EPIPHYSEAL PLATE REPAIR USING FAT INTERPOSITION TO REVERSE PHYSEAL DEFORMITY

An experimental study

Thesis submitted in March 1989 for
the degree of Doctor of Medicine in
the University of Adelaide

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SUMMARY

A predictable method of reversal of physeal (epiphyseal plate) damage at the level of deformity is not currently available.

A reproducible model of a peripheral partial bone bridge to the proximal medial tibial physis was created in lambs at the age of 6 weeks. Three months after the initial procedure, reversal of the bone bridge lesion was achieved by excision of the bone bridge and autologous fat interposition from the ipsilateral knee fat pad.

There were 3 groups of animals with lesions to the physis. At reversal in the first group of animals, C.T. scans determined that the size of defects was 17.2 $\pm$ 2.2% of the total physeal area. For the second group of animals, the size of the defect was 28.0 $\pm$ 6.0%. For the third group of animals, the lesion was 33.7 $\pm$ 7.2%. Using an unpaired Student's *t* test, there was a statistically significant increase in size of lesion between groups ($p < 0.0001$).

With interposition of fat radiological criteria indicated success in the resumption of physeal growth in 85.7% of cases in the first group, 54.5% in the second group, and 50.0% in the third group.

The survival of the fat and its maintaining of position were critical factors to the success of the reversal procedures. For example, in the first series of experiments, success is 100% if two animals unable to satisfy both criteria are eliminated from analysis.

Measurements of the areas of the physeal defects using C.T. images between the initial (reversal) procedure and autopsy at three and six

months showed a decrease in size of the defects. These, however, failed to reach a statistically significant level of change in the size of the defects. This is presumptive evidence of the inability of physeal cartilage to repair.

Histological assessment showed that there was minimal evidence of physeal stimulation or resumption of a normal physis in the defects. A spur of cartilage could be seen to arise centrally from the physis and to point towards the metaphysis. In this region there was loss of normal physeal chondrocyte organization. Under polarized light microscopy the phenotypical expression showed metaplasia of the hyaline cartilage to fibrocartilage. Chondrons of physeal disorganization occurred.

It is postulated that correction of deformity occurs by a non-union effect, which allows the remaining normal physis to function and grow, rather than by a process of interstitial physeal repair.

In the first group of animals where the area of defect was 17.2 $\pm$ 2.2%, the defect may be resected with a 100% success rate. With larger defects, reversal of deformity may be partial or may not occur. Success was determined by the size of the lesion and the absence of bone bridge reformation.

The clinical significance of this study is that peripheral defects of $<$ 17.2% of growth plate area will respond successfully to physeal interpositional surgery.