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Overlays or mattresses to prevent pressure sores?

Mattresses are more likely to be cost effective and patients prefer them

Research pp 1413, 1416

In this issue of the *BMJ* (pp 1413, 1416) the Pressure Trial Group reports the results of a randomised controlled trial of two methods of preventing pressure ulcers and an economic analysis of that trial.[1 2] The European Pressure Advisory Panel defines a pressure ulcer as an area of localised damage to the skin and underlying tissue caused by pressure, shear, friction, or a combination of these.[3] Regardless of aetiology the problem of pressure ulcers in poorly mobile patients is common and can be encountered in many health settings, including at home, in community care, and in intensive care.[4–6] Incidence varies from 0% to 17% for home care, 2.2% to 23.9% for long term care, 0.4% to 38% for hospitals, and 8% to 79% for intensive care.[5 6]

Pressure ulcers are a source of distress to patients, are costly to manage, and can lead to litigation.[4] Their severity is scored by standardised grading systems.[3] Many of the predictive risk scores have reasonable sensitivity but limited specificity and may only be useful for particular groups of patients.[3 5] These imperfect tools mean that moderately expensive preventive technologies are applied to many more patients than will actually require them. The limited ability to target these interventions decreases their cost effectiveness.

The problem of pressure ulcers is given little space in established textbooks of pathology, medicine, surgery, or intensive care—perhaps reflecting a lack of interest by doctors.[3 7] A recent literature review identified only three randomised controlled trials in the intensive care literature over a 20 year period,[3] and a Cochrane review identified only 41 randomised controlled trials across all clinical settings, including accident and emergency departments as well as operating theatres.[4] The systematic review concluded that the relative effectiveness of alternating pressure surfaces was unknown.

In this context we should welcome the PRESSURE trial, which investigated two preventive technologies which have a large difference in acquisition costs. This was a large randomised controlled trial of almost 2000 poorly mobile patients at high risk of developing pressure ulcers in a variety of settings but excluding intensive care. The interventions compared were alternating pressure mattresses and alternative pressure overlays (the mattresses costing four times as much to buy as the overlays). The trial was methodologically rigorous, using a variety of scores to identify patients at risk and to grade their ulcers. It also used appropriate tools to adjust for factors that might bias outcomes.

This study found little difference between the two devices. Notably, fewer patients expressed dissatisfaction with mattresses than with overlays. The trial also provided further empirical evidence of important risk factors for pressure ulcers.

The lack of difference between these technologies is, perhaps, unsurprising. Given the similarity of the technologies the expected 50% reduction in pressure ulcers may have been too optimistic. Moreover, 349 patients (18%) did not receive the intended device, and 600 patients (30%) were changed from the mattress to which they were randomised. A per protocol analysis might have provided additional insights into the relative efficacy of these devices.[8] Choice of trial size often involves pragmatic considerations as well as statistical science.[9]

The economic evaluation conducted as part of this trial also used robust methods, and in many respects the authors went as far as they could with the data available.[2] The sample size of the trial meant that at conventional 5% significance levels there was no evidence of a difference in either costs or days free from pressure ulcers. This is a common problem with randomised controlled trials, which are rarely adequately powered with respect to economic outcomes.

One of the limitations with the economic evaluation was the reliance on a single clinical outcome: days free from pressure ulcers. It is unclear whether this measure captures all the benefits that might be important to patients. The economic evaluation still provides useful information for decision makers, however, in finding that the alternating pressure mattresses have an approximately 85% chance of being considered cost effective compared with the alternating pressure overlays.[2] This means that a decision to use alternating pressure mattresses has about a 1 in 7 chance of being wrong.

The PRESSURE trial and its economic analysis, despite some limitations, is welcome and will help healthcare providers to make better decisions when buying alternating pressure devices. It also raises the standard for research in the neglected but important field of preventing and treating pressure ulcers.

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Proteinuria, renal impairment, and death

If reducing proteinuria improves cardiovascular outcomes, urine dipstick testing will become crucial in hypertension

Research p 1426

It has been known for many years that patients with chronic kidney disease have a significantly increased risk of cardiovascular morbidity and mortality. Many studies have shown that, in renal failure, increasing proteinuria and worse renal function are associated with more rapid progression and a higher incidence of cardiovascular events. Does the same hold true for patients with relatively minor degrees of renal impairment and low levels of proteinuria, even microalbuminuria? Is the combination especially important? And does this affect the management of patients?

First some technicalities. Proteinuria usually refers to protein that is detectable in urine with conventional urine dipsticks, and the amount of protein can vary from 300 mg to several grams a day. Proteinuria can be quantified reliably and easily by using spot urine protein: creatinine ratios, where normal is said to be <20 mg/mmol (but see below). Just to confuse the issue, diabetologists have for many years measured albumin (rather than total protein) excretion in urine as an excretion rate (mg/day or μg/minute) and more recently as albumin:creatinine ratios. In patients with low levels of proteinuria the two results may be quite different since much of the urinary protein in this setting may not be albumin. Microalbuminuria refers to very low excretion rates of albumin (>2.5 (men) or >3.5 (women) to 30 mg/mmol), not detectable by conventional urine dipsticks. Defining a normal cutoff is proving increasingly difficult as it becomes clear that even within the normal range higher levels of protein excretion are associated with poorer vascular and renal outcomes.

A large body of data indicates that in patients with relatively normal renal function a lower glomerular filtration rate is associated with increased risk for poor cardiovascular outcomes. Most recently the rates of cardiovascular outcomes in the ALLHAT study (high risk hypertensive patients treated first line with either chlorthalidone, amlodipine or lisinopril) were re-analysed to look for the effect of renal function.[1] A low glomerular filtration rate (GFR; present in >5500 patients (13%)) independently predicted increased risk for coronary heart disease, and patients with a baseline GFR <53 ml/min/1.73 m^2 had a 32% higher risk of heart disease than those with GFR >104 ml/min/1.73 m^2. Many elderly patients in particular will have a GFR within this lower range and are not at high risk for developing progressive renal failure. In this study none of the anti-hypertensive agents were better at protecting patients with reduced GFR from fatal coronary heart disease or non-fatal myocardial infarction. Similarly, in the cardiovascular health study of 4893 low risk subjects with a predicted GFR of 15-130 ml/min/1.73 m^2, each 10 ml/min lower GFR throughout the range was associated with a 5% increased risk for cardiovascular disease.[2]

Proteinuria has been known to be a marker for cardiovascular disease for some time, both in diabetic and non-diabetic patients. Various hypertension studies have demonstrated the risk posed by proteinuria—for example, in the INSIGHT study (nifedipine and diuretics for treatment of hypertension) proteinuria was as important a risk factor for cardiovascular events as abnormal serum creatinine, and equal to a previous myocardial infarction.[3] The LIFE study (losartan intervention for end points in hypertension) showed a similar finding, increasing albuminuria being associated with increased risk of cardiovascular end points, fatal and non-fatal stroke, and cardiovascular mortality, as a continuous effect, with no threshold. Left ventricular hypertrophy, coronary artery calcification, and carotid artery stenosis are all more common in apparently normal individuals with increasing proteinuria, even within the normal range. Microalbuminuria is also associated with a failure of nocturnal dipping in blood pressure, insulin resistance, and abnormal vascular responses to various stimuli. Finally, in the Copenhagen heart study the risk for coronary heart disease or death doubled once microalbuminuria exceeded 5 μg/min, a very low threshold previously considered well within "normal."[4]

The best data on the interaction of proteinuria and renal impairment on cardiovascular outcomes come from a large study recently reported from Japan. This was a huge population survey including a total of 96 739 normal individuals aged 40-79 who were followed for 10 years. At outset 3% of the men and 2% of the women had proteinuria (assessed simply by dipstick analysis), and 3% had a GFR <60 ml/min/1.73 m^2. Compared to those without proteinuria, those with proteinuria had 1.8 to 2.9 times the (age adjusted) risk of death from coronary heart disease, cardiovascular disease, and all other causes. The age adjusted risk of death was 1.3 to 2.1 times higher for stroke, cardiovascular disease, and all other causes among people with the lowest GFR category (<60 ml/min/1.73 m^2) than those with the highest GFR category (>100 ml/min/1.73 m^2). Men with both proteinuria and

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