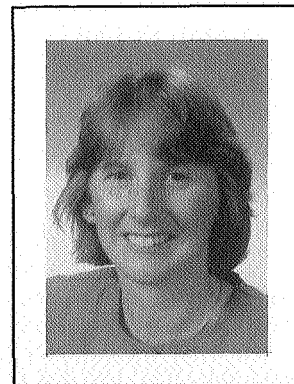


Evidence of acute inflammation in the periodontal ligament subsequent to orthodontic tooth movement in rats

Stephanie M. Cooper M.D.Sc.(Adel)*

Milton R. Sims M.Sc.D.(Boston),Ph.D.(Adel)#

Adelaide, Australia



ABSTRACT: Experimental orthodontic tooth extrusion can result in red cell diapedesis through the PDL vascular wall. Diapedesis is an early sign of acute inflammation. At the ultrastructural level, red cell migration is demonstrated occurring through the endothelial junction of a postcapillary-sized venule. This phenomenon is considered to be indicative of unphysiological tooth loading.

Reference: Aust.Orthod.J.11(2):107-109

MeSH words: PDL/anatomy/microcirculation; microscopy/electron; mice

Key words: Mouse molar PDL, TEM, capillaries, diapedesis

INTRODUCTION

Tissue reactions to orthodontic tooth movement on the tension side have been studied extensively with the light microscope (Reitan 1985). Valuable contributions at the ultrastructural level have recently been provided by Rygh and his co-workers (1976, 1986), Lew (1986), Cooper (1988) and Crowe (1989). The periodontal ligament (PDL) response is determined not by the applied load alone, but by the load per unit area (or pressure), and the duration of load application. Proffit (1986) recommended the use of light orthodontic forces in preference to heavy forces, as the lighter forces were said to be compatible with the survival of cells within the PDL and a remodeling of the tooth socket by a relatively painless 'frontal resorption' of the alveolar bone.

Animal experiments have indicated that increased levels of cyclic-AMP appear after about four hours of sustained pressure (Davidovitch and Shamfield 1975). However, there is scant documentation reporting immediate morphologic or biochemical changes (Yamasaki et al. 1982) in the PDL incident to orthodontic tooth movement.

During an ultrastructural investigation by the authors to determine the presence of tissue channels in tensioned rat molar periodontal ligament, the observations of the present communication were recorded. In the principal study, an ionic tracer, sodium ferrocyanide, based upon the Prussian blue reaction (Chase 1959) was utilised to mark the water-rich tissue channels of the extravascular tissues using tris (ethylenediamine) cobalt III chloride (Browning 1980) as the precipitating ion. A 1.0N (100g) continuous, extrusive load was applied to the right maxillary first molar of eight rats for 30 minutes, as described by Lew et al. (1989), to study the tension effects on tracer distribution. The left maxillary first molar served as the control. The 1.0N force used in the present investigation was comparable to forces used by other authors in their studies on tooth movement of rat molars; Gianelly (1969) utilised tipping forces of 0.5N-1.5N, Azuma (1970) applied tipping forces of 0.8N-2.0N, and Lew (1986) a 1.0N extrusive force.

Each rat was then perfused at 120 mm Hg via the common carotid arteries with the full ionic tracer regime. The maxillae were dissected out and demineralised at 40°C in a solution of 0.1M EDTA in 0.06M cacodylate buffer and 2.5 percent glutaraldehyde. Mid-sagittal cuts of the mesial root isolated the apical foramina. The tissue was prepared for transmission electron microscope (TEM) examination. The four best paired experimental and control blocks, based on midsagittal sectioning through the mesial root canal and apical foramina, were morphometrically analysed in the study. Unstained sections of apical PDL cut in the silver interference range were viewed on a Jeol 100S TEM.

In the course of the investigation, early signs of acute inflammation, or trauma, were observed. In two of the four animals examined, diapedesis of red blood cells through open junctions was occasionally noted on the experimental side of the PDL (Figure 1). There were no

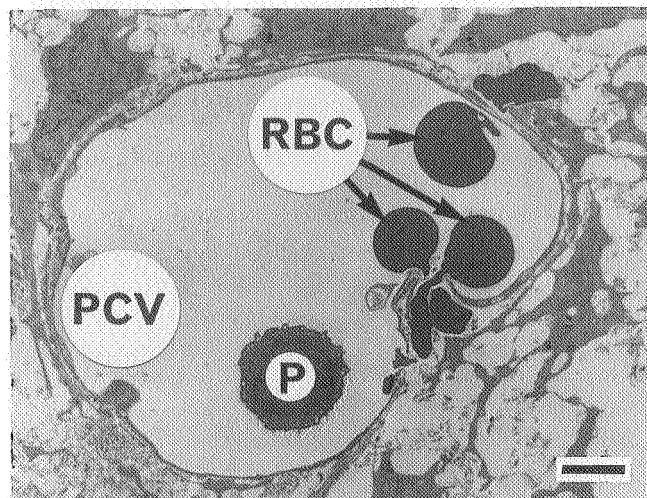


Figure 1. Diapedesis of red blood cells (RBC) through the vascular wall of a postcapillary-sized venule (PCV) on the experimental tension side of apical periodontal ligament. A polymorph (P) is present in the vessel lumen
Magnification x 3,500
Bar = 3 µm

*Postgraduate student, University of Adelaide; now in private practice, Canberra

#Reader in orthodontics, University of Adelaide

obvious signs of trauma observed on the control side. Diapedesis of red blood cells, an early sign of acute inflammation, is believed to be a passive phenomenon resulting from hydrostatic pressure squeezing the cells through a small defect in the vascular endothelium (Robbins, Angell and Kumar 1981). It was felt that this tissue damage was a consequence of the relatively heavy 1.0N extrusive force which resulted in clinically observable extrusion of the rat molars.

Oppenheim (1944) described vascular haemorrhage in tension zones, but failed to mention the duration and magnitude of the force used. Lew (1986) postulated that the 1.0N tensile force used in his study could have injured the PDL and led to an inflammatory response.

Rygh conducted a preliminary TEM investigation of the tension side in the PDL of the rat in 1976. He concluded that the tissue changes seemed to represent active tissue transformation and proliferation within physiological limits. Macapanpan, Weinmann and Brodie (1954), Khouw and Goldhaber (1970) and Rygh (1976) reported a slight dilation of the blood vessels in the tension zones of the PDL.

Furthermore, Rygh et al. (1986) reported the presence of macrophage-like cells seen in the vicinity of the blood vessels of rat molar PDL tensioned with tipping loads of 0.3N and 2-28 days duration. The frequent occurrence of phagocytosed cell remnants indicated that injury is inflicted on the PDL in areas of tension with an acute inflammatory response. Studies of suture expansion in rats by Ten Cate, Freeman and Dickinson (1977) also showed a transient polymorph response followed by a macrophage influx. Subsequently, Ten Cate and Freeman (1981) concluded that the cellular events occurring during suture expansion and orthodontic tooth movement were similar. These authors suggested that orthodontic tooth movement involved tissue damage, inflammation and repair by scar tissue. The process was followed by active and immediate remodelling of the scar to regenerate the PDL.

Rubin (1983) proposed that the reaction of the PDL to force is dependent upon biologically effective units of force, which are a function of force and time. The concept of biological force units may only be valid if the so called 'force' is within the physiological tolerance of the tissue. It is likely that the 1N tensile load applied to the rat maxillary first molar cannot be considered physiological due to the marked clinical extrusion observed.

The clinical significance of the ultrastructural observations of acute trauma in the PDL is that light appliance activation, compatible with cell stimulation and survival (Proffit 1986), should be used in orthodontic tooth movement. Ironically, current orthodontic literature (Engstrom et al. 1988) persists in using the term 'force' in an implied physiological context. A clinical demarcation line between pathological and physiological orthodontic tooth loading has yet to be established for man.

Acknowledgments

This report was based on an investigation supported by a National Health and Medical Research Council Grant No. 880348 and the Australian Society of Orthodontists' Foundation for Research and Education.

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Correspondence to:
Dr M.R. Sims
Department of Dentistry
University of Adelaide
North Terrace
ADELAIDE
South Australia 5000