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.

FEMORAL COMPONENT REVISION USING IMPACTED MORSELLISED CANCELLOUS GRAFT

Sir,

May I offer some comments on the interesting paper published in the November 1996 issue by Malkani et al entitled 'Femoral component revision using impacted morsellised cancellous graft'.

The authors have shown experimentally that the axial and torsional stability of the femoral component after impaction grafting and recementing is very little less than that achieved using the same stem after a simulated primary cemented arthroplasty. They are justified in their conclusion that impaction grafting ". . . provides immediate stability of the implant". Their evidence, however, does not entitle them to conclude also that the operation ". . . restores the integrity of the proximal femur . . .".

To make such a suggestion on the basis of their experimental work is to confuse the mechanical and biological aspects of this procedure. The provision of immediate stability of the implant is the mechanical goal of the operation and in vivo is technically demanding in that it requires care and attention to detail, although it is not usually technically difficult when the cortical tube is intact. The restoration of the integrity of the proximal femur, however, is what the surgeon hopes will occur as the graft is progressively vascularised and replaced by host bone, and is fundamentally a biological matter. This will not occur unless stability is achieved at operation, but at the present time it is not certain that it will regularly occur even if stability is achieved. Moreover, whether such replacement of the graft is necessary for a satisfactory clinical outcome of a stable reconstruction in the longer term is, again, not known.

Although this operation, as far as the femur is concerned, has been in use for almost ten years, its scope is far from fully established and it is being applied steadily to more and more challenging cases. In this respect, it is important to place the experimental work of Malkani et al in an appropriate context. In their experiments, the femoral diaphyses were intact, but in many cases in which this method is being used today, there are major diaphyseal defects including, sometimes, complete circumferential loss of whole segments of the femur. It is dangerous to apply their conclusions to such difficult problems in which the creation of stability is very much more demanding than in the experimental situation. Much still remains to be learned about the biology and limitations of the procedure.

Finally, it is entirely inappropriate to refer to this operation as "the Ling technique". The operation, without cement, was first performed in Exeter in 1985. Marked subsidence of the femoral component occurred although the patients' pain was relieved. The decision to add cement to the procedure was taken in early 1987 and was based on the satisfactory results obtained in the acetabulum, using impacted grafts and cement, by Shroff et al from Nijmegen. The first impaction grafting using cement in the femur in the series reported from Exeter was actually done by Gie in May of 1987. The subsequent development of the operation and the instrumentation was a joint effort between orthopaedic surgeons and engineers in Exeter and Nijmegen.

R. S. M. LING, OBE, MA, BM, FRCS, Hon FRCS Ed
Wonford Road
Exeter, UK.

Author's reply:

Sir,

We thank Professor Ling for his comments regarding our paper and agree that one cannot conclude from a cadaver model that impaction grafting will restore the integrity of the proximal femur. With the use of cancellous graft and cortical strut grafts we are restoring femoral bone deficiency, but the extent of bone restoration is largely based on revascularisation which we accept is a biological process.

We also agree that our revision model had an intact cortical canal. We created a sclerotic, thin proximal femur. There were no diaphyseal defects or segmental bone loss. The stability of compaction grafting used in massive bone loss will have to be judged on an individual basis.

We agree that long-term clinical data will determine the true efficacy of this type of reconstructive procedure and acknowledge
the parts played by Professor Slooff and Mr Gie in its development.

A. L. MALKANI, MD
University of Louisville School of Medicine
Louisville, USA.

LUDLOFF’S MEDIAL APPROACH FOR OPEN REDUCTION OF CONGENITAL DISLOCATION OF THE HIP

Sir,
We read with interest the article ‘Ludloff’s medial approach for open reduction of congenital dislocation of the hip’ by Koizumi et al in the November 1996 issue. We do not agree, however, with the authors’ suggestion that the medial approach for open reduction is unsatisfactory in the treatment of congenital dislocation of the hip.

There are two major reasons for their less successful results compared with other series. First, they did not divide the psoas tendon and after operation they applied a spica cast in extreme abduction. The psoas tendon is an important extra-articular barrier to reduction. It must always be sectioned to reduce the hip concentrically and to prevent further subluxation or redislocation. Immobilising the hip in forced abduction is one of the factors in the development of avascular necrosis (AVN) of the femoral head. Secondly, the authors do not take into account the age of the patient at operation. Age has a marked influence on the outcome of hips treated by this approach, with better radiological and clinical results in those operated on at seven to 18 months. Under seven months of age there is a considerable risk of damage to vascular structures with the medial approach and a higher rate of AVN of the femoral head when compared with older age groups, although they are of group I according to Kalamchi and MacEwen’s classification. Over the age of 18 months, soft-tissue procedures are not suitable and it is better to perform an acetabular reconstruction, a proximal femoral osteotomy or both. Better results can be obtained if the child has not walked before surgery.

We would like to know how many hips have been treated with an orthosis before surgery, as this may also influence the results.

H. ÖMEROĞLU, MD
A. Y. TABAK, MD
A. BICIMOĞLU, MD
Ankara Numune Hospital
Ankara, Turkey.


Authors’ reply:

Sir,
We read with interest the article by Koizumi et al entitled ‘Ludloff’s medial approach for open reduction of congenital dislocation of the hip’. The findings were at variance to our own experience with this procedure.

Between 1985 and 1992 we performed medial open reduction on 83 hips in 68 children with developmental dysplasia of the hip. Our mean follow-up was 38 months (7 to 90). Twelve had avascular necrosis (AVN) (14%), 19 had acetabular dysplasia (23%) and of these 12 had further procedures.

Our management differed from that of Koizumi et al since we always divided the psoas tendon as described by Ludloff. We immobilised the patient in 90° of flexion and 60° of abduction rather than in the ‘frog position’. We never used the Pavlik harness after the age of nine months. Our evaluation of AVN was by Salter’s criteria rather than those of Kalamchi and MacEwen in that we included the mild cases of AVN which Koizumi et al discarded as Kalamchi type I.

Our follow-up was shorter, but of the 25 patients followed for more than four years, three had further surgery and another is dysplastic but is under observation. We have not seen the development of Kalamchi and MacEwen type-II changes in any of our patients at a late stage, only as a development of AVN recognised in the first two years after the operation.

The mean age at operation was much younger in our series (7.2 months; 1 to 23). We recognise, as described by Koizumi et al, that the results are better in younger children. Of the ten hips operated on at ten months of age or younger, eight were satisfactory and two unsatisfactory. In our series of the 60 hips operated on at ten months of age or younger there were six with AVN and six with acetabular dysplasia, with three of these having had further surgery, whereas of the 23 hips in patients aged 11 months or older, six had AVN and 13 had acetabular dysplasia, of which nine had had further procedures.

We therefore feel that medial open reduction is a worthwhile procedure in the child under ten months of age. Over the age of ten months we are concerned about the complications of the procedure. To label the operation as unsatisfactory, however, assumes that there is a safe alternative without risk of complica-
tions. Our comparative studies on operations on 524 hips, as yet unpublished, suggest that in the child over ten months of age the risks of AVN, acetabular dysplasia and the need for further surgery are as great, or greater, in children treated by closed reduction or open reduction by the anterolateral approach when compared with those treated by medial open reduction.

N. S. BROUGHTON, FRCS, FRCS Ed, FRACS
H. K. GRAHAM, MD, FRCS Ed, FRACS
G. R. NATTRASS, FRCS C, FRACS
I. P. TORODE, DABOS, FRCS C, FRACS
P. D. MARSHALL, FRCS Orth
M. O’SULLIVAN, FRACS
Royal Children’s Hospital
Melbourne, Australia.


Authors’ reply:

Sir,

We thank Dr Broughton and his colleagues for their response to our paper.

The presence of Kalamchi type-II necrosis tends to be clear by ten years of age, and therefore it is difficult for us to recognise it with less than ten years of follow-up. Radiological deterioration occurred after ten years of age in the unacceptable hips, especially Kalamchi type-II cases. We consider therefore that we must follow up the children until skeletal maturity.

We do not agree that the results of medial open reduction are better in younger children, especially those under ten months of age, because in our series of ten hips operated on at ten months of age or younger, three were satisfactory but seven were unsatisfactory. We now consider that we need not perform open reduction in such young children because we are able to treat them by closed reduction.

In older children, the radiological results of patients who were treated by closed reduction were better than those managed by open reduction at long-term follow-up.

W. KOIZUMI, MD
H. MORIYA, MD
K. TSUCHIYA, MD
T. TAKEUCHI, MD
M. KAMEGAYA, MD
T. AKITA, MD
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Chiba City, Japan.

BRACHIAL PLEXUS INJURIES

Sir,

We read with interest the Instructional Course Lecture entitled ‘Brachial plexus injuries’ by Birch1 in the November 1996 issue.

Unfortunately, in the special investigations section the information provided on electrodiagnostic testing is incomplete and may be misleading.

Studies on sensory conduction should be done shortly after injury to the brachial plexus. A normal sensory evoked potential obtained from an anaesthetic finger indicates that the lesion is preganglionic. If this response is missing the injury has involved the dorsal root ganglion, the nerve distal to the ganglion, or both. There is no need to wait for three weeks to perform this non-invasive study. If the lesion is preganglionic the peripheral nerve fibres remain intact and sensory studies will continue to be normal throughout. In a patient with a lesion at the dorsal root ganglion or distal to it peripheral conduction starts decreasing precipitously within a week after injury.

EMG changes in the form of fibrillation and positive sharp waves depend on Wallerian degeneration and appear somewhat later. Paraspinal denervation may appear within 14 days after injury and will attest to proximal, i.e. root, involvement. Denervation in the musculature of the upper limb may be apparent three weeks after injury.

We recommend that orthopaedic surgeons familiarise themselves with electrodiagnostic studies and peripheral nerve physiology and take advantage of this relatively non-invasive diagnostic tool.

A. FAST, MD
M. A. THOMAS, MD
Montefiore Medical Centre
New York, USA.


Author’s reply:

Sir,

I thank Drs Fast and Thomas for their comments, but cannot agree.

I think that our article makes plain the value of neurophysiological studies: the methods which we now use were developed and applied by our colleagues on the principles laid down by Bonney and Gilliatt.1 This foundation of preoperative investigation was modified by the observations of Dawson and Scott3 on the recording of cortical sensory evoked potentials and taken forward by Landi, Copeland, Wynn Parry and Jones.2 This body of work has been the mainstay of diagnosis in many hundreds of our cases.

Despite this, there are still problems: a surgeon relying on neurophysiological studies may be misled in a number of ways. There may be confusion related to: loss of conduction in preganglionic injury from extensive damage to the dorsal root ganglion and spinal nerve; loss of conduction from associated vascular lesions without interruption of the nerve; the inability to distinguish between axonotmesis and neurotmesis; and the limited ability to quantitate the extent of injury by pre- or intraoperative studies. We have seen conduction persisting for up to seven days after rupture; in one case such conduction was noted at ten days.

Bonney introduced a policy of urgent exploration and repair over 20 years ago and showed that repair of the artery and nerves at urgent exploration gave the best chance of functional recovery. It should be clear that preoperative neurophysiological investigations can contribute little to diagnosis when intervention occurs within days of injury.

Future developments should seek greater precision. This applies to the recognition of the level of the intradural lesion, whether this be central or peripheral to the transitional zone; to the recognition of selective injury to the central or dorsal root; and in the quantitation of the extent of damage.

As Dr Fast points out, it is axiomatic that surgeons engaged in this work should be familiar with neurophysiological studies, but they must also be aware of the considerable pitfalls.

R. BIRCH, FRCS
Royal National Orthopaedic Hospital
London, UK.


**SIMPLE BONE CYSTS TREATED BY INJECTION OF AUTOLOGOUS BONE MARROW**

Sir,

I read with interest the article in the November 1996 issue on simple bone cysts treated by injection of autologous bone marrow by Lokiec et al because the authors attributed their success entirely to the alleged osteogenesis from the injected cells. They ignore the possibility that, in their technique, the trauma to the cyst wall may play an important, if not crucial, role in the subsidence of the lesion. This possibility, which has been mentioned in the literature at least twice, cannot be dismissed. The authors’ technique, including, as it does, the use of a cannulated needle and a trocar, with “multiple perforations through the cyst wall” and an instruction to “break all the intralesional septa” is not “almost atraumatic”. As has been suggested, it may instigate formation of new vascular channels, previously impeded, which will allow continuous drainage of the cyst.

If this is the explanation of the success of their technique, the relevance of the autologous precursor cells is questionable as has been that of the steroids now being used locally. At least neither is particularly harmful.

J. COHEN, MD
Franciscan Children’s Hospital
Boston, USA.


Sir,

We read with interest the article by Lokiec et al in the November 1996 issue concerning the treatment of simple bone cysts by percutaneous autologous marrow grafting. The aetiology of simple bone cysts (SBC) is unknown. It has been suggested that they may occur after trauma due to failure of absorption of an intraosseous haematoma. Based on the electron-microscopic analysis of cyst membranes Ekkernkamp, Muhr and Lies and Chigira et al believe that the cysts represent an intraosseous synovial cyst. High levels of oxygen scavengers have been isolated in cyst fluid and implicated in the associated bone destruction.

Others believe that the cyst represents the inability of the interstitial fluid to escape from the bone due to blockage of the venous drainage system. Ekkernkamp, Muhr and Lies and Chigira et al have directed treatment toward decreasing the intraosseous pressure, successfully employing decompression by a cannulated screw or multiple cortical perforation.

The operative technique used by Lokiec et al also included “disrupting the lining membrane and making multiple perforations through the cyst wall”. We think that it is likely that the healing achieved by Lokiec et al is influenced by or is the result of the multiple perforations made in the wall of the cyst.

H. W. B. SCHREUDER, MD
R. P. H. VETH, MD
Academic Hospital
Nijmegen, The Netherlands.


**Author’s reply:**

Sir,

We appreciate very much the interesting comments and points raised by Dr Cohen and Drs Schreuder and Veth. Both letters address a similar point regarding the possible effect of multiple perforations through the cyst wall. We thank them for allowing us to explain again the technique in order to prevent misunderstandings. The cortex of the cyst is perforated only once with a thin trocar. After evacuation of the fluid by aspiration, the same needle is used to disrupt the lining membrane in the periphery of the cyst as well as to break all intralesional septa in a multilocular lesion. From the same entry point, with the same needle, we make multiple perforations through the distal cyst wall only into the medullary cavity and never in the periphery of the cyst. Multiple perforations ensure communication of the cyst cavity with the medullary space.

We have used the same method in patients who were treated in the past by steroid injections. Our results did not differ from those published in the literature, with only 50% of cysts showing complete healing while 50% of patients required more than one injection.

Our experience, compared with the injection of steroid, convinces us that our success can be directly attributed to the activity of the osteoprogenitor cells in the marrow stroma.

S. WIENFROUB, MD
Dana Children’s Hospital
Tel-Aviv, Israel.

**CARE OF THE POLYTRAUMATISED PATIENT**

Sir,

We found the review by Tscherne and Negel in the September 1996 issue most informative.
We would, however, appreciate clarification of the phrasing of the authors' instructions regarding early diagnostic procedures including "a lateral film of the head and cervical spine under traction". We recommend that films are obtained as recommended by the American College of Surgeons and supported by the ATLS Working Party for the Royal College of Surgeons of England.\footnote{ATLS Student Manual, 5th Edition. Roentgenographic studies, 336.}

In order to demonstrate adequately all seven cervical and the first thoracic vertebrae they recommend that "The patient’s shoulders are routinely pulled down when obtaining the lateral c-spine film".\footnote{Tscherne H, Negel G. Care of the polytraumatised patient. J Bone Joint Surg [Br] 1996;78-B:840-52.} "If all seven cervical vertebrae are not visualised on the lateral roentgenogram, a lateral swimmer’s view of the lower cervical and upper thoracic area may be obtained".\footnote{ATLS Student Manual, 5th Edition. Roentgenographic studies, 336.}

We feel that this emphasises that the traction is directed through the patient’s arms rather than through the spinal axis, which might be implied from the description in the review.

A. M. COLLIER, FRCS Ed
P. CAMPBELL, FRCS, FRCS Orth
York District Hospital
York, UK.


Author’s reply:

Sir,
We apologise for this unclear phrasing concerning the radiological visualisation of all seven cervical and the first thoracic vertebrae. Our recommendation is similar to the ATLS guideline in that the shoulders are routinely pulled down in order to obtain a lateral view of all the cervical vertebrae. In those cases in which this is not possible a lateral swimmer’s view of the lower cervical and upper thoracic area should be performed.

H. TSCHERNE, MD
Hannover Medical School
Hannover, Germany.

SURVIVAL ANALYSIS OF JOINT REPLACEMENTS

Sir,
In their review entitled ‘Survival analysis of joint replacements’ published in the September 1993 issue Murray, Carr and Bulstrode stress the need for the inclusion of confidence limits for the cumulative probability of survival. This is to be applauded. They follow with an erudite analysis of the commonly used methods and come to the undeniable conclusion that because the failure of joint replacements is non-linear and includes small numbers of subjects at the end of the study periods, a special form of variance analysis must be utilised. Appropriately, they quote Rothman’s equation as the method of choice for all such confidence limits in joint replacement survival studies. It is therefore surprising that the formula presented is not the Rothman equation, but a poorly transcribed and corrupted copy. This gives wildly erroneous confidence limits which do not match those presented on the demonstration figure.

The formula quoted is:

$$P^* = \frac{NP}{(N + Z^2)} + \frac{Z^2}{2N} \pm Z \sqrt{\frac{P(1-P)}{N} + \frac{Z^2}{4N}}$$

where \(N\) = effective number at risk, \(Z\) = the standard deviation constant and \(P\) = the cumulative probability.

Reference to the true Rothman formula:

$$P^* = \frac{N}{(N + Z^2)} \left[ \frac{P + \frac{Z^2}{2N} \pm Z \sqrt{\frac{P(1-P)}{N} + \frac{Z^2}{4N}}} \right]$$

shows that it can similarly be simplified to \(A x (B+C\pm D)\). In this instance, however, the values of \(C\) and \(D\) are appropriately significant to the eventual confidence limits.

It is unfortunate that an equation so critical to the argument of their paper has received such scant attention that this glaring error has been missed. One wonders how many authors have struggled to utilise the formula and in the end adopted a less appropriate measurement of variance.

R. D. FERDINAND, FRCS
I. M. PINDER, FRCS
Freeman Hospital
Newcastle-upon-Tyne, UK.


Author’s reply:

Sir,
We are grateful to Messrs Ferdinand and Pinder who have correctly identified that Rothman’s equation is inaccurately transcribed in our paper. They include the correct version in their letter. This is unfortunate as it may have made it difficult for people to calculate confidence limits. Hopefully, to minimise problems caused by this error, it will be possible to correct the formula on the CD-ROM version of the Journal.

We are pleased that Ferdinand and Pinder agree that confidence intervals should be calculated in survival analysis of joint replacements, and that the Rothman equation is one of the methods of choice if the number of failures is low.

D. MURRAY, FRCS
Nuffield Orthopaedic Centre
Oxford, UK.

THROMBOPROPHYLAXIS AND DEATH AFTER TOTAL HIP REPLACEMENT

Sir,
I write concerning the paper in the November 1996 issue entitled ‘Thromboprophylaxis and death after total hip replacement’ by Murray, Britton and Bulstrode.\footnote{Murray DW, Britton AJ, Bulstrode C. Thromboprophylaxis and death after total hip replacement. J Bone Joint Surg [Br] 1996;78-B:557-60.} Although meta-analysis of such varied trials may be misleading I feel that this excellent article is a welcome addition to the literature providing evidence that our perception of the efficacy of thromboprophylaxis may be incorrect and that fatal pulmonary embolism may not be affected by anticoagulant prophylaxis.

Morbidity from DVT, however, is not addressed in this paper and the issue of postphlebitic syndrome (PPS) is dismissed in a single sentence. There are now four published studies on the
incidence of PPS after total hip replacement. Francis et al\(^2\) studied
hip and knee arthroplasty and showed that 58% (11/19) of patients
with asymptomatic thrombi developed symptomatic venous
insufficiency, then we must question the use of routine anticoagulant
prophylaxis. This is an important issue, because if we
assessing prophylaxis and mortality from thromboembolism. Sim-
ilarly, the association of DVT with morbidity requires further
study and clarification. This is an important issue, because if we
are unable to influence the rate of fatal PE or of late venous
insufficiency, then we must question the use of anticoagulant
prophylaxis at all.

M. A. McNALLY, MD, FRCS Orth
Nuffield Department of Orthopaedic Surgery
Oxford, UK.

1. Murray DW, Britton AR, Bulstrode CJK. Thromboprophylaxis and
863-70.

2. Francis CW, Ricotta JJ, Evarts CM, Marder VJ. Long-term clinical
observations and venous functional abnormalities after asymptomatic
venous thrombosis following total hip and knee arthroplasty. Clin

3. McNally MA, McAlinden MG, O’Connell BM, Mollan RAB.
Postphlebitic syndrome after hip arthroplasty: 43 patients followed at

arthroplasty predispose to chronic venous insufficiency? J Arthro-

5. Skinner JA, Zahn H, Porteous MJL, Thomas EM, Kakkar VV.
Does DVT matter in hip surgery? Venous functional sequelae at five

Author’s reply:

Sir,

Since our paper concerned mortality we did not discuss morbidity
in depth. McNally presents evidence that postphlebitic syndrome
(PPS) is important after THR. No studies, however, have yet
shown that prophylaxis affects PPS after THR. Like McNally, we
continue to question the use of routine anticoagulant
prophylaxis.

D. MURRAY, FRCS
Nuffield Department of Orthopaedic Surgery
Oxford, UK.

1. Murray DW, Britton AR, Bulstrode CJK. Thromboprophylaxis and
863-70.

2. Dunsmuir RA, Allan DB, Davidson AG. Early postoperative mortal-
ity following primary total hip replacement J R Coll Surg Edinb 1996;
41:185-91.

Author’s reply:

Sir,

Port and Stothard have made important points. There is no good
evidence in terms of morbidity that the benefit of prophylaxis
outweighs the risks. One of the conclusions in our paper is that
randomised control trials are needed to study the effect of prophy-
laxis on morbidity.

The interpretation of mortality data after THR is difficult. The
figures at three months may overestimate the number of deaths
caused by THR, partly because a proportion of patients would
have died anyway during that period and also because surgery
may precipitate a death that would otherwise have normally occurred later in the year. This tends to reinforce our view that death after THR is not such a great problem as is generally perceived.

D. MURRAY, FRCS
Nuffield Orthopaedic Centre
Oxford, UK.

Sir,
We read with interest the paper in the November 1996 issue entitled ‘Thromboprophylaxis and death after total hip replacement’ by Murray, Britton and Bulstrode.

We believe that the recommendation that patients having a total hip replacement should no longer receive pharmacological thromboprophylaxis may be ill-considered. The authors state that “there is not enough evidence in the literature to conclude that any form of pharmacological thromboprophylaxis decreases the death rate after total hip replacement”.

In spite of the large number of patients reviewed, however, the conclusion is based on quite a small number of deaths and an even smaller number of cases of fatal pulmonary embolism (FPE). Hence, as demonstrated by the width of their confidence intervals, the estimates of death rates and relative risks are not very precise. Furthermore, the pattern of observed FPEs suggests a substantial protective effect from both heparin and warfarin, although this is not statistically significant. The results are not even at variance with a fivefold reduction in FPE. In addition, the high overall death rates for patients on heparin and warfarin relative to patients without prophylaxis indicate confounding by indication, i.e., it was more likely for patients with a poor prognosis to receive prophylaxis than it was for patients with a good prognosis. This would lead to unduly high death rates in patients on heparin and warfarin and unduly low death rates in patients without prophylaxis.

We appreciate that FPE occurs rarely, but hip replacements are performed with increasing frequency and there is no reason that recommendations on the usage of thromboprophylaxis should be based on anything other than a fair assessment of its advantages and disadvantages.

J. RANSTAM, PhD
Eredenen Medical Statistics
Lund, Sweden.
B. A. SWIERSTRA, MD, PhD
University Hospital
Rotterdam, The Netherlands.


Author’s reply:

Sir,
There is circumstantial evidence from meta-analysis in all fields of medicine and surgery to suggest that thromboprophylaxis should decrease the fatal PE rate after total hip replacement. Our paper shows that there is not enough information in the literature on total hip replacement to either support or refute this. If total hip replacement was a ‘high-risk’ procedure with a rate of fatal PE between 1% and 10% we would agree that routine prophylaxis was indicated. We have clearly shown, however, that this is not so and that the rate is an order of magnitude lower. The potential benefit of prophylaxis is small and we are concerned that it may not outweigh the risks, which include deaths related to the prophylaxis. We have analysed the overall death rate and have concluded that “there is not enough evidence in the literature to conclude that any form of pharmacological thromboprophylaxis decreases the death rate after THR”. It is not known whether routine prophylaxis increases or decreases the overall death rate or has no effect at all. Until more information is available we feel that guidelines recommending routine prophylaxis are not justified.

D. MURRAY, FRCS
Nuffield Orthopaedic Centre
Oxford, UK.

WHICH PRIMARY TOTAL HIP REPLACEMENT

Sir,
In preparing a review of my experience with the PCA ‘E’ series hip, I became aware of a significant error in the article ‘Which primary total hip replacement’ by Murray, Carr, and Bulstrode published in the July 1995 issue. Table I refers to a 57% eight-year failure rate with the PCA ‘E’ series hip. Both the articles by Malchau, Herberts and Ahnfelt and Owen et al which are quoted are reports on the original PCA prosthesis. The PCA ‘E’ series has a femoral stem with considerable modifications in dimension. It was only introduced clinically in 1989. With a minimum follow-up of more than five years in 114 cases, I have experienced only two failures (1.8%), compared with the 57% failure rate erroneously reported in this article.

D. S. HUNGERFORD, MD
Johns Hopkins University
Baltimore, USA.


Author’s reply:

Sir,
Professor Hungerford has pointed out that the PCA ‘E’ series was introduced in 1989 and not 1983 which was when the PCA was introduced. As a result neither the publications of Malchau et al nor Owen et al apply to the PCA ‘E’ series. We obtained information about implants from the manufacturers and also asked them to check our data to try to prevent errors such as this. Professor Hungerford suggests that the modifications to the dimensions introduced with the ‘E’ series will result in improved results. This highlights the point made in our paper, that if the design is modified it should be considered to be a new implant and its name changed.

D. MURRAY, FRCS
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