MEDULLARY LAVAGE REDUCES EMBOLIC PHENOMENA AND CARDIOPULMONARY CHANGES DURING CEMENTED HEMIARTHROPLASTY

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We randomised 24 patients before they had a cemented hemiarthroplasty for hip fracture to receive either thorough or minimal saline lavage of the femoral canal. We then determined the effect in each group on the thromboembolic and cardiopulmonary responses to the pressurised insertion of cement, using transoesophageal echocardiography to show the echogenic embolic response.

We found a statistically significant reduction in both the duration of the response and the number of large emboli in patients who had had thorough lavage as compared with the control group with minimal lavage. There was also less disturbance of pulmonary function, as assessed by the change in end-tidal CO₂ levels and oxygen saturation, in patients who had had thorough lavage. Three patients had a significant fall in blood pressure during cement insertion; all had only minimal lavage.

We consider that thorough lavage should be an essential part of the preparation of the proximal femur before cement insertion.

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The pressurised insertion of cement into the medullary canal of the femur during hemiarthroplasty causes an embolic response which can be shown by transoesophageal echocardiography. Showers of material are seen to pass through the right atrium and enter the pulmonary circulation (Wenda et al 1989; Christie et al 1994) and may be responsible for the cardiorespiratory changes which follow the insertion of cement (Kim and Ritter 1972; Modig et al 1975; Samii et al 1980). Venting the femur and the use of pulsatile lavage of the medullary canal have both been shown experimentally to reduce these changes (Breed 1974; Herndon, Bechtol and Crickenberger 1974; Kallos et al 1974; Sherman et al 1983), probably by decreasing the embolic load of fat and marrow which enters the venous circulation (Wheelwright et al 1993).

Our study aimed to establish whether preliminary lavage of the medullary canal during hemiarthroplasty reduced the embolic load and the severity of cardiorespiratory changes when cement is inserted.

PATIENTS AND METHODS

From November 1993 to April 1994, 24 patients with displaced intracapsular fractures of the femoral neck gave informed consent for transoesophageal echocardiography during their treatment by cemented hemiarthroplasty. Random selection was made between minimal washout of the medullary canal before cement insertion and extensive washout by allocation of alternate cases to groups. Both groups of patients had been fully ambulant before their fracture, were medically fit (ASA grade I or II) and well matched for age and sex (Table I).

At operation, a 5 MHz biplane echocardiography probe (Hewlett Packard, Wokingham, UK) was placed in the

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Table I. Details of the two groups of patients

	Lavage group (n = 12)	Control group (n = 12)
Mean age in yrs (range)	72.1 (68 to 85)	73.6 (69 to 88)
Male:female	3:9	2:10
ASA Grade I	9	9
II	3	3

oesophagus of each patient to monitor embolic material passing through the heart. A standard technique was used for both groups, with the same surgical approach, preparation and reaming of the proximal femur. In the thorough lavage group, however, the reamed canal was copiously irrigated using at least one litre of saline with continuous suction and thorough drying of the cavity. We aimed to maximise the removal of medullary canal contents. In the control group, minimal lavage used less than 50 ml of saline, and was followed by drying of the canal. Cement insertion in both groups included distal containment using a cement restrictor and proximal pressurisation. A Hastings bipolar prosthesis (De Puy International Ltd, Leeds, UK) was then inserted.

Echocardiographic recordings were made on video tape and studied later by one observer who was unaware of the group of the patient. Heart rate, blood pressure, arterial oxygen saturation and end-tidal CO₂ levels were also monitored throughout each operation.

We graded the embolic response seen on echocardiography as described in our previous study (Christie et al 1994):

Grade I. A few fine emboli.

Grade II. A cascade of many fine emboli.

Grade III. A cascade of fine emboli with some emboli of more than 1 cm in diameter.

Grade IV. Large embolic masses of more than 3 cm in diameter.

We also recorded the total duration of the embolic response.

RESULTS

There was no embolic response before surgery or during the approach to the hip. During reaming of the femur there was a transient grade-II response in three patients from each group, but this was not associated with any significant changes in heart rate, blood pressure, oxygen saturation or end-tidal CO₂ levels.

There was no embolic response during lavage, but cement insertion, pressurisation and insertion of the prosthesis produced showers of emboli in all patients. We found significant differences between the groups in the mean maximal grade of embolism, the mean duration of the response and the mean number of emboli larger than 3 cm in diameter ($p < 0.05$; Table II). During the appearance of embolic cascades there was a fall in oxygen saturation and end-tidal CO₂ levels; the mean fall was less in the lavage group, the difference reaching statistical significance for end-tidal CO₂ levels (Table II). Three patients had reductions of systolic blood pressure greater than 20 mmHg, or of over 10 mmHg diastolic pressure during emboli showers; all three were in the control group.

We found no obvious difference in the postoperative morbidity or in the incidence of cardiorespiratory problems between the two groups. No patient developed clinical signs of fat embolism.

DISCUSSION

Animal studies suggest that thorough lavage of the medullary canal removes some of the marrow and fat which may otherwise be forced into the venous circulation during cement pressurisation at a simulated arthroplasty (Sherman et al 1983; Orsini et al 1987; Wheelwright et al 1993). Our findings support this; we have shown that thorough lavage reduces both the number of large emboli and also the total duration of the embolic response. Experiments have shown that both the volume and pressure of lavage influence the efficacy in reduction of embolic load (Byrick et al 1989). We found that the use of high-volume irrigation alone produced a reduction in embolisation. It seems possible that the use of high volumes of irrigating solution followed by adequate drying of the canal may be as effective as commercially available high-pressure, high-volume pulsatile-lavage units, but this requires further study.

Acute hypotension, transient hypoxaemia and elevation of pulmonary artery pressure are well known to occur

Table II. Changes observed during cementing and prosthetic insertion

	Lavage group (n = 12)	Control group (n = 12)
Grade of maximal embolic response*		
I	2	0
II	4	1
III	4	9†
IV	2	2
Mean duration of embolic response (sec)	270.4 (100 to 720)	421.9 (175 to 925)†
Mean number of large emboli	2.3 (0 to 10)	7.1 (0 to 18)†
Mean fall in oxygen saturation (%)	1.4 (0 to 6)	2.3 (0 to 4)
Mean fall in end-tidal CO ₂ (mmHg)	1 (0 to 4)	5.5 (0 to 10)†

* see text

† significance of difference $p < 0.05$ (Mann-Whitney U test for non-parametric data)

during cemented arthroplasty both in patients (Charnley 1970; Modig et al 1975) and in animals (Byrick, Kay and Mullen 1987). Fat embolism has been implicated (Sevitt 1972; Breed 1974), but the exact pathophysiological mechanisms are poorly understood and other factors such as the pharmacological action of methylmethacrylate (McMaster, Bradley and Waugh 1974), the generation of thromboplastins (Modig et al 1975) and the release of prostaglandins (Byrick et al 1991) have been suggested.

Our findings suggest that marrow embolism plays a definite role in that reduction of the embolic load, produced by thorough lavage, also reduced the changes in oxygen saturation and end-tidal CO_2 . Fat and marrow embolism appears to produce a marked discrepancy in ventilation/perfusion, with relative overventilation of some alveoli which are underperfused because of occlusion by microemboli. This results in a fall in the end-expiratory alveolar CO_2 levels and a reduction in the partial pressure of oxygen in pulmonary venous and arterial blood. These small changes in partial pressure produce only a minimal change in oxygen saturation and it is not surprising therefore that the changes in end-tidal CO_2 were more marked than those for oxygen saturation. The transitory nature of the changes confirms the theory that homeostatic mechanisms operate to divert pulmonary blood from areas where microemboli have produced vasoconstriction, at the expense of increasing the fraction of right-to-left shunted blood (Goodman, Seaber and Silver 1978; Ries et al 1993).

We found no direct relationship between the severity of the embolic response and the observed impairment of cardiorespiratory stability, although this has been demonstrated during other invasive intramedullary procedures (Christie et al 1995). However, we only saw hypotensive episodes in our minimal-lavage control group, with more severe fat and marrow embolism. It is possible that the monitoring methods which we used were not sensitive enough to detect minor changes in blood pressure or that other factors such as pre-existing respiratory impairment or subclinical ischaemic heart disease may have influenced the response.

Cardiorespiratory changes are not caused only by the injection of cement; pressurisation of the canal without cement has also been shown to produce haemodynamic changes in animal experiments (Crout et al 1979; Orsini et al 1987). Blood levels of the monomer of cement in experimental animals have been shown to be much lower than those required to produce systemic effects (Peebles et al 1972; McLaughlin et al 1973). It is not clear whether the vascular changes are caused purely by the mechanical action of emboli occluding the microcirculation, or by the activation of mediators such as thromboplastins and prostaglandins. Many chemical mediators have been detected in patients and in experimental animals (Byrick et al 1991; Wheelwright et al 1993), but no causal relationship has been established; Byrick et al were unable to prevent haemodynamic instability by using ibuprofen to inhibit

prostaglandin release. It may be that a combination of factors is involved in producing cardiovascular instability, but our knowledge of the relative importance of each remains vague.

None of the patients in our present series had clinical signs of fat embolism and there was no evidence that the physiological changes in the control group were detrimental to their subsequent recovery. However, it is clear that the embolic phenomena which occur during cement insertion may sometimes be associated with severe hypoxia (Jones 1975; Alexander and Barron 1979; Mebius and Hedenstierna 1982) or even intraoperative death (Powell et al 1970).

Further research is required to improve our knowledge of the pathophysiological mechanisms and to identify the 'at-risk' population. Until there is more precise information, we recommend that thorough lavage of the medullary canal should be regarded as essential in the preparation of the proximal femur for cemented arthroplasty. During the insertion of cement, the anaesthetist should carefully monitor cardiorespiratory stability and be prepared to correct rapidly any hypotension or hypoxia that develops.

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