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.

RADIO-OPAQUE AGENTS IN BONE CEMENT INCREASE RESORPTION

Sir,

I read with interest the article in the January 1997 issue by Sabokbar et al entitled ‘Radio-opaque agents in bone cement increase bone resorption.’ To my surprise the authors conclude that “radio-opaque agents are not essential in assessing the state of implants”. Even if zirconium dioxide and barium sulphate do contribute to clinical osteolysis by the entrapment of cement particles in the bearing causing increased polyethylene wear, this is certainly not the only mechanism behind osteolysis.

It would be wrong to cease to use the available radio-opaque agents. Gross migration and marked osteolytic lesions will be apparent when plain ‘bone cement’ has been used, but tiny lesions, slowly increasing lucencies and slight implant migration, all signs of pending failure, may not be seen. It is unrealistic to rely on serial radiographs which may be of poor quality and are often missing.

Radio-opaque bone cement is important to control the quality of surgery. While awaiting the development of safer radio-opaque agents we must continue to use the existing products.

˚A. S. CARLSSON, MD
Malm¨o University Hospital
Malm¨o, Sweden.


Author’s reply:

Sir,

We agree with Dr Carlsson that radio-opaque agents are useful for assessing the state of implants but we do not feel that they are essential. There is now evidence to suggest that these agents are harmful; they probably contribute to third-body wear and to osteolysis. Consideration has to be given as to whether they should be used and, before firm guidelines can be issued, clinical studies are needed to determine if their use results in an increased number of implant failures. In the meantime it is necessary to develop better radio-opaque agents and to determine if it is possible to decrease the dose of those in current use in order to minimise harm but keep the advantages.

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

THROMBOPROPHYLAXIS AND DEATH AFTER TOTAL HIP REPLACEMENT

Sir,

We agree with the conclusion of Murray et al in their paper in the November 1996 issue entitled ‘Thromboprophylaxis and death after total hip replacement’ that the earlier reports of a death rate of 2% to 3% from pulmonary embolism (PE) after total hip replacement (THR) are too high, but do not accept that their meta-analysis has shown that prophylaxis is unnecessary.

They point out that “the studies were so varied in content and quality that the results of our analysis must be interpreted with some caution.” The technique of meta-analysis works best when comparing randomised clinical trials and even then the results may change dramatically depending on which studies are included. They refer to papers published from 1968 to 1973 to dismiss their own estimate of a possible slight benefit to prophylaxis, although they have already argued that the death rate from PE, and other causes, after surgery has decreased since then.

Their presumed current death rate of between 0.07% and 1.6% ignores a recent paper which examined 1162 THRs for six months after operation. These procedures were performed between 1990 and 1991. Most of the patients received no prophylaxis, although all ‘high-risk’ patients were treated, and the overall death rate from PE was 0.34%, with three of the deaths occurring in hospital. This contrasts with the most recent analysis of the UCLA patients’ receiving warfarin in which there was only one death from PE (presumed but not confirmed) out of 1099 THRs performed between 1987 and 1993 (0.09%). In the total UCLA series in which warfarin has been used since 1970 there has been only one death in over 4100 operations (0.02%). Balderson et al have also reported no deaths in 1372 patients with a low-dose warfarin regime. These results contrast with the 0.34% in the
TRAUMA MANAGEMENT

Sir,

The editorials of the January 1997 issue by Fulford1 and Court-Brown and McQueen2 highlighted the main problems in organising trauma care. How did we come to this situation and what are the solutions?

The principles practised at the Birmingham Accident Hospital in the UK are the continuity, generality and totality of trauma care with very early involvement of consultant surgeons. These principles are simple and achievable, but become impossible if there is 'traumaphobia' when inexperienced and unsupervised junior doctors are put in charge of critically injured patients who often have involvement of multiply interacting systems which require rapid resuscitation, surgical interaction and critical care. Resuscitation is now left to accident and emergency physicians who do not perform surgical treatment or critical care. ATLS training is now required by any applicant for higher surgical training but orthopaedic surgeons usually do not participate in resuscitation.

We believe that subspecialisation is the main culprit in the degeneration of surgical teaching. Young trainees believe that surgery involves muscle, speed and physical dexterity, but the true requirements are judgement and pre-, intra- and postoperative care. Sadly these aspects of surgery are not appropriately taught. The traumaophobias of orthopaedic surgeons stems mainly from difficulty in making immediate decisions with limited information.

Only 8% of orthopaedic surgeons have expressed specialist interest in trauma in the UK. It is thus clear that if we decide that larger central hospitals should employ full-time traumatologists, there are not enough suitably trained surgeons to appoint to these posts. Primary care in trauma is extremely important; even in the hands of very experienced surgeons reconstruction often does not yield a satisfactory outcome. If we continue to allow future surgeons to be trained by traumaphobes, nominally on call but not involved in the resuscitation, surgical and critical care of trauma victims, they will simply create their replicas and trauma care will not improve.

Trauma care must change and this will be implemented by non-medically qualified people. The pressure of managed health care will require generalists and it is very likely that trauma care, including fractures, will be taken over by general surgeons. If we want to make a real effort to improve trauma training and care, those who are interested in it must be put in charge.

E. K. ALPARI, MD, FRCR
University of Birmingham
Birmingham, UK.

J. BULL, CBE, MD, FRCP
Birmingham Accident Hospital
Birmingham, UK.


Author's reply:

Sir,

Thank you for asking me to comment on the letter from Mr Alpar and Dr Bull. I agree that traumaphobia and subspecialisation have
both contributed to the current poor quality of trauma management in the UK. It is interesting to speculate as to why only 8% of orthopaedic surgeons have an interest in trauma, given that more than 50% of the admissions to our units follow injury. Historically, trauma services in the UK have never been well organised and successive reports dealing with the inadequacies of the service have been ignored. The situation is unlikely to improve until the profession accepts that orthopaedic trauma needs specialist consultants and adequate resources.

Subspecialisation has certainly accentuated the problem although its increase can only be to the good of the patient. Subspecialties, however, tend to concentrate on reconstructive surgery and are therefore essentially elective, except for the largest of these groups, trauma, which has over 50% of the total workload but a relatively small percentage of the resources.

Patients are demanding better treatment and, as stated in the editorial, I firmly believe that if the profession does not deal adequately with the problems of our poor trauma system, the government will try to deal with it on our behalf. Based on previous experience, this solution may not be satisfactory to us.

E. A. MELAMED, MD
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Edinburgh, UK.

RECURRENT ROTATIONAL DEFORMITY OF THE FEMUR AFTER STATIC LOCKING OF INTRAMEDULLARY NAILS

Sir,
We wish to comment on the report by Krettek et al1 in the January 1997 issue entitled 'Recurrent rotational deformity of the femur after static locking of intramedullary nails'. They recommend the use of large unslotted nails because of their increased torsional strength. The illustrations of their first case, however, indicate that the entry site for the initial nail was placed too laterally, resulting in malposition with consequent secondary rotational deformity. At revision the second nail entered at the same position and, although initial connection was obtained, the deformity recurred.

We believe that this was partly due to the use of the same entry point and failure to ream the femur generously, which would have allowed a nail of the original size to occupy a slightly different position in the wider cavity.

E. A. MELAMED, MD
C. ZINMAN, MD
Rambam Medical Centre
Haifa, Israel.


Authors' reply:

Sir,
We appreciate the comments made by Drs Melamed and Zinman and agree with their observation that the nail in case 1 was originally malpositioned, resulting in a valgus deformity of the femur. We disagree, however, with their conclusion that the recurrent rotational deformity resulted from the placement of the second nail along the same intramedullary path.

Primary rotational deformities result from locking the bone to the implant in malrotation. Secondary rotational deformities, as reported in our study, occur after the bone has been fixed in the correct position and result from the failure of the bone-implant construct to resist rotation. At the time of the first correction in case 1, the two main fragments could be rotated freely around the inserted nail before locking and even after the nail was statically locked, could still be rotated manually against each other, returning to their corrected position once the rotational force had been released. The problem was not in achieving the correction, but in maintaining it.

The secondary deformity was due to the failure of the bone-implant construct to maintain the corrected position, and not because of the lateral starting point. This was also the reason for the recurrent deformity in case 2 which had a proper starting point, but developed a secondary rotational deformity because a slotted nail of small diameter was inserted. Our cases illustrate the risk of using rotationally weak implants.

Over-reaming and the use of a similar nail of small diameter, as suggested by Drs Melamed and Zinman, will decrease the bone-implant contact, diminish the resistance of the bone-implant construct to rotation and increase the risk of recurrent deformity in these cases.

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M. BLAUTH, MD
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PHYSICAL EXAMINATION IS SUFFICIENT FOR THE DIAGNOSIS OF SPRAINED ANKLES

Sir,
We read with interest the article in the November 1996 issue by van Dijk et al1 entitled 'Physical examination is sufficient for the diagnosis of sprained ankles'. It postulates that delayed physical examination, carried out several days after injury, provides more accurate diagnosis than early assessment.

In their illustration of the anterior drawer test (Fig. 2), the authors state that when a rupture of the anterior talofibular ligament (ATFL) is present, forward movement of the talus produces a dimple in front of the lateral malleolus. They rightly explain the physiology behind the dimple sign to be the negative pressure drawing the skin inwards at the site of ligament rupture.

We wish, however, to draw the authors' attention to our article3 which first described and explained the dimple sign in a ruptured lateral ligament. In our experience, the dimple also appears with combined rupture of the ATFL and calcaneofibular ligaments, not only with rupture of the ATFL as indicated in the legend.

Furthermore, our article clearly states that the dimple sign could not be elicited in ankles examined at seven days or more after injury. This was due to organised haemato ma and repair tissue blocking the communication between the joint and the subcutaneous tissues. The dimple was present in all ankles examined within 48 hours of injury. Therefore, although the passage of time can indicate more clearly the extent of injury based on differences in amount of swelling, haematomata discoulouration and a positive drawer test, it cannot do so on the basis of the dimple sign.

A. J. ARAID, FRCS I, FRCS Ed, FRCS O
J. WONG, MCh, FRCS I
Salamanca Medical Centre
Bahrain.

Authors' reply:

Sir,
We wish to comment on the report by van Dijk et al1 in the January 1997 issue entitled 'Recurrent rotational deformity of the femur after static locking of intramedullary nails'. They recommend the use of large unslotted nails because of their increased torsional strength. The illustrations of their first case, however, indicate that the entry site for the initial nail was placed too laterally, resulting in malposition with consequent secondary rotational deformity. At revision the second nail entered at the same position and, although initial connection was obtained, the deformity recurred.

We believe that this was partly due to the use of the same entry point and failure to ream the femur generously, which would have allowed a nail of the original size to occupy a slightly different position in the wider cavity.

E. A. MELAMED, MD
C. ZINMAN, MD
Rambam Medical Centre
Haifa, Israel.


Authors' reply:

Sir,
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Over-reaming and the use of a similar nail of small diameter, as suggested by Drs Melamed and Zinman, will decrease the bone-implant contact, diminish the resistance of the bone-implant construct to rotation and increase the risk of recurrent deformity in these cases.

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物理检查是诊断踝关节扭伤的足够条件

Sir,
我们有兴趣阅读1996年11月刊载的由van Dijk et al1所发表的文章‘物理检查是诊断踝关节扭伤的足够条件’。文章断言延迟的物理检查（即受伤后数天进行的检查），比早期进行的检查能够提供更准确的诊断。

在他们关于前抽屉试验的插图（图2）中，作者说明当前交叉韧带（ATFL）断裂时，前向移动的距骨将产生踝关节外侧突起。他们正确解释了使皮肤向内凹陷的生理学机制。

然而，我们希望引起作者注意我们的文章3，其中我们首次描述和解释了在断裂的外侧关节囊中出现的突起。在我们的经验中，该突起与外侧韧带和跟腓韧带的联合断裂有关，而不仅限于外侧韧带的断裂，如文中所述。

此外，我们的文章清楚地指出，突起标志不能在踝关节受伤7天或更长时间后被激发。这是由于组织性血肿和修复组织阻塞了关节与皮下组织之间的沟通。突起在所有在48小时内接受检查的踝关节中存在。因此，尽管时间的推移可以更清楚地显示伤害的程度，根据肿胀、血肿、皮肤色变和阳性抽屉试验，它不能在突起的基础上做如此的判断。

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J. WONG, MCh, FRCS I
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After ten days the defect is filled with vascular inflammatory cells. Collagen molecules start to grow along a fibrin mesh and fibroblasts start to grow into the wound at five days. Subsequently, phagocytic activity takes up to six days to occur after injury, which is required when performing the anterior-drawer test. Cell lysis, granulation tissue, and wound healing will occur, however, in only 50% of patients with a lateral ligament rupture. The predictive value of a positive test (PV+) when the anterior-drawer test was performed within 24 hours of injury was 98%, and at the delayed physical examination, five days after injury, it was 94%. The absence of a skin dimple, however, correlated poorly with the absence of lateral ligament rupture; a PV+ of 29% at 24 hours and of 36% at delayed physical examination.

Performing an anterior-drawer test at four to five days after injury will not disturb wound healing, but will give the best available information due to diminished pain and swelling. If a skin dimple does occur during the anterior-drawer test there is a high correlation with rupture of the lateral ligament; a skin dimple only occurs if the examiner can produce an anterior drawer.
Theoretically, from the point of wound healing, swelling and pain will occur, however, in only 50% of patients with a lateral ligament rupture.

The sensitivity and specificity of the dimple phenomenon when the anterior drawer test was performed within 24 hours were 26% and 98%, respectively, and at the delayed physical examination 55% and 87%, respectively.

ARTICULAR Penetration in Garden-I fractures of the HIP

SIR,

I read with interest the article in the March issue entitled ‘Articular penetration is more likely in Garden-I fractures of the hip’ by Hernigou and Besnard.1

As shown by Pauwels,2 fractures of the femoral neck in abduction, which correspond to the Garden-I type of the authors, result in stressing of the fracture site in compression. They should not be operated on since they heal spontaneously.3 After two or three days in bed, the patient can stand and walk with partial weight-bearing.

In my experience such fractures, stressed in compression, usually unite very quickly and crutches may be discarded after three or four weeks. This conservative approach would have avoided the discomfort of useless surgery in all cases in the Garden-I group of the authors and would have prevented the 41% incidence of “technical imperfection” and probably the avascular necrosis. Operative treatment is mandatory only in the exceptional occurrence of secondary displacement of the fragments.

P. MAQUET, MD
Chirurgie de l’Espérance Montignée
Liege, Belgium.


Authors’ reply:

SIR,

We thank Dr Maquet for his comments on our article. We agree with him that Garden-I fractures, commonly referred to as abducted or impacted injuries, may be treated conservatively without operation.

We are not convinced, however, that all such fractures should be treated without operation, since there is a risk of secondary instability and displacement in up to 14% of patients treated without osteosynthesis, particularly in elderly debrided patients. Although conservative treatment of these fractures is possible in young patients, partial weight-bearing with crutches is not suitable for an 80-year-old patient with Parkinson’s disease and a rotator-cuff lesion in one shoulder. There are indications for osteosynthesis of abducted fractures, particularly in the elderly, and our aim was to stress that, contrary to common opinion, these injuries are not so easy to operate on.

P. HERNIGOU, MD
P. BESSARD, MD
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Creteil, France.


Authors’ reply:

SIR,

We thank Dr Correll for his letter. We agree that the problem of valgus deformity can be solved by an angular correction or a medial distal femoral hemi-epiphysodesis. We did not suggest that a Syme’s amputation would solve this problem. He also suggested that lengthening and preservation of the foot should be performed in all unilateral cases of fibular hemimelia and that the Ilizarov method gives excellent results. We wish to challenge this statement.

We do not see how he could reach these conclusions with a minimum follow-up of only five years. To assess patients who had foot surgery at the age of one year and lengthenings at the age of five and ten years and at skeletal maturity, a minimum follow-up of at least 15 years is needed. Numerous long-term studies have shown that children with fibular hemimelia treated by early amputation and prosthetic fitting have athletic and psychological functions closely approaching those of a non-handicapped child of the same age; there are no similar long-term studies in patients who had preservation of the foot and repeated Ilizarov procedures.

Numerous problems such as disuse atrophy, joint stiffness, the effect on the growth plates and the osteopenia of immobilisation which may follow excessive lengthening still remain to be solved before we can conclude that the Ilizarov method is the treatment of choice for these children.

In many of these patients it is not length but the function of the foot and ankle that is the most important aspect. The Ilizarov method can restore length and alignment of the lower limb, but can it restore function and motion to severely deformed and stiff ankle and subtalar joints? The psychological impact of multiple operations, hospital admission and missing schooldays has not been addressed by Dr Correll. Ghoneem et al1 reported that there were no long-term psychological or functional disturbances in their patients but it is not clear how many of the patients had more than one lengthening; the average follow-up was only three years, which may follow extensive lengthening still remain to be solved before we can conclude that the Ilizarov method is the treatment of choice for these children.

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