Guest Editorial

Orthopaedic surgery

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Johan Walters completed his specialist training at Groote Schuur Hospital (GSH) and followed this with a postgraduate fellowship in lower-limb reconstructive surgery at Harlow Wood Hospital in Mansfield (Nottinghamshire), UK, in 1980 - 1981. At the end of 1981 he joined the GSH full-time staff in Orthopaedics. He was appointed to the Pieter Moll and Nuffield Chair of Orthopaedic Surgery at UCT in 1995 as Head of the Department. His current interests are the knee joint, in particular knee replacement surgery, and the development of orthopaedic and medical devices.

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The need to continue learning and actively seek knowledge is never more acutely appreciated than when the consequences of not doing so are unwittingly imposed on our patients. I recently learned this very lesson when I was engaged in a quest for understanding the failure of joint replacement surgery and methods by which the outcomes could be improved.

Part of this research has been the questioning of the traditional wisdom of how joint replacement procedures fail. Excluding the technical reasons, such as dislocation or breakage, which are easily understood, the replacement may fail because of loosening. Broadly speaking, the currently accepted understanding for loosening identifies two common reasons, i.e. immediate or early, by infection with bone lysis and consequent loosening, or late loosening, which is associated with wear of the bearing surfaces – mostly polyethylene on metal. These particles evoke an inflammatory reaction with granuloma formation and bone resorption by the inflammatory mediators, resulting in loosening of the implant.

The latter was termed aseptic loosening because no organisms were isolated on culture, or frozen section at the time of revision surgery failed to reveal significant numbers of inflammatory white cells, and on histological examination large numbers of particles or wear debris with an inflammatory response are demonstrated. However, this chain of events does not take place in all joint replacement procedures. It is difficult to explain why in some there may be wear or even severe wear of the polyethylene but no granulomatous reaction has been evoked, while in others there is virtually no wear detected, yet they develop a periprosthetic granuloma and bone loss and loosening.

Recent advances in the better understanding of how organisms behave and of microbiological techniques such as sonification of the biofilm around the implant and prolonged culture have produced numerous reports showing that in cases labelled aseptic loosening, organisms of low virulence such as *Propionibacterium acnes* (a skin commensal) can be identified in up to 72% of cases. Furthermore, histological examination in these cases is similar to a foreign body reaction rather than the leukocyte infiltration one might expect from the more commonly recognised infections.

If it is true that this actually represents an infection, albeit a very low-grade one, then the possibility exists that all cases which fail by loosening are septic, with organisms of widely ranging virulence, most likely from contamination of the wound site at the time of surgery. This would explain why we observe a range of wound reactions clinically. *Staphylococcus aureus* or *Escherichia coli* are highly virulent organisms and clinical infections by these organisms appear immediately, within days after surgery. *S. epidermidis*, an organism of much lower virulence, may grumble for 3 - 5 years before becoming apparent. Hence, wounds contaminated by skin commensals, which inherently are of even lower virulence, may remain dormant for much longer periods.

Under 'normal' circumstances, the body's innate immunity deals with these invaders or misplaced symbiotic boarders. However, it is possible that in certain cases these organisms are not 'overcome' but they become established in a biofilm on the implant surface. Observations of the postoperative period in patients having undergone total knee replacement demonstrate in about 15% of cases an unusually prolonged period of pain and swelling. To date no long-term study has been undertaken to determine the outcome of the procedures.

Personal observations of infections and loosening of joint replacement arthroplasty have resulted in two postulates. Firstly, the occurrence of an unusually aggressive postoperative wound reaction, where no obvious infection has been identified, may have some bearing on later failure of the joint by infection. Secondly, it is possible that the competence of the immune systems of 'normal' individuals is not constant, and in fact may fluctuate. It is not unreasonable to assume that every wound created surgically, irrespective of the aseptic technique, becomes contaminated by organisms during the surgical procedure. If such a procedure is undertaken during a period of 'questionable immune competence' the individual's ability to overcome these invaders may be compromised.

In an attempt to explore this further a discussion with Professor Novitsky, Haematology, UCT, with regard to one's innate ability to overcome these invaders, resulted in him suggesting that the administration of autologous blood transfusion may play a contributing role.

And here is the lesson I learnt.

Despite all the convoluted thought relating to this issue, newly identified problems relating to blood transfusion had escaped my attention. While the common complications of blood transfusion are well known, to my professional embarrassment over the last 10 years there has been much published literature relating to the down-regulation of the immune system caused by the administration of homologous blood, in journals other than orthopaedic ones.

The clinical consequences of transfusion-induced immune modulation (via T cells called T_{regs}) have been well documented as associated with increased infection in cardiac and oncological surgery, as well as less organ rejection in transplant patients. Hence, administering blood, which is not that uncommon in hip and knee replacement patients, exposes them to an increased risk of infection ... our worst outcome. Somehow, I had become locked in my own orthopaedic cocoon and had not been aware of this fact, even though it has not been proven yet.

Had I read more widely, beyond the scope of my field of expertise, perhaps my practice would have changed earlier, to the benefit of my patients. Hence reading, in particular beyond the comfort zone of common practice, is an imperative for all clinical practitioners.