Correspondence

Polylactic acid pins

Sir—We would like to comment on the article "Activation of the complement system and adverse effects of biodegradable pins of polylactic acid (Biofix) in osteochondritis dissecans" by Tegnander et al. (*Acta Orthop Scand* 1994; 65 (4): 472–475). The authors ascaribed the inflammatory postoperative events they observed in patients implanted with poly(L-lactide) pins to complement activation. To prove this point, they carried out an ELISA in vitro assay to determine complement activation by resorbable pins made of poly(L-lactide).

There is a general consensus that complement activation is an essential method for assessing the biocompatibility of implantable and extracorporeal devices. However, for the proper interpretation of the results, care must be taken to designed the experiment adequately. After reading the experimental protocol used by the authors in their experiments, we wish to make the following comments.

In the in vitro experiments, the poly(L-lactide) pins were exposed to plasma, while plasma alone was used as a control. The authors attributed an increase in the C5a des Arg level in the poly(L-lactide)/plasma system to the complement activation by the poly(L-lactide) pins.

This statement, based on the results of only two test groups as used by the authors, is not fully justified.

At least two additional test groups would have to be used to prove the statement made by the authors. Such test groups are necessary to prove the statement made by the authors. They should comprise:

1. Plasma and a test piece having the same shape as the poly(L-lactide) pins used by the authors, but produced from any biomaterial which has been shown not to activate the complement.

2. Plasma and a test piece having the same shape as the poly(L-lactide) pins used by the authors, but produced from any biomaterial which has been shown to activate the complement.

For the additional two test groups, the C5a des Arg level should be measured and compared with the results obtained by the authors concerning the plasma/poly(L-lactide) system, in order to draw sound conclusions.

It is astonishing that in 4 of the 8 plasma control specimens used by the authors the measured levels of the C5a des Arg were in the range of 19–33 ng/mL, values which are generally considered to be the upper limit in a healthy population. It is not clear why these values are relatively high. Hence, it may be assumed that the siliconized surface of the test tubes used by the authors contributes to complement activation.

The presence of a cellular reaction in the synovial fluid might support the authors' suggestion that the complement activation occurred in vivo because of the presence of poly(L-lactide) pins, and was not a consequence of the inadequately designed in vitro test procedure.

Have the authors carried out this examination?

The biocompatibility of this class of polymers is well documented in a number of articles. In consequence, the suggestion made by the authors that the results obtained with poly(L-lactide) "warrant further study on the biocompatibility of this material" does not seem justified.

We believe that the authors' answers to the above comments may help towards a better understanding of the postoperative problems which they observed in their patients.

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Sir—We have noted the critical remarks by Dr. Mainil-Varlet regarding our paper, and should like to make the following comments.

At first glance, the notion of a control group consisting of a non-activator of complement with an identical shape appears logical. However, complement activation will occur in a particular physiochemical micromilieu created at the surface of a particle and, therefore, the selection of an appropriate negative control with an identical surface seems, at best, difficult. The message which can be drawn from our paper is that the presence of poly(L-lactide) pins in a plasma environment leads to an increased generation of C5a, whether this is a direct consequence of the material itself, or an indirect consequence of changes occurring at the material-plasma interface.

We could easily have included known activators of complement (e.g., zymosan or sephadex particles). This would be relevant in quantifying the potential for complement activation of poly(L-lactide) pins compared to other materials, which was not the scope of our short report.

It is not at all surprising that in heparinized plasma incubated at 37 °C for 2 h, the C5a levels may be in the range described in our paper, possibly due to some inherent enzyme activity when blood is withdrawn into syringes or tubes. Similarly, the C5a level in serum is higher than in plasma because of enzymatic activity induced during coagulation. To illustrate this effect in serum, we have found C5a levels in sera incubated at 37 °C for 30 min in the range of 15–70 ng/mL (Bergh et al. 1993), whereas C5a levels in the EDTA-plasma of healthy individuals are in the range of 6.4—18.8 ng/mL (Bergh and Iversen 1992).

Although the total number of reports on the biocompatibility of poly(L-lactide) is large, we believe that complement activation has not been satisfactorily investigated. Taking into account the not inconsiderable number of reports on inflammatory complications in patients, we disagree with Dr. Mainil-Varlet and suggest further studies on the complement activating potential of poly(L-lactide) pins. In particular, such studies should be performed at various stages during polymerization of the material. Moreover, studies of complement-activating potential of material removed during its biological degradation would be relevant.

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Simmonds' test

Sir—I read with interest the review by Professor Gillquist on the book "Sports Injuries, Mechanisms, Prevention, Treatment" by F H Fu and D A Stone.

It seems that the error in the book regarding the correct denomination of the Thompson test for Achilles tendon rupture has not been corrected. In the original book, it is described as "Thomas test", in the review as "Thomsens test".

Given the history of the test, the correct denomination should be Simmonds' test. In the article on the topic of diagnosis of the ruptured Achilles tendon published 5 years before the Thompson papers (Simmonds 1957), Dr. Simmonds described the "calf squeeze test".

Drs. Thompson and Doherty published jointly a paper exclusively dedicated to such a test in 1962 (Thompson and Doherty). Essentially the same article was published by Dr. Thompson on his own in the same year in *Acta Orthop Scand* (Thompson 1962).

To maintain some degree of historical accuracy, I teach my residents to describe the test as "calf squeeze test" and ascribe it to Dr. Simmonds, while telling them about the work by Drs. Thompson and Doherty.

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