerks are common in syncope.[4] However, syncope can usually be correctly identified by the presence of precipitating factors and prodromal symptoms. Syncope often occurs on prolonged standing or when rising quickly, particularly if associated with peripheral vasodilatation. Syncope is unusual when recumbent, unless it is of cardiac origin. Fright, painful stimuli, cough, and micturition (particularly in older people) may also be triggers. Syncopal attacks are preceded by a feeling of lightheadedness, dizziness, nausea, ringing in the ears, and the vision "going grey"—features that are rare in epilepsy. Incontinence is rare, and recovery of consciousness usually occurs within a minute.[3]

Non-epileptic attack disorder may be characterised by semi-purposeful thrashing of all four limbs that waxes and wanes in intensity over many minutes, and

some patients exhibit prominent pelvic movements and back arching, often with evidence of retained awareness.[2] Recovery is variable and may be much quicker than expected from the duration of the attack.[3]

In this week's *BMJ*, McCrory and colleagues describe what they call "concussive convulsion," (p 171) another potential pitfall in the diagnosis of epilepsy.[5] Convulsions that occur within seconds of an impact to the head have been widely assumed to represent a form of post-traumatic epileptic seizure, but McCrory and colleagues suggest that these are a non-epileptic phenomenon. Studying a series of 22 well documented attacks, some captured on video, that occurred after minor head trauma during Australian football, the authors were able to ascertain the benign nature of these attacks. The convulsions occurred within two seconds of the impact and resembled tonic-clonic seizures. The convulsions were usually brief, but some lasted for over two minutes. Recovery was quick; indeed, in two cases the players were alert and oriented within seconds of the convulsive event. Of particular importance is that, after a mean follow up of 3.5 years, no subjects developed epilepsy. This corroborates Jennett's observation over 20 years ago that seizures confined to the time of the head injury are not associated with subsequent epilepsy.[6]

The clinical characteristics of concussive convulsions seem to differ somewhat from those after syncope. There is a more prominent tonic phase, and the attack lasts longer. Electroencephalography shows that syncopal convulsions are non-epileptic, but it remains to be established whether these events arise as a result of an epileptic discharge. McCrory *et al* argue that they do not. Alternatively, we suggest that they represent an acute symptomatic but benign seizure. Pathophysiology notwithstanding, the observations provide helpful prognostic information. Concussive convulsions should be distinguished from seizures that occur within the first week of head injury rather than instantly after impact. These carry a 25% risk of later epilepsy.[6]

Every effort should be made to reach a firm diagnosis in cases of possible epilepsy. If doubt remains after the first event it is usually wise to await further events and reach a secure diagnosis, rather than initiate anti-epileptic treatment prematurely.

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Lack of oats toxicity in coeliac disease

Toxic fraction makes up less of total protein than in other cereals

In his pioneering study of "the harmful effects of certain types of cereal on patients suffering from coeliac disease," Dicke showed that wheat and rye could reproducibly trigger anorexia, diarrhoea, and steatorrhoea in these patients.[1] Soon after, using the same prolonged fecal balance studies, Dicke found that oats were also noxious whereas corn, rice, and potatoes were not.[2 3] Reports suggesting that barley was toxic came later.[4 5] Simultaneously, the "injurious constituent of wheat" was found to be its prolamin (or alcohol soluble protein), gliadin.[3] Secalin, hordein, and avenin, the prolamins of rye, barley, and oats respectively, were thus considered as the toxic fractions of these cereals.

However, whereas the noxious effects of wheat, barley, and rye could be reproduced, the harmful effects of oats remained controversial—observed by some workers,[2 3] denied by others,[6 7] and variable for still others.[5 8] This uncertainty stems from several factors. Firstly, the early studies included small numbers of patients (from two to 12) and followed them for short periods (from several weeks to less than three months). Secondly, the methods used to determine the harmful effects varied from insensitive functional tests—balance studies[2 3 8] and xylose tests[5] —to histological[4] and biochemical studies[7] of small intestinal biopsies that were sometimes difficult to interpret. These methodological limitations are relevant when considering the variability of gluten sensitivity from patient to patient; several years of a gluten containing diet are sometimes necessary before a patient will relapse.[9] However, despite the variability of the clinical and histological responses observed after these early challenges with oats, it seems clear that taking small amounts of oats (about 50 g) for short periods (less than one month) is not generally noxious whereas more than 100 g for longer than a month leads to recurrence of steatorrhoea, a strong sign of serious mucosal damage.[2 3 8]

A recent Finnish study avoided the pitfalls of variability by following a large number of patients (92) for one year and using stringent histological criteria of mucosal damage. Patients recently diagnosed or in remission were given a gluten free diet and randomised to receive either no oats or 50 g of oats a day. Severe cases were excluded. After one year, the two groups showed no significant difference in clinical symptoms, laboratory measures, or histological criteria. All the newly diagnosed patients were in clinical and histological remission. The conclusion is straight-

forward: moderate amounts of oats (40-60 g/day) are not toxic in most patients with coeliac disease.[10]

The recent study by Srinivasan *et al* points in the same direction.[11] Ten patients, including two who were particularly sensitive to gluten, consumed 50 g of oats as porridge daily for three months while maintaining a strict gluten free diet. During the challenge the patients remained symptom free and maintained low titres of antiendomysium and antiangliadin antibodies. Quantitative histological evaluation at the end of the observation period showed no change in mucosal appearance, in particular no increase in intraepithelial lymphocyte count. Thus, as in the Finnish study, although with a smaller number of patients and shorter duration of challenge, moderate amounts of oats proved non-toxic. These findings have recently been confirmed in patients with dermatitis herpetiformis,[12] and in the case of the Finnish study, by a five year follow up.[13]

Although concordant, these studies do not show that larger daily amounts of oats (100-160 g) would be equally non-toxic in these patients. Indeed, as suggested by the early experiments,[2 3 8] large amounts of oats are theoretically likely to be toxic. The oat is a member of the Avena tribe, whereas wheat, rye, and barley are members of the neighbouring Triticeae tribe, both tribes being part of the Pooideae subfamily. Thus, avenin, the prolamin of oats, is genetically less like gliadin than are secalin and hordein. Despite this greater difference, sequence homologies (and weak immunological cross reactivity) have been found between avenin and the prolamins from barley, wheat, and rye.[14-16] Moreover, avenin accounts for only 5-15% of the total protein in oats compared with the 40% contribution from gliadin in wheat and the prolamins in rye and barley.[14] Thus, taking into account a smaller number of toxic sequences per unit weight of avenin and the smaller amounts of avenin as a proportion of the oat seed proteins, it seems likely that only considerable amounts of oats consumed over long periods will be toxic for patients with coeliac disease.

However, until randomised studies are performed on large enough numbers of patients consuming large amounts of oats for long periods, it seems reasonable to assume that moderate amounts of oats may be consumed by most patients without risk.[10 11] As noted by Watson,[17] in Scotland "it would have been obvious many years ago if coeliac children and adults who are taking porridge relapsed."

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Hysterectomy: will it pay the bills in 2007?

Treatment of choice for cancer, but a choice of treatment for menorrhagia

To study the indications for hysterectomy is to study the interface between medicine and society. In California barely half of all women will carry their uterus to the grave,[1] whereas a gynaecologist in Saudi Arabia may do no more than one hysterectomy a year and, as often as not, this will be a lifesaving operation for catastrophic obstetric haemorrhage. In Britain hysterectomy rates are somewhere between these two extremes. To understand the variations, do not gaze endlessly at histological specimens but examine the societies from which they originate.

Perceived abnormal bleeding acounts for 70% of hysterectomies in pre-menopausal British women, and in most cases of "menorrhagia," menstrual blood loss is within the "normal" range.[2] Much of the variation in hysterectomy rates is therefore attributable to the psychosocial factors that influence demand.[3]

Provider factors are also important. Women general practitioners are less likely than their male counterparts to refer women with menstrual symptoms for a specialist opinion;[4] and hysterectomy, like cholecystectomy and tonsillectomy, varies considerably in frequency from surgeon to surgeon.[5] This is not to

say that gynaecologists exploit women for personal gain or take some sort of covert delight in the procedure; doctors' wives, after all, undergo hysterectomy as often as controls matched for social class.[6] Yet hysterectomy rates are not only variable but labile: a public education campaign in the Italian speaking cantons of Switzerland resulted in a sharp fall in hysterectomy rates compared with the control cantons (French and German speaking).[7]

It is tempting to conclude that a lot of unnecessary surgery is going on and that we should campaign against it. But what are the effects of hysterectomy? Although in the short term women who have had a hysterectomy score worse than non-surgical controls on measures of wellbeing, if the same women are followed prospectively their scores improve.[8] An authoritative study failed to confirm fears that hysterectomy increases the risk of urinary incontinence.[9] And while hysterectomy is a major operation with serious morbidity and, very rarely, mortality,[10] it may also save lives by reducing the risk of uterine cancer. The relative risks of operative mortality and death from cancer are such that hysterectomy is the safer option.[11] Risk of cancer is reduced still further by removing the ovaries, although this increases the risk of ischaemic heart disease unless hormone replacement therapy is taken. However, oestrogen replacement does not correct the loss of libido that follows oophorectomy, perhaps as a result of androgen deficiency.[8] Preliminary evidence that hysterectomy may predispose to ovarian failure, even when the ovaries are conserved, is a subject of current research.

How should hysterectomy be performed? Vaginal hysterectomy is associated with fewer complications than abdominal hysterectomy.[12] However, this has not been confirmed by a recent systematic review,[13] and many surgeons feel uncomfortable with the vaginal route in the absence of prolapse. Laparoscopically assisted vaginal hysterectomy is a new technique which is currently under evaluation in a large trial and a nationwide observational study, both funded through the NHS R and D programme.

Should the cervix be removed during abdominal or laparoscopic hysterectomy? Surgical morbidity is often the result of removal of the cervix, which lies close to the ureters and bladder and from which it must be carefully dissected with a consequent risk of vault haematoma or urological injury.[14] This, along with a suspicion that the presence of the cervix may enhance orgasm, has led to calls for "sub-total" hysterectomy. Removal of the cervix was strongly advocated in the first half of the century, in part because of the risk of cancer. However, the residual risk of this disease in women with a history of regular negative cervical smears is sufficiently low to be traded off against lower complication rates.[14]

What about other surgical alternatives? Various techniques to remove the endometrium while leaving the remainder of the uterus in situ have been investigated.[15] Although these procedures result in lower morbidity and shorter hospital stay than hysterectomy, up to 30% of patients will eventually lose their uterus. Endometrial surgery provides additional choice, but overall rates of surgical treatment have not declined and may have risen.[16]

About one woman in seven will decline hysterectomy if she can be shown to have blood loss within the normal range.[17] For these and other women, medical treatments are appropriate. However, referral is almost always followed by surgery,[18] and medical treatments may merely delay a surgical "solution." The new progesterone-coated intrauterine device proposes to revolutionise non-surgical management. Recently licensed in this country, the levonorgestrel-bearing device substantially reduces menstrual flow (and seems to be much more effective than oral medical treatment) while also providing effective contraception and reducing the risk of sexually transmitted diseases.[19] Whether this will remove hysterectomy from its pre-eminent place in the repertoire of gynaecological treatments remains to be seen. Although the first hysterectomy was carried out in 1822, [20] it has become a mainstay of gynaecological practice. It is quite probable that the operation has "peaked" and will now decline in incidence. There is no "correct" hysterectomy rate, but "correct" practice is to make explicit the trade offs between this operation and an increasing number of alternatives.

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Drinking before sedation

Preoperative fasting should be the exception rather than the rule

Traditionally, patients are starved of food and fluid for several hours before being given a general anaesthetic. However, in the early days of anaesthesia a drink was often recommended before the procedure,[1] and a fluid fast became commonplace only after the publication of Mendelson's landmark study in 1946.[2] In it he described the risk of gastric acid aspiration during obstetric anaesthesia with the consequent development of pneumonitis. He also showed that human gastric acid injected into the airway of rabbits caused radiographic changes similar to those described after acid aspiration in pregnant women.

The findings of these studies have since been extrapolated to all forms of general anaesthesia, resulting in patients being deprived of fluid from midnight before a morning anaesthetic and allowed only a light breakfast before afternoon surgery. Furthermore, with increasing numbers of patients undergoing invasive procedures requiring intravenous sedation, the "nil by mouth from midnight" request has spread to medical wards. In particular, patients are starved before elective gastroscopy, endoscopic retrograde cholangiopancreatography, and colonoscopy. However, this prolonged fast of fluid is illogical for two reasons: firstly, in fasting patients the stomach can secrete up to 50 ml of gastric juice an hour;[3] and secondly, it has been shown that ingested clear fluids rapidly leave the stomach of healthy people, with about half the volume disappearing in 10-20 minutes.[4]

Recently, workers have questioned the benefits of a fluid fast before anaesthesia. Prolonged fluid deprivation has been shown to increase the volume and decrease the pH of gastric juice, both of which increase the likelihood and consequences of gastric acid aspiration. Sutherland *et al* used an orogastric tube to measure the volume and pH of gastric juice in 100 anaesthetised patients who were randomly assigned either to drink 150 ml of water two to three hours before their anaesthetic or to fast from midnight[5]: the volume and pH of residual gastric juice was 20.6 ml and 2.05 respectively in the patients who drank compared with 29.9 ml and 1.72 in the patients who fasted.

The same group has since repeated the study and shown that the volume and pH of residual gastric contents of 300 consecutive patients randomised to drink 150 ml of coffee or orange juice given with ranitidine three hours before anaesthesia were identical to those in patients who did not drink.[6] In that study the patients who drank suffered less thirst compared with those who did not. A further randomised controlled study by Phillips *et al* showed similar residual gastric volumes and pH in 100 patients starved conventionally (19 ml and 2.26 respectively) or allowed unrestricted intake of clear fluid until two hours before their anaesthetic (22 ml and 2.64).[7]

In response to these studies the Canadian Anaesthetists' Society produced guidelines stating that a fluid fast of more than three hours is unnecessary in healthy patients undergoing surgery.[8] Furthermore, gastroscopy has been used to visualise and aspirate all gastric juice in sedated patients undergoing gastroscopy and has validated the previous studies in which orogastric tubes were used to obtain stomach fluid. Eighty eight patients were randomised to drinking either 330 ml of water two hours before their endoscopy or an overnight fast.[9] After oesphageal intubation, all gastric juice was aspirated and the volume and pH measured and found to be virtually identical in the two groups. In addition, mucosal views were excellent in all the examinations.

Although patients who are likely to have delayed gastric emptying (through underlying disease or drug treatment) should not drink before anaesthesia or intravenous sedation, there is now overwhelming evidence in favour of allowing patients who fulfill grades I or II of the American Society of Anesthesiologists classification of physical status to drink clear fluids up to two hours before the procedure. This message should now be disseminated to all medical and nursing staff to ensure that patients do not suffer uncomfortable thirst and that their procedure is not cancelled because of an inadvertent drink.

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Non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease

Use it sooner rather than later to assist the "respiratory muscle pump"

Between a fifth and a third of patients admitted to hospital with hypercapnic respiratory failure secondary to acute exacerbation of chronic obstructive pulmonary disease will die in hospital, despite selective use of mechanical ventilation. [1-13] In severe chronic obstructive pulmonary disease, hyperinflation places the respiratory muscles at a mechanical disadvantage and they function close to their maximum capacity.[14 15] During acute exacerbations, elastic and resistive loads on the respiratory muscles increase and may lead to ventilatory failure. The ensuing tissue acidosis further impairs respiratory muscle function, producing a vicious cycle.[16] Thus a logical approach is to assist the compromised "respiratory muscle pump".

Non-invasive positive pressure ventilation (NIPPV) employs a nasal or full face mask to administer ventilatory support from a flow generator and is established in the treatment of patients with a variety of chronic hypoventilatory syndromes. A role in acute exacerbation of chronic obstructive pulmonary disease (COPD) was suggested by early open and case control studies[9-11] and has recently been confirmed by three randomised controlled trials (see table).[5-7]

Bott *et al* randomised 60 patients with exacerbations of chronic obstructive pulmonary disease to either standard treatment (antibiotics, bronchodilators, corticosteroids, respiratory stimulants, and oxygen) or standard treatment plus non-invasive ventilation on a medical ward.[5] Nine of the 30 patients receiving standard treatment died in contrast to only one of the 26 who accepted non-invasive ventilation (relative risk 0.13, 95% CI 0.02-0.95). (Of the four who did not receive ventilation, two died.) Four patients who failed to respond to standard treatment were offered non-invasive ventilation, though three required intubation.

Brochard *et al* selected 85 patients with incipient acute respiratory failure from a pool of 275 and treated 42 with standard therapy and 43 with additional non-invasive ventilation.[6] Patients were excluded if they needed immediate intubation, had heart failure, pneumonia, asthma, or sepsis, or were postoperative. In the standard treatment group, 74% were intubated and 29% died compared with 26% ($p<0.001$) and 9% ($p=0.02$) respectively in the ventilated group. Furthermore, life threatening complications were more frequent in the standard treatment group and hospital stay was longer (see table)[6]

In a well matched group of patients, Kramer *et al* found a significant reduction in the need for intubation in patients treated with non-invasive ventilation (one of 11) over those receiving standard treatment (eight of 12).[7] Mortality was lower than in the other studies and may reflect sample size, the intensive care setting, or longer duration of ventilation.

Although these studies seem conclusive, they could not be blind since "sham" ventilation is not feasible. Significant placebo effect and bias in management may have influenced the outcome. In two of the studies, investigators making clinical management decisions were unaware of which treatment arm a patient was in until ventilation was started,[5 6] and in the third the decision to intubate was not made by the investigators.[7] The lack of a clear protocol for establishing the need for intubation is therefore a weakness in two of the studies since it allows management bias, and although Brochard *et al* clearly specified criteria for intubation, they were complex.[6] In two studies significant reductions in breathlessness scores and respiratory rate were found in the ventilated groups.[5 7] However, breathlessness was measured by visual analogue scale rather than validated questionnaire. One recent controlled study has shown no beneficial effect of non-invasive ventilation given twice a day for three hours, but patients were less severely affected than in other studies since all recovered without the need for mechanical ventilation.[17]

Both the initial response to non-invasive ventilation and the severity of the ventilatory failure at presentation are important predictors of success. Ambrosino *et al*

Table 1 Randomised controlled trials of NIPPV in respiratory failure secondary to acute exacerbations of COPD

Entry criteria	Exclusion criteria (selected)	Main findings (Standard *v* NIPPV)	Comment on study
Bott *et al*[5]			
Clinical diagnosis of chronic airflow obstruction and: Pa_{O_2}<7.5 kPa Pa_{CO_2} >6 kPa Age ≤80 years	Severe non-respiratory disease, prior NIPPV	Reduced mortality (30% *v* 4%, P=0.014) Reduced breathlessness	No objective criteria for intubation Non-validated dyspnoea questionnaires
Brochard *et al*[6]			
Increased dyspnoea for<2 weeks and: Pa_{O_2}<6.0 kPa pH<7.35 Respiratory rate >30 breaths/min	Respiratory rate<12 breaths/min, need for immediate intubation, asthma, heart failure, pneumothorax, sepsis, postoperative period	Reduced mortality (29% *v* 9%, P=0.02) Reduced intubation (74% *v* 26%, P<0.001) Reduced hospital stay (35 days *v* 23 days, P=0.02) Reduced life threatening complications (48% *v* 16%, P=0.001)	Carefully selected population Detailed criteria for intubation Sealed envelopes used for treatment allocation
Kramer *et al*[7]			
Moderate to severe dyspnoea and: Pa_{CO_2} >6 kPa pH<7.35 Respiratory rate >24 breaths/min	Need for immediate intubation, inability to cooperate or fit mask, inability to clear secretions, uncontrolled arrhythmias, systolic blood pressure <90 mm Hg	Reduced intubation (73% *v* 31%, P<0.05) Reduced breathlessness Reduced respiratory rate at 1 hour	Non-validated dyspnoea questionnaires Lower mortality—may reflect intensive care setting

found that at one hour the blood pH was lower and $PaCO_2$ higher in patients in whom non-invasive ventilation would be unsuccessful. Rapid improvement in pH and respiratory rate in the first hour of non-invasive ventilation has been shown to be an important predictor of success.[5 17] Reversing the vicious cycle of ventilatory failure and acidosis is likely to be easier at an earlier stage since the level of assistance required may be less both in terms of time using the ventilator and magnitude of pressure support. Furthermore, other consequences of hypoventilation, such as mucus retention, are less likely. Thus non-invasive ventilation should be instituted at an early stage when the pH falls below 7.35 [5 6] and the respiratory rate exceeds 30 breaths per minute. If there is no improvement in these parameters in the first one to two hours, intubation should be considered.[5 18] Two studies have shown that the more severe the episode of ventilatory failure (indicated by the degree of acidosis) the smaller the chances of success.[6 8] Non-invasive ventilation cannot be recommended for patients who require immediate intubation because none of the controlled trials included such patients. The duration of ventilation will depend on need and tolerance, but in two controlled trials a mean of only six to eight hours per day was efficacious.[5 6]

Which health professionals should initiate non-invasive ventilation is not clear, but the list may include pulmonary function technicians, physiotherapists, nurses, and junior medical staff, depending on local interest and expertise. Nursing staff under medical guidance should probably play a major role, especially as a 24 hour service is required. Earlier reports that non-invasive ventilation was time consuming for nursing staff[13] have not been confirmed.[5 7]

A trial of non-invasive ventilation should be considered early in the course of acute respiratory failure secondary to exacerbation of chronic obstructive pulmonary disease as a means of avoiding intubation and reducing hospital stay and mortality.

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Don't treat shackled patients

And keep trying to understand what the Nuremberg trials taught us

Last week Britain was shocked by the report of a young man who was shackled to a bed until two hours before he died of stomach cancer. He was shackled because he was a prisoner. Last year doctors and others had to protest about women prisoners being forced to give birth while shackled.[1] Understandably doctors unused to treating prisoners in NHS hospitals are not sure about "the rules." But they should be. Doctors should simply refuse to treat patients who are shackled, and doctors' organisations should support them without quibble. This is the state making doctors participate in unethical acts in the way that was described in the *BMJ*'s December 7 issue marking the 50th anniversary of the Nuremberg doctors' trials.

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