

Correspondence

We welcome letters to the Editor concerning articles which have recently been published. Such letters will be subject to the usual stages of selection and editing; where appropriate the authors of the original article will be offered the opportunity to reply.

Letters should normally be under 300 words in length, double-spaced throughout, signed by all authors and fully referenced. The edited version will be returned for approval before publication.

The Gotfried percutaneous compression plate compared with the conventional classic hip screw for the fixation of intertrochanteric fractures of the hip

Sir,

I was most interested to read the paper by Kosygan et al¹ in the January 2002 issue entitled a 'The Gotfried percutaneous compression plate compared with the classic hip screw for the fixation of intertrochanteric fractures of the hip'.

The authors conclude that the minimally invasive percutaneous compression plating (PCCP) procedure has theoretical rather than practical advantages and that in the present study "no practical benefit was associated with its use". On the basis of postoperative complications alone, it is difficult to see how they arrive at this conclusion. They report a total of six complications (11.5%) in the PCCP group and 21 (37.5%) in the classic hip screw (CHS) group (Table III). The magnitude of this difference was ignored by the authors and not subjected to statistical analysis. Using the chi-squared test, an independent statistician has found this difference to be clearly significant ($p = 0.0014$). Furthermore, the complications described include chest infections (one in the PCCP group, four in the CHS group) and cardiac complications (one in the PCCP group, six in the CHS group). Despite these findings, the authors state that "the complications after operation were similar in both groups". This merits some explanation. Since in addition blood loss and transfusion requirements were less in the PCCP group, the practical advantages and benefits speak for themselves. This study demonstrates that reducing surgical trauma in this group of patients (mean age 82 years) significantly reduced postoperative morbidity.

There are a number of important principles associated with the use of the PCCP foremost of which is prudent reduction of the fracture. Regrettably, this principle has been misunderstood by the authors, leading them to conclude that because the PCCP is a fixed-angle device it cannot be universally applicable. In current practice, the appropriate angle of a CHS/DHS plate is determined by the neck-shaft angle achieved on reduction of the fracture. Thus CHS/DHS plates with various degrees of valgus and varus are available with these devices. In marked contrast, the PCCP philosophy is that of adapting the fracture to the angular configuration of the plate, i.e. of reducing the fracture to a

neck-shaft angle of 135°. No varus angulation (neck-shaft angle less than 135°) is accepted. The rationale of this technique is to subject the fracture and the sliding fixation device to a compressive load rather than a bending load. In practice, this can be achieved very simply. When the fracture is initially in varus, this is done by simply applying suitable traction on the fracture table, or when there is excessive valgus, by releasing traction. The ultimate result will be a neck-shaft angle of 135° without any material change in the reduction. This technique is most important in unstable fractures with multiple fragments. During reduction, a template on the image-intensifier screen is used to verify that the desired angle of 135° has been achieved.

Another cardinal principle of the PCCP technique, not addressed by the authors, is the reduction of sagging of the posterior fracture and the maintenance of this reduction for the entire duration of the operation. Dedicated instrumentation has been developed to accomplish this task efficiently and without undue effort on the part of the assistant, as described previously.²

A further feature of the PCCP procedure is fixation using double telescoping neck screws as opposed to single-axis fixation with the CHS. This provides rotational stability to the femoral neck-head fragment, with the aim of preventing cut-out.³ In support of this, the authors report no cut-outs in the PCCP group but two in the CHS group, which were revised to intramedullary fixation. Cut-out is a frequently reported complication with CHS/DHS devices, but none was observed in association with the PCCP. It is surprising that the authors failed to comment on this finding.

Independent, randomised prospective studies are needed to confirm the results obtained by the inventor² and these should include assessment of all the appropriate parameters. The present study¹ has not considered any functional outcomes, which must be an integral part of any such investigation. In addition, in regard to the PCCP, the important concept of preservation of the lateral trochanteric wall² has received no mention. The statement in the abstract which claims that at six months there was no difference in rates of healing of the fracture or in failure of the implant is not referred to at all in the Results section, which clearly implies that these parameters were not formally evaluated in the study. Finally, no reference is made to a recently published paper, which looks at the results of 97 patients treated with the PCCP, and includes functional results.²

According to the authors' findings the learning curve involved only two cases, in the 52 patients 30 units of blood were saved, there was a significant reduction in postoperative complications and there were no 'cut-outs'. What therefore has led them to conclude that "we cannot therefore recommend its (PCCP) widespread introduction at this time"? On the basis of their findings should not their conclusion be exactly the opposite?

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1. Kosygan KP, Mohan R, Newman RJ. The Gotfried percutaneous compression plate compared with the conventional classic hip screw for the fixation of intertrochanteric fractures of the hip. *J Bone Joint Surg [Br]* 2002;84-B:19-22.
2. Gotfried Y. Percutaneous compression plating of intertrochanteric hip fractures. *J Orthop Trauma* 2000;14:490-5.
3. Gotfried Y, Cohen B, Rotem A. Biomechanical evaluation of the percutaneous compression plating system (PCCP) for hip fractures. *J Orthop Trauma*: in press.

Authors' reply:

Sir;

We thank Dr Gotfried for his letter and his interest in our article.

Most of the so-called "complications" which occurred after surgery were relatively uncomplicated, required little specific treatment and were included for the sake of completeness. It is inconceivable that most of the events described can be causally related to the choice of implant, especially since there was no significant difference in the major parameters measured including length of hospitalisation, postoperative rate of rehabilitation and mortality. It is clear that the figures for each individual minor complication were too small for formal statistical analysis and Dr Gotfried has only been able to do so by aggregating all the data together. The fact that he has been able to produce a p value in those circumstances which he claims to be significant, indicates his confusion between clinical relevance and quasi-statistical significance.

We cannot accept that a fracture should be adapted to fit an implant which is under evaluation. We consider that if the implant will not fit satisfactorily into an individual patient then it is the implant which requires modification, not the patient.

Our paper reported two 'cut-outs' in the CHS group but none in the PCCP group. Quite some time has elapsed since our paper was submitted during which it has become apparent that one case which was considered to be united clearly was not since a cut-out occurred. In a second case the lower telescoping screw in the neck dissociated from the rest of the implant requiring its reinsertion. Union in that case may also not have occurred.

Clearly, the PCCP is not without its problems. As expected with any implant it does have a technical failure rate and its use certainly does not guarantee an absence of cut-out.

Any move towards minimally invasive surgery must be applauded and the PCCP may be a move in the right direction. It does not appear to perform any better than the classic hip screw and certainly it was without effect on the most important objective indicators in this case. This is, of course, to be expected since the PCCP is no more than yet another internal fixation device. The problem with this 'unsolved fracture' is the inherent adverse biology of the patient, something that is unlikely to be addressed by simply using smaller incisions! Further trials of this device are essential before it is considered to be a panacea as suggested by its inventor.

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Cardiac output during hemiarthroplasty of the hip

Sir;

I read with interest the article by Clark et al¹ in the April 2001 issue entitled 'Cardiac output during hemiarthroplasty of the hip'.

In the context of no significant change in mean arterial pressure and heart rate between the two groups, how do the authors explain the disproportionate fall in stroke volume and the cardiac output? The patients in the group with cemented prostheses by definition must have launched a compensatory physiological response, such as an increase in the heart rate, to dampen the effect of reduction in stroke volume.

Did the authors detect any difference in the fluid requirement of the patients in the two groups? Is it correct to assume that none of the patients in the study received any vasopressor agents?

Did the authors detect any episodes of dysrhythmia (including bradycardia), hypoxia or hypotension during surgery, particularly at the time of cementation in any of their individual patients? We have previously observed that cardiorespiratory disturbances such as dysrhythmia, hypoxia, and a significant decrease in systolic blood pressure occurred during cementation and that these changes preceded the fatal events in all the patients who died intraoperatively during cemented hip arthroplasty at our institution.²

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1. Clark DI, Ahmed AB, Baxendale BR, Moran CG. Cardiac output during hemiarthroplasty of the hip. *J Bone Joint Surg [Br]* 2001;83-B:414-8.

2. Parvizi J, Holiday AD, Ereth MH, Lewallen DG. Sudden death during primary hip arthroplasty. *Clin Orthop* 1999;369:39-48.

Authors' reply:

Sir;

We thank Mr Parvizi for his interest in our paper. To reply to his various points, first the study utilised a transoesophageal Doppler technique and not transoesophageal echocardiography. Secondly, some of the patients were receiving beta-blocking agents for mild cardiac disease and may have been unable to produce a significant physiological response as a compensatory tachycardia secondary to a decrease in cardiac output. The drug history is not a variable which we controlled for, as was stated in the paper. If showers of emboli were taking place, then the patients' compensatory physiological response may have been to elevate or reduce the systemic vascular resistance (SVR). This could only be measured by a pulmonary artery catheter. An elevated SVR, implying circulatory vasoconstriction, would give rise to the observed phenomenon of reduced cardiac output and stroke volume, a small reduction in mean arterial pressure (MAP) and no change in heart rate. Pulmonary artery catheters were not used in our study because there is a recognised incidence of adverse events.

The MAP did show a reduction in the group with cemented prostheses, but as the size of the study was small this may be representative of a type-II error and may not be large enough to show a statistically significant decrease in MAP. This potential limitation was addressed in the paper.

Thirdly, there was no significant difference in fluid requirements between the groups and none of the patients received any vasopressor agents unless there was a clear indication from the attending clinician, when an intravenous bolus of 3 mg of ephedrine was used. This occurred in two patients. The measurement of the MAP at the point of cementing was taken before any agents were given.

Finally, we were fortunate to have no deaths intraoperatively during the study. During surgery we did not detect any episodes of hypoxia, hypercarbia, hypotension or dysrhythmia at or around the time of cementation, with the exception of occasional ventricular ectopic beats which were present before the induction of anaesthesia. Monitoring included pulse oximetry, end-tidal capnography and intraoperative analysis of the ST segment of the ECG. Since intermittent non-invasive monitoring of the blood pressure was used, there may, however, have been transient undetected periods of self-resolving hypotension which would only have been detected by invasive arterial monitoring.

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Tuberculosis of the thoracic spine

Sir,

We read with interest the article in the August 2001 issue by Mehta and Bhojraj entitled 'Tuberculosis of the thoracic spine'.

The antitubercular therapy (ATT) advised by the authors¹ was four drugs (rifampicin, isoniazid, ethambutol and pyrazinamide) for one year. The accepted regime^{2,3} is four drugs (rifampicin, isoniazid, ethambutol and pyrazinamide) for two months and then withdrawal of pyrazinamide. The remaining three drugs are continued for the next four months and then rifampicin and isoniazid for another six months. Ethambutol should not continue for the full regime since it causes dose-related retrobulbar neuritis.²

The indications of surgery for group A cited by the authors are as follows:

a) Spinal deformity of more than 30°. No patient, however, in the reported study had any deformity.

b) Significant neurological deficit at presentation. Only five patients in this group had a neurological deficit of which three were in Frankel C. The extent of the neural deficit of the remaining two patients is not mentioned. The patients with neural deficit in whom MRI showed a relatively preserved cord with evidence of oedema/myelitis and predominantly fluid collection in the extradural space, may recover on non-operative management.^{3,4}

c) Failure to respond to non-operative management. It is premature to label a patient without a neural deficit on ATT as a non-responder after six to eight weeks. In a patient with a neurological deficit the conservative trial should not be more than three to four weeks.⁵ In both categories labelling the patients as non-responders at six to eight weeks is unconvincing.

d) Persistent severe pain. In patients with Pott's disease with a stable, undeformed vertebral column, proper rest, bracing, ATT and analgesics effect substantial relief from pain in all cases. Pain *per se* should not be an indication for a major procedure such as thoracotomy, debridement and fusion of vertebral bodies. Seven patients operated on in this group had no deformity, instability or neurological deficit. The indication for surgery in these patients is obscure.

Spinal tuberculosis without unsightly kyphosis and neurological symptoms is a medical rather than a surgical condition. Surgery should be reserved for those patients who have advanced tuberculosis with unacceptable complications such as paraplegia and/or deformity.⁶

The aim of management is to allow healing of the disease by the judicious use of modern antitubercular drugs and surgery.

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1. Mehta JS, Bhojraj SY. Tuberculosis of the thoracic spine: a classification based on the selection of surgical strategies. *J Bone Joint Surg [Br]* 2001;83-B:859-63.
2. Watts HG, Lifoso RM. Tuberculosis of bones and joints. *J Bone Joint Surg [Am]* 1996;78-A:288-98.
3. Jain AK, Kumar S, Tuli SM. Tuberculosis of spine (C1-D4). *Spinal Cord* 1999;37:362-9.
4. Jain AK, Jena A, Dhammi IK. Correlation of clinical course with magnetic resonance imaging in tuberculous myelopathy. *Neurol India* 2000;48:132-9.
5. Myung Sang Moon. Spine update tuberculosis of the spine controversies and a new challenge. *Spine* 1997;22:1791-97.
6. Tuli SM. *Tuberculosis of the skeletal system*. 2nd ed. Bangalore, etc. Jaypee Brothers Medical Publishers PVT Ltd, 1997:241.

Author's reply:

Sir,

We thank Dr Jain and Dr Sinha for their interest in our article. Our response to their points is as follows:

1) Although an 'accepted regimen' is suggested this does not disqualify others such as the one we have used with success for several years. There is no 'accepted' protocol for antitubercular chemotherapy in spinal tuberculosis. It is important to adhere to the principles of multidrug chemotherapy and to provide cost-efficient and effective treatment for the patient.

2) Although ethambutol causes retrobulbar neuritis, it has a well-defined place in the chemotherapeutic armament for the treatment of tuberculosis. It has been recommended as a single dose of 1200 to 1500 mg (20 mg/kg body-weight) in the morning or as divided doses for 12 to 18 months.¹ The ocular side-effects of ethambutol are noted to be reversible.² Patient education, regular ophthalmological assessments and discontinuance of the drug when side-effects appear are recommended.^{3,4} Ethambutol has been documented as being safe in children over five years of age in a dosage of 15 mg/kg/day.^{5,6}

3) Group A. Five patients in group A had a neurological deficit of which three were in Frankel C, and two in Frankel D, with intact sphincters. Both of the last two recovered completely after the operation. An error in the text has appeared as regards the deformity. None of the cases had a deformity greater than 30°.

4) Our article gives guidelines for the choice of approach once surgery is indicated, and does not delve into the problems as to when surgery should be performed. It is now common knowledge that uncomplicated tuberculosis of the spine is a medical disease. Surgery only on advanced cases and on severe deformities should be a thing of the past and we must try to strive towards preventing such deformities.

5) Pain *per se*, is not an indication for surgery as we have found that a substantial number of patients show improvement after commencing the chemotherapy and do so early, within the first few weeks. If however, the pain does not improve at all, continues to be severe or becomes worse, we recommend an operation.

6) It seems arbitrary to state that it is too soon to label non-responders at six to eight weeks. We actively seek a non-tubercular aetiology if there is no clinical or radiological response by six to eight weeks. In patients with a neurological deficit, the time frame for operation clearly depends on the intensity of the deficit. For example, we do not operate on patients with upper motor hyperreflexia in the lower limbs even at three months, while a dense/progressive deficit probably needs surgical attention much earlier than after three to four weeks.

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1. Tuli SM. *Tuberculosis of the skeletal system: bones, joints, spine and bursal sheaths*. 2nd ed. Bangalore, etc: Jaypee Brothers Medical Publishers PVT Ltd, 1997.
2. Srivastava AK, Goel UC, Bajaj S, et al. Visual evoked responses in ethambutol induced optic neuritis. *J Assoc Physicians India* 1997;45:847-9.
3. Sivakumaran P, Harrison AC, Marschner J, Martin P. Ocular toxicity from ethambutol: a review of four cases and recommended precautions. *N Z Med J* 1998;111:428-30.
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