

Promoting breast feeding in the community

Breastfeeding groups and peer counselling must be integrated into wider programmes



VLADISLAV GANSOVSKI/FOTOLIA

Breast feeding has well known advantages for mothers and children, yet its rates are lower than recommended. Two linked randomised controlled trials assess the effectiveness of different approaches for promoting breast feeding in the United Kingdom.^{1,2}

Several international guidelines exist on how to improve the rates of breast feeding. The World Health Organization stresses the importance of developing community based support networks to help ensure appropriate feeding of infants and young children—for example, mother to mother support groups and peer or lay counsellors, to which hospitals and clinics can refer mothers on discharge.³

The National Institute for Health and Clinical Excellence (NICE) recommends informal and practical education about breast feeding in the antenatal period and peer support programmes to increase rates of initiation and the duration of breast feeding in women on low incomes. It stresses the need for strategies to recruit and retain peer or volunteer supporters, including provision of formal salaries, paid incentives or honorary contract schemes, and supervision to ensure quality of the service.⁴

Unicef UK recommends that all breastfeeding mothers should be informed of the professional and voluntary support available, and that they should be given contact details of community midwives, voluntary counsellors, and any breastfeeding support groups.⁵ Interventions should be available to mothers in the local community, according to local need. Such interventions may include peer support, telephone contact, informal groups, or long term one to one support.⁶

The two linked randomised controlled trials challenge these recommendations. Hoddinott and colleagues report no improvement in breastfeeding rates six to eight weeks after the provision of breastfeeding groups in relatively deprived areas of Scotland.¹ MacArthur and colleagues found that a universal antenatal breastfeeding peer support service for a multiethnic deprived population in Birmingham did not increase the initiation of breast feeding.² Will this new evidence induce WHO, NICE, and Unicef UK to revise their recommendations?

Hoddinott and colleagues assessed a complex intervention—groups were set up in which a female health professional met weekly with four (interquartile range 2-6) pregnant and breastfeeding women. At least half of the time at group meetings was devoted to social interaction. Fewer pregnant women attended these meet-

ings than breastfeeding women (10% *v* 69%), who first attended a group 36 (28-35) days after their baby was born. The trial does not mention whether the intervention included individual support for the management of early lactation problems; so it can be assumed that this support was the same in the intervention and control groups. The study found no significant difference in breastfeeding rates at six to eight weeks. The authors conclude that resources may be better directed to the first two weeks after birth when the highest proportion of women stop breast feeding.

This recommendation is the subject of the second randomised controlled trial.² In Birmingham, 11 trained peer support workers were added to the team of a primary care trust to contact pregnant women twice, at 24-28 weeks gestation and at around 36 weeks' gestation. About 74% of women in the intervention group met with one of these workers once (mean duration of meeting 13 minutes); only 41.5% of these women had a second contact. Intrapartum and early postpartum hospital care was the same in the intervention and control groups. During the support sessions, support workers discussed with women the benefits and convenience of breast feeding, the perceived difficulties, and their partners' and families' attitudes towards breast feeding. The study found no significant difference in the initiation of breast feeding between groups (cluster adjusted odds ratio 1.11, 95% confidence interval 0.87 to 1.43; *P*=0.40).

These details indicate that rather than questioning the general recommendation about promotion and support of breast feeding through peer counsellors and breastfeeding groups before and after childbirth in primary care, we should look at how this recommendation is put into practice. Informing pregnant women about why they should breast feed does not help them fulfil their decisions; for this, they need information before they start breast feeding about how best to do it (immediate and prolonged skin to skin contact after birth, no separation, baby led frequent feeding, no supplements, good latch on).

In addition, the decision about whether to breast feed or bottle feed may be taken during pregnancy by only a minority of women.⁷ As far as support after discharge is concerned, breastfeeding groups should ideally catch mothers during the first or second week after birth, when the incidence of suboptimal breast feeding is high, and they should combine interactive social support with effective management of lactation problems.⁸

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Adriano Cattaneo epidemiologist, Health Services Research, Epidemiology and International Health, Institute for Maternal and Child Health, 34137 Trieste, Italy cattaneo@burlo.trieste.it

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Finally, because so many determinants are associated with the initiation, exclusivity, and duration of breast feeding,⁹ single interventions—even if shown to be effective by properly conducted randomised trials and systematic reviews—are unlikely to be effective in real life situations. This is especially the case in women who come from a deprived background. Effective interventions will have a higher chance of producing results if embedded in a national or local plan that is tailored to specific needs.¹⁰

The recommendations of WHO, NICE, and Unicef UK are valid, but interventions based on breastfeeding groups and peer counsellors should be integrated into multifaceted programmes to promote breastfeeding. To be effective, such interventions should include skilled management of early problems with lactation.

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Use of emergency oxygen in adults

Implementing new acute oxygen guidelines will require education, resources, and a change in culture



Oxygen is often given by medical, nursing, and paramedical staff in community and hospital settings.¹² However, this treatment is commonly neither prescribed nor targeted to a specific saturation. As with any treatment, indications and potential contraindications exist, and complications may occur. In acute emergencies high flow oxygen can save lives by preventing severe hypoxaemia. However, excessive oxygen can cause harm, and inadequate supervision of its use has been reported in both prehospital and hospital audits.^{3,4} To resolve some of these problems, the British Thoracic Society recently published guidelines on the use of emergency oxygen in adults.¹

Despite the importance of oxygen uptake, transport, and delivery to the tissues, the physiology and pathophysiology of impaired oxygen delivery is surprisingly poorly understood by many emergency caregivers. Furthermore, the role of oxygen therapy is an area of medicine where strong opinions exist, despite there being relatively few randomised controlled trials. The resulting controversy has also led to different practices in different settings.

Conflicting advice is often given to health professionals during training, which can result in confusion. For example, many practitioners believe that oxygen alleviates breathlessness in non-hypoxaemic patients, and that high concentration oxygen is beneficial in uncomplicated myocardial infarction and stroke, despite evidence to the contrary.^{1,5,6} Similarly, unnecessary concern about the danger of carbon dioxide retention in all patients with chronic obstructive pulmonary disease can deny potentially life saving treatment—for example, when pneumonia is the cause of acute illness. In light of these

difficulties, guidance is long overdue and the British Thoracic Society guidelines are welcome.¹

The full document is extensive, well referenced, and the product of wide consultation of key professional societies and practitioner groups.¹ The guideline provides a comprehensive review of the theory of acute oxygen therapy, and it covers respiratory physiology, monitoring, management of individual conditions, mask types, prescribing, and how oxygen services should be developed. It provides important practical advice, including modification of prescription charts and how to set the correct oxygen flow on a flow meter.

The methodology is robust, but the evidence is still largely based on observational studies and expert opinion, presumably because randomised controlled studies can potentially disadvantage untreated patients. An executive summary of key recommendations contains the essential information needed by most practitioners, and there are useful sections that specifically meet the needs of ambulance and community staff.

Several aspects of the guidelines deserve emphasis. Firstly, treatment should be targeted to oxygen saturation. Thus, the concentration of inspired oxygen should be varied to maintain the saturation in the target range, in much the same way that insulin is adjusted to achieve a target blood glucose range. In general, the guidelines suggest aiming for physiological rather than raised or “supranormal” oxygen levels, except in certain circumstances such as carbon monoxide poisoning. The recommended target saturation is 94-98% unless the patient is hypercapnic or at risk of hypercapnia, when a lower target range of 88-92% is recommended. Considerable effort has been made to define and justify these target

R M Leach consultant physician, Department of Respiratory and Critical Care Medicine, Guy's and St Thomas' NHS Foundation Trust, London SE1 7EH

Richard.Leach@gstt.nhs.uk
A C Davidson director, Lane Fox Respiratory Unit, Guy's and St Thomas' Hospital NHS Foundation Trust

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ranges and to acknowledge the limitations of a set range. For instance, a fall in saturation of >3% within the normal range (94-98%) may still be important (for example, in pulmonary embolism), and yet many elderly people have a saturation <94% without apparent illness. Targeting treatment to a specific saturation range means that oximetry must be available wherever oxygen is given and this has considerable resource implications. Importantly, the guidelines highlight the poor reliability of detecting cyanosis and the potential pitfalls of oximetry including the lack of warning in carbon monoxide poisoning and the effects of nail varnish.

Secondly, the guidelines emphasise the importance of establishing the cause of tissue hypoxia and show that treatment should encompass more than simply increasing the concentration of inspired oxygen. Thus, in many circumstances, oxygen delivery is best improved either by correcting anaemia or improving cardiac output (or both), or correcting hypoventilation (with non-invasive ventilation) and ventilation-perfusion mismatch with physiotherapy and appropriate positioning of the patient. It should be noted that studies in critical illness have shown no benefit from increasing oxygen delivery to supranormal levels.⁷ Thirdly, a key recommendation is that the dose of oxygen, method of delivery, and target saturation should be written on the drug chart and oxygen saturation monitored and documented in both emergency and routine situations. The guidelines suggest that emphasising that oxygen is a drug might lead to improvements in its appropriate use and in patient safety.

The guidelines highlight the difficulty of identifying people at risk of hypercapnia.¹⁸ They propose that once identified these people should be warned of the danger and issued with oxygen alert cards. This strategy may reduce inappropriate admissions of patients with chronic obstructive pulmonary disease and hypercapnia.⁹

The section on the pathophysiology of hypercapnia in patients with chronic obstructive pulmonary disease and others at risk of hypoventilation questions the widely held, but largely incorrect, view that “reduced hypoxic drive” is central to the mechanism of progressive hyper-

capnia with oxygen treatment.¹⁰ It highlights the double danger of more profound “rebound” hypoxaemia and acute acidosis after sudden (mistaken) withdrawal of oxygen following the development of oxygen induced hypercapnia. The dangers and potential detrimental effects of hyperoxia on cerebral and coronary arteries are clearly outlined, and the routine use of oxygen in stroke, myocardial infarction, obstetric emergencies, and poisoning is questioned unless shock or hypoxaemia are present.^{5 6 11}

The authors of the guideline are to be congratulated on providing a sensible, comprehensive, and practical guide to acute oxygen therapy. However, delivering many of these recommendations will require education at the hospital and community level, the provision of oximeters to monitor saturation in the community and prehospital setting, and a change in “oxygen culture,” particularly if the potential dangers of hyperoxia are accepted. Protecting patients from excessive oxygen requires not only identification of those at risk but communication of that risk to others. This again will require a change in practice for many.

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Heart failure with a normal ejection fraction

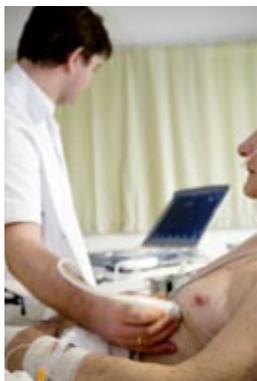
Is underdiagnosed, has a high mortality, and lacks evidence based treatment

The typical image of a patient with heart failure is of a breathless person with a large flabby heart, which contracts poorly with a reduced left ventricular ejection fraction. However, many patients, mainly elderly women, have symptoms of heart failure but their hearts are not enlarged. Echocardiography shows a relatively normal left ventricular ejection fraction but usually with some left ventricular hypertrophy. Because systolic function was thought to be normal or near normal, the term “diastolic heart failure” was coined for this group of patients. However, we now know that systolic function is not entirely normal, and the problem is not only caused by diastolic dysfunction; hence the term

“heart failure with a normal ejection fraction” is more appropriate.

Recent epidemiological studies have shown that heart failure with a normal ejection fraction is now a more common cause of hospital admission than systolic heart failure in many parts of the world.^{1 2} Mortality is also similar for both types of heart failure.¹ Yet recognition of heart failure with a normal ejection fraction is poor and few trials of treatments exist.

In contrast to systolic heart failure, where an echocardiogram can easily show a dilated ventricle and reduced left ventricular ejection fraction, the lack of good non-invasive indices of diastolic function



ARNIO MASSIE/SPL

John E Sanderson professor of clinical cardiology, Department of Cardiovascular Medicine, University of Birmingham, Birmingham B15 2TT
 j.e.sanderson@bham.ac.uk
Gabriel W K Yip associate professor, Department of Medicine and Therapeutics, Chinese University of Hong Kong, Hong Kong
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makes heart failure with a normal ejection fraction more difficult to diagnose. Recent European Society of Cardiology guidelines suggested a diagnostic algorithm for heart failure with a normal ejection fraction.³ In essence, if a breathless patient has left ventricular hypertrophy, left atrial enlargement, and evidence of a raised left atrial pressure either by B-type natriuretic peptide (which is especially useful in primary care) or newer echocardiographic indices then the diagnosis of heart failure with a normal ejection fraction is highly likely.

The pathophysiology behind the symptoms is complex, and not only diastolic abnormalities are involved. In the normal heart, left ventricular twist during systole (which stores energy), motion of the mitral annulus towards the apex during systole (which also helps suck blood into the atrium), and the corresponding untwisting process and recoil in early diastole when that energy is released to generate the negative intraventricular pressure gradient or suction in early diastole, are tightly coordinated both temporally and functionally. This process is followed by the rapid motion of the mitral annulus back towards the base of the heart, which also aids ventricular filling by moving the mitral annulus around the column of the incoming blood.^{4,5} All these aspects of ventricular function increase on exercise, not only to accelerate ventricular ejection, but more importantly to enable rapid filling of the ventricle during a shortened diastole while maintaining a low filling pressure. In patients with heart failure and a normal ejection fraction, this close relation between systole and diastole is disrupted, and recent studies have shown a variety of abnormalities of systolic and diastolic function—reduced myocardial systolic strain, reduced ventricular systolic rotation at rest (which fails to increase normally on exercise), reduced mitral annular motion in systole and diastole, and delayed ventricular untwisting associated with reduced left ventricular suction.^{6,7} Other factors are also involved, such as increased arterial stiffness, which affects ventricular function, and impaired heart rate responses to exercise, similar to patients with systolic heart failure.⁸

Thus, both systolic heart failure and heart failure with a normal ejection fraction have systolic and diastolic functional abnormalities to various degrees and differ mainly by ventricular size. This is probably because infarction—the most common cause of systolic heart failure—is a powerful remodelling stimulus, whereas with hypertension and diabetes—the main causes of heart failure with a normal ejection fraction—the remodelling process is slower and initially hypertrophy occurs without chamber dilatation. However, hypertension can eventually produce ventricular dilatation and typical systolic heart failure if poorly treated.²

Few large scale treatment trials have been conducted in heart failure with a normal ejection fraction because in the early trials of heart failure a reduced left ventricular ejection fraction was a prerequisite for entry. However, one trial found that the angiotensin receptor antagonist candesartan modestly reduced hospital admissions for heart failure but did not significantly affect mortality in patients with heart failure with a

normal ejection fraction.⁹ However, a small randomised controlled trial found that diuretics alone reduced symptoms and significantly improved quality of life, but that adding ramipril or irbesartan was not more efficacious.¹⁰

Another recent study also evaluated the effect of the angiotensin receptor blocker irbesartan on mortality and cardiovascular morbidity in 4128 patients with heart failure with a normal ejection fraction. It found no benefit of irbesartan over placebo in reducing mortality or morbidity from cardiovascular disease.¹¹ These negative results are surprising. Fibrosis of the left ventricle is increased with left ventricular hypertrophy and hypertension. Angiotensin converting enzyme inhibitors and angiotensin receptor blockers can block the fibrogenic action of angiotensin experimentally and have been shown to reduce fibrosis in patients with hypertension.¹² Fibrosis and altered collagen in left ventricular hypertrophy may have a deleterious effect on overall myocardial architecture, particularly ventricular twist and torsion. Nevertheless, the reduction of fibrosis may be an important therapeutic target, and the ongoing studies of spironolactone in heart failure with a normal ejection fraction will be interesting.

In summary, heart failure with a normal ejection fraction is a mixed bag of differing pathologies and aetiologies that in combination cause the elderly heart to fail. Despite being common, this type of heart failure is often not recognised, and evidence based treatment—apart from diuretics for symptoms—is lacking.

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