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Apical limit of root canal instrumentation and obturation, part 2. A histological study

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Summary

The results of an *in vivo* histological study involving apical and periapical tissues following root canal therapy after different observation periods demonstrated the most favourable histological conditions when the instrumentation and obturation remained at or short of the apical constriction. This was the case in the presence of vital or necrotic pulps, also when bacteria had penetrated the foramen and were present in the periapical tissues. When the sealer and/or the gutta-percha was extruded into the periapical tissue, the lateral canals and the apical ramifications, there was always a severe inflammatory reaction including a foreign body reaction despite a clinical absence of pain.

***Keywords*: apical limit, root canal instrumentation/ obturation, tissue reactions.**

Introduction

The first scientific basis for modern clinical Endodontology was established by Davis (1922). He was the first to suggest that careful treatment of the apical tissue was a requirement for success in root canal treatment.

Since then many histological studies, based on apical biopsies including the surrounding periapical tissues, or endodontically treated teeth which were extracted at varying periods of time, confirmed his observation (Hatton *et al.* 1928, Blayney 1929, Nygaard-Østby 1939–44, Laws 1962, Nyborg & Tullin 1965, Engström & Spångberg 1967, Seltzer *et al.* 1968 and 1969). All these studies agreed that, in vital pulp treatment, partial pulpectomy was preferred to total pulp removal. Langeland (1957, 1967, 1976, 1981, 1987 and 1995), Lin *et al.* (1984) described in detail the progression of the pulp necrosis and demonstrated that vital pulp tissue with nerves and vessels remain in the most apical part of the main canal even in the presence of a large periapical lesion. As a logical clinical consequence of these observations, they suggested terminating instrumentation and obturation at the apical constriction, just short of the radiographic apex, even in the presence of a periapical lesion. They also concluded that, in time, all pulp tissue will be involved by necrosis, finally including apical tissue and the tissue contained in the ramifications, and bacteria will establish themselves in the periapical lesion as far as the necrosis occurs (Pascon *et al.* 1987, Oguntebi *et al.* 1982, Lin *et al.* 1996). Even in this extreme situation, the most appropriate level in limiting the endodontic operation is still the apical constriction: the area located inside the root canal (Langeland 1995).

The purpose of the present study was to investigate the histopathological response of the intracanal pulp tissue, of the pulp tissue contained in the lateral canals, the apical ramifications, and of periapical tissues to endodontic procedures when performed short of or beyond the apical constriction, in both vital and necrotic pulp conditions.

Materials and methods

The experimental material consisted of 41 human teeth, a total of 49 roots, obtained from 36 patients (14 males, 22 females) aged 16–65 years. All human subjects who participated in the experimental investigation gave their informed consent after the nature of the procedure and possible discomforts and risks had been fully explained (Table 1).

Ten of these cases (nine teeth) came from a previous investigation (Ricucci *et al.* 1990) consisting of biopsies of the apex and periapical bone following instrumentation and $Ca(OH)_2$ medication (cases 1–10). Nineteen teeth had been exposed to caries and/or operative damage, requiring endodontic treatment for pulpal/periapical involvement (cases 11–29).

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Table 1

Case Number	Patient	Sex	Age■	Tooth/Root	Pathology	Diagnosis	P.a. lesion	Medication	Duration medication (days)	Procedures	Apical level procedures (mm from rad. apex)	Obser-vation period	P.a. lesion at the moment of the biopsy	Clinical/ radio-logical evaluation of the treatment	Biopsy
1	AP	M	18	14/Palatal root	None	Vital	No	$Ca(OH)_2$	7	Instrumentation only	−1.5	7 days	No	--	Extraction
2	AP	M	18	14/Buccal root	None	Vital	No	$Ca(OH)_2$	7	Instrumentation only	−1.5	7 days	No	--	Apical/P.a. biopsy
3	GV	M	48	12	Fracture	Necrotic	Yes	$Ca(OH)_2$	15	Instrumentation only	−1	15 days	Yes	--	Apical/P.a. biopsy
4	RM	F	18	14/Buccal root	Caries	Necrotic	No	$Ca(OH)_2$	35	Instrumentation only	−1	35 days	No	--	Apical/P.a. biopsy
5	MS	F	57	11	Caries	Necrotic	Yes	$Ca(OH)_2$	7	Instrumentation only	−1.5	7 days	Yes	--	Apical P.a. biopsy
6	AIR	M	54	12	Iatrogenesis (prosthetic preparation)	Nectrotic	Yes	$Ca(OH)_2$	15	Instrumentation only	−1.5	15 days	Yes	--	Apical/P.a. biopsy
7	PS	F	55	11	Caries	Vital	Yes	$Ca(OH)_2$	14	Instrumentation only	−1	14 days	Yes	--	Apical/P.a. biopsy
8	PS	F	55	21	Caries	Vital	No	$Ca(OH)_2$	14	Instrumentation only	−1	14 days	No	--	Apical/P.a. biopsy
9	SC	F	34	21	Caries	Vital	No	$Ca(OH)_2$	21	Instrumentation only	−1.5	21 days	No	--	Apical/P.a. biopsy
10	ER	M	19	11	None	Vital	No	$Ca(OH)_2$	82	Instrumentation only	−1.5	82 days	No	--	Apical/P.a. biopsy
11	EA	F	30	11	Caries	Vital	No	Cresatin	7	Instrumentation only	−1.5	7 days	No	--	Apical/P.a. biopsy
12	FR	M	19	12	Iatrogenesis (pulp capping)	Necrotic	Yes	$Ca(OH)_2$	27	Complete endo treatment	−1.5	48 days	Yes	--	Apical/P.a. biopsy
13	PA	M	22	11	Caries	Necrotic	Yes	$Ca(OH)_2$	7	Complete endo treatment	−1.5	48 days	Yes	--	Apical/P.a. biopsy
14	MTS	F	18	21	Iatrogenesis (Pulp capping)	Necrotic	Yes	No	--	Complete endo treatment	−1.5	3 years	No	Successful	Apical/P.a. biopsy
15	MI	F	23	12	Caries	Necrotic	Yes	No	--	Complete endo treatment	Beyond apex	6 years	Yes	Doubtful	Apical/P.a. biopsy
16	SP	F	24	14/Buccal root	Caries	Necrotic	Yes	$Ca(OH)_2$	20	Complete endo treatment	−2	5 months	Yes	Doubtful	Apical/P.a. biopsy
17	SA	F	44	22	Caries	Necrotic	Yes	Cresatin	7	Complete endo treatment	Beyond apex	13 months	Yes	Doubtful	Apical/P.a. biopsy
18	LA	M	37	21	Caries	Vital	No	No	--	Complete endo treatment	Beyond apex	4 months	Yes	Failure	Apical/P.a. biopsy

Table 1—*Continued.*

Case Number	Patient	Sex	Age■	Tooth/Root	Pathology	Diagnosis	P.a. lesion	Medication	Duration medication (days)	Procedures	Apical level procedures (mm from rad. apex)	Observation period	P.a. lesion at the moment of the biopsy	Clinical/radiological evaluation of the treatment	Biopsy
19	EP	M	31	22	Caries	Vital	No	No	--	Complete endo treatment	−1.5	82 days	No	--	Apical/P.a. biopsy
20	EP	M	31	24/Buccal root	Caries	Vital	No	No	--	Complete endo treatment	−1.5	62 days	No	--	Apical/P.a. biopsy
21	FM	M	42	22	Caries	Necrotic	Yes	$Ca(OH)_2$	10	Complete endo treatment	−1.5	18 days	Yes	--	Apical/P.a. biopsy
22	DO	F	23	21	External root resorption	Vital	No	$Ca(OH)_2$	7	Complete endo treatment	−1□	1 year	Yes	Failure	Apical/P.a. biopsy
23	RS	M	39	44	Caries	Necrotic	Yes	$Ca(OH)_2$	7	Complete endo treatment	−1	4 months	Yes	--	Apical/P.a. biopsy
24	GC	F	20	23	Caries	Necrotic	Yes	No	--	Complete endo treatment	−1	1 year	Yes	Failure	Apical/P.a. biopsy
25	GS	M	22	11	Caries	Necrotic	Yes	No	--	Complete endo treatment	−1	4 years	Yes	Failure	Apical/P.a. biopsy
26	MG	F	18	22	Caries	Necrotic	Yes	No	--	Complete endo treatment	−0.5	4 years	Yes	Failure	Apical/P.a. biopsy
27	VV	M	62	21	Caries	Necrotic	Yes	$Ca(OH)_2$	21	Complete endo treatment	−1	40 days	Yes	--	Apical/P.a. biopsy
28	LV	M	65	12	Caries	Necrotic	Yes	$Ca(OH)_2$	14	Complete endo treatment	−1	35 days	Yes	--	Apical/P.a. biopsy
29	SZ	F	21	21	Caries	Necrotic	No	Cresatin	7	Complete endo treatment	−1.5	10 years and 8 months	Yes	Failure	Apical/P.a. biopsy
30	MI	M	25	35	Caries	Vital	No	No	--	Complete endo treatment	−2	6 years	No	Successful	Extraction
31	MF	F	43	12	Caries	Vital	No	No	--	Complete endo treatment	−2	2 years	No	Successful	Extraction
32	AC	F	31	27/mesial root	Caries	Vital	No	Cresatin	7	Complete endo treatment	−1.5	5 years	No	Successful	Extraction
33	AC	F	31	27/palatal root	Caries	Vital	No	Cresatin	7	Complete endo treatment	−2	5 years	No	Successful	Extraction
34	AC	F	31	27/distal root	Caries	Vital	No	Cresatin	7	Complete endo treatment	−2	5 years	No	Successful	Extraction
35	AP	F	47	12	Iatrogenesis (Prosthetic preparation)	Necrotic	Yes	$Ca(OH)_2$	9	Complete endo treatment	−1	3 years	No	Successful	Extraction
36	AMS	F	39	15	Iatrogenesis (Pulp capping)	Necrotic	Yes	$Ca(OH)_2$	17	Complete endo treatment	−1	5 months	No	Successful	Extraction

Table 1—*Continued.*

Case Number	Patient	Sex	Age■	Tooth/Root	Pathology	Diagnosis	P.a. lesion	Medication	Duration medication (days)	Procedures	Apical level procedures (mm from rad. apex)	Observation period	P.a. lesion at the moment of the biopsy	Clinical/radiological evaluation of the treatment	Biopsy
37	FL	F	16	35	Caries	Vital	Yes	No	--	Complete endo treatment	−1.5	3 years and 1 month	No	Successful	Extraction
38	AL	F	26	46/mesial root	Caries	Vital	No	No	--	Complete endo treatment	−1.5	7 years and 3 months	No	Successful	Extraction
39	AL	F	26	46/distal root	Caries	Vital	No	No	--	Complete endo treatment	−1.5	7 years and 3 months	No	Successful	Extraction
40	EP	M	23	45	Caries	Necrotic	Yes	No	--	Complete endo treatment	Beyond apex	6 years and 6 months	No	Doubtful	Extraction
41	GA	F	45	23	Caries	Vital	No	No	--	Complete endo treatment	−2	5 years and 4 months	No	Successful	Extraction
42	AM	F	31	26/mesial root	Caries	Vital	No	$Ca(OH)_2$	7	Complete endo treatment	Beyond apex	3 years and 5 months	No	Successful	Extraction
43	AM	F	31	26/palatal root	Caries	Vital	No	$Ca(OH)_2$	7	Complete endo treatment	−1.5	3 years and 5 months	No	Successful	Extraction
44	AM	F	31	26/distal root	Caries	Vital	No	$Ca(OH)_2$	7	Complete endo treatment	−1	3 years and 5 months	No	Successful	Extraction
45	VV	M	62	46/mesial root	Caries/Perio	Necrotic	Yes	Cresatin	7	Complete endo treatment	−1.5	7 years	No	Successful	Extraction
46	VV	M	62	46/distal root	Caries/Perio	Necrotic	Yes	Cresatin	7	Complete endo treatment	−1.5	7 years	No	Successful	Extraction
47	VV	M	62	36/mesial root	Caries	Necrotic	Yes	$Ca(OH)_2$	7	Complete endo treatment	−1.5	6 years and 7 months	No	Successful	Extraction
48	VV	M	62	36/mesial root	Caries	Necrotic	Yes	$Ca(OH)_2$	7	Complete endo treatment	−0.5	6 years and 7 months	No	Successful	Extraction
49	AP	F	55	33	Caries/Perio	Vital	No	$Ca(OH)_2$	7	Complete endo treatment	−1	1 year	No	Successful	Extraction

■The age of the patient is intended at the moment of the endodontic treatment.
□The obturation was 1 mm short of the radiographic apex. Actually, in the histologic sections it protruded into the periapical tissue from an anticipated foramen.

Endodontic therapy was performed by the same operator, using the same technique. In all cases prophylaxis, local anaesthesia, rubber dam application, disinfection of the field with 30% H_2O_2 followed by 5% tincture of iodine were performed (Möller 1966). During instrumentation copious amounts of 1% NaOCl were used followed by thorough aspiration. The canals were obturated with laterally condensed cold gutta-percha, and a zinc oxide-eugenol sealer (Pulp Canal Sealer; Kerr Manufacturing Co., Romulus, MI, USA).

After observation periods varying from 18 days to 10 years and 8 months, during which recall follow-up radiographs were taken, biopsies of the apex with surrounding periapical tissue were taken. After elevating a flap, a circular trepan bur 6 mm in diameter rotating at a low speed was used, to obtain a cylinder of bone including the root apex. The remaining sectioned surfaces were smoothed and bevelled. Retrograde cavities were prepared and filled with amalgam.

Thirteen of the teeth (20 roots) were scheduled for extraction, for orthodontic or prosthetic reasons, or because of longitudinal fractures, all following endodontic therapy which included 1% NaOCl/$Ca(OH)_2$ irrigation/medication and obturation (cases 30–49) performed five months to seven years and three months previously.

In all cases the medical and dental history were recorded, at least one diagnostic radiograph was taken and an initial diagnosis established on the basis of a combined clinical and radiographic examination.

The teeth were grouped as those with a vital pulp without bacterial colonization in the root canal, and those with a necrotic pulp where bacterial colonization had advanced into the root canal regardless of how far (Langeland *et al.* 1976, Anderson *et al.* 1981, Lin *et al.* 1984, Langeland 1987). The rationale for this classification is that there is a distinct correlation between the clinically observable condition and the histological facts. Clinically it was checked and recorded whether or not there was continuous, blood filled pulp tissue in the canal orifice(s), which constituted a basis for one-visit therapy. According to this classification, governed by microbiological facts regardless of the presence/absence of a periapical lesion, 24 cases were classified as 'cases with vital pulp' and 25 as 'cases with necrotic pulp'. The latter two or more visits were considered mandatory, with $Ca(OH)_2$ used as an intracanal medicament (Table 1).

The specimens were immediately immersed in a 10% neutral buffered formalin solution. Radiographs at right angles to the long axis of the root and photographs were taken of all specimens after fixation. The specimens were shipped in fixative to the University of Connecticut for histological processing and evaluation. Serial sections 5 μm thick were cut and stained alternately with hematoxylin and eosin, Masson's trichrome, or Brown-Brenn techniques. Special care was taken to obtain and locate the sections that included material/tissue contact and the apical foramen(ina) in direct continuation with the periapical tissues.

Results

Observations

In all cases remnants of pulp tissue and dentine chips often intermixed with sealer were found at different levels in the root canals. The accidental presence of pulpal debris and dentine chips was mainly limited to the pulp wound surface (Fig. 1b, c; 5c, d), but in some cases chips and debris were displaced into the apical and periapical tissue.

Cases with procedures limited within the canal

In 43 of the 49 roots the procedures were limited within the apical constriction. In 10 of these roots only instrumentation and $Ca(OH)_2$ medication had been undertaken, and in one case instrumentation followed by Cresatin medication. In three cases of this group which exhibited a periapical radiolucency prior to the treatment (cases 3, 5 and 6), periapical lesions were found histologically, with a dense concentration of both chronic inflammatory (plasma cells, macrophages, mast cells, lymphocytes, foreign body cells) and acute inflammatory cells (neutrophilic leucocytes). In case 7 a vital pulp stump was found with a moderate concentration of inflammatory cells, in continuation with the periapical lesion.

In the remaining seven cases of this group without obturation, in which no periapical radiolucency was present prior to the treatment (cases 1, 2, 4, and 8–11), a vital pulp stump was observed showing differing degrees of chronic inflammation. Chronic inflammatory cells were also present in the periapical tissue in four cases (4, 8, 9 and 11). In three cases (1, 2 and 10) inflammation was confined to the apical pulp stump, in the presence of a healthy periodontal ligament (Fig. 1c).

In the group with root canal obturation, 19 cases were clinically classified as successful (cases 14, 30–39, 41 and 43–49). These showed a vital pulp stump with chronic inflammatory cells. A healthy periodontal ligament was observed in case 14 (Fig. 7c). Fragments of periodontal ligament attached to the extracted roots were found, free of inflammation (in cases 30, 31 and 37) (Fig. 4e; 8c).

Differing degrees of inflammation were present in the apical and periapical tissues (cases 12, 13, 16, 19, 20, 21, 23,

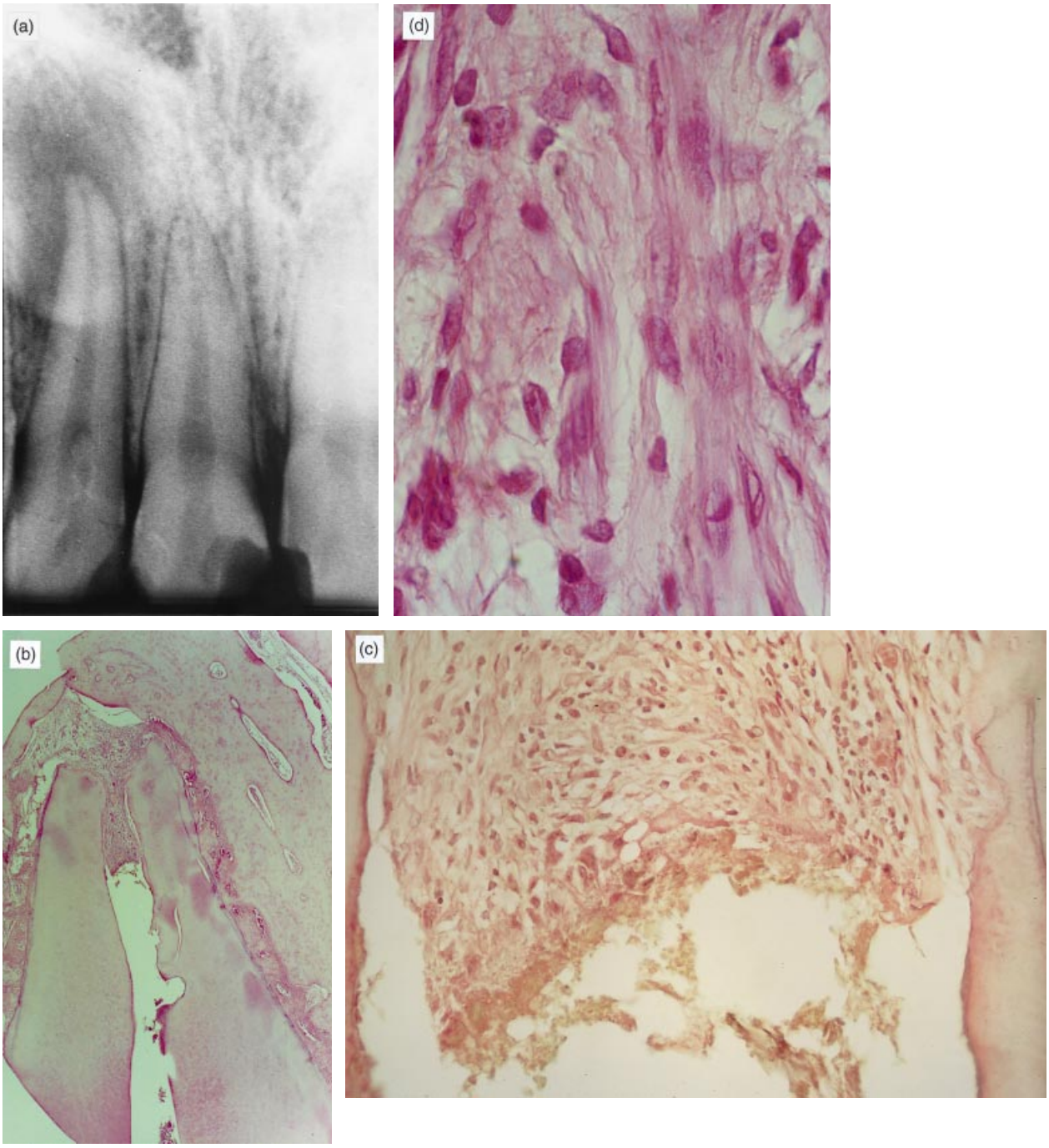

Fig. 1 Case 10 (Table 1). (a) A 19-year-old male. Tooth 11 had two composite restorations with no apparent cement or base mesially. No widening of periodontal/periapical space. Lag time not known. Asymptomatic: the pulp responded within normal limits to sensitivity tests (cold, hot, electric). The canal was instrumented to approximately 1.5 mm from the radiographic apex, up to a size 60 K-file. At the end of the instrumentation the canal was rinsed with sterile saline, dried and filled with $Ca(OH)_2$. After 84 days – during which no post-operative symptoms were present – the canal was reopened, washed, dried and left empty; a sterile cotton pellet was put into the pulp chamber and a temporary filling inserted. A flap was elevated and a biopsy of the apex with periapical tissue taken. (b) Root tip including apical foramen. 1.1 mm of pulp tissue remains short of the apical constriction. Foramen opens beyond the apical constriction. Apart from small irregularities, the canal wall is clean. Minor accumulation of debris on the wound surface. H & E; (× 12). (c) Debris on the wound surface, scattered chronic inflammatory cells below. H & E; × 200. (d) Healthy pulp tissue from around the apical constriction with fibroblasts and a few scattered inflammatory cells. H & E; × 500.

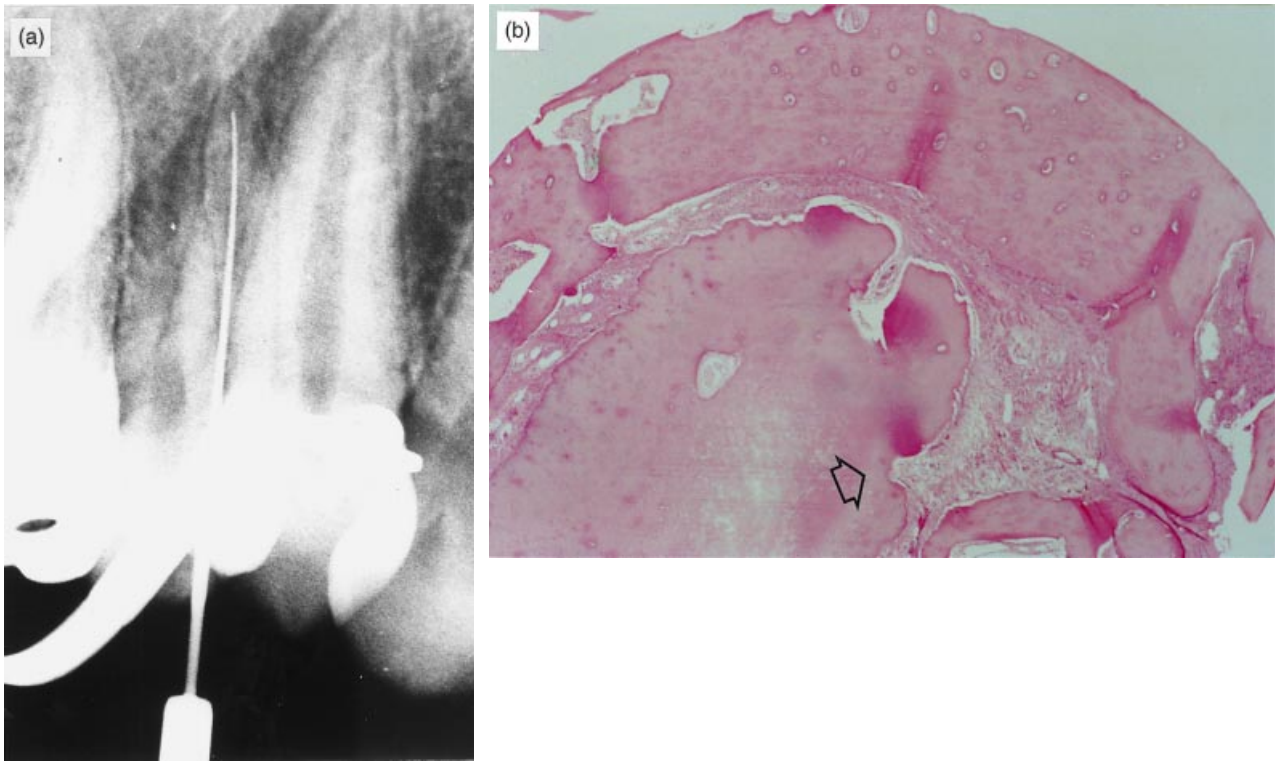

Fig. 2 Case 2 (Table 1). (a) A 18-year-old male. The first upper premolar was scheduled for extraction for orthodontic reasons. It was caries free with a supposedly healthy and uninflamed pulp tissue. Treatment as for case 10. Working length at about 1.5 mm from the apex. $Ca(OH)_2$ mixed with sterile saline used as intracanal dressing. After 1 week a flap was elevated and a biopsy taken, including the bone with the buccal root tip. Immediately after, the tooth was extracted. (b) Buccal root tip with PDL and periapical bone. Main foramen short of the apex. Apical ramification (arrow). There is another lateral canal a short distance away from the main canal. H & E; × 15.

27 and 28) which could not be clinically classified as success or failure because the observation periods were too short.

Calcifications were present in the most apical part of the canal (cases 14, 30, 35, 38 and 39) narrowing the canal lumen but in no case was there total closure. This phenomenon coexisted with an inflamed pulp stump (Fig. 7c, d).

In cases 24–26 and 29 – classified as failures – severe inflammatory reactions were observed in the periapical tissues. In all cases bacteria were identified in the debris present in the apical part of the canal and in adjacent dentinal tubules. In case 24 food debris was noted, which was histologically identified as vegetable material, infected by bacterial colonies in the most apical part of the canal, which had been left exposed to the oral environment for a long period (Fig. 5c, d).

Cases with overfilling

Overfilling was present in six cases (15, 17, 18, 22, 40 and 42). In one of these (case 22), with the obturation seemingly ending short of the apex radiographically, histologically showed termination on the buccal aspect of the root, several millimetres short of the anatomical apex (Fig. 6c, d)).

In all cases severe inflammatory reactions were observed in the periapical tissues. Periapical necrosis and inflammation were present near the extruded sealer (Fig. 3c, d; 6d).

Pain was not present in any of the cases, and in cases 15 and 17 there was a considerable reduction in size of a previous periapical lesion (Fig. 3a, b).

Cases with healed lesions

In eight cases with previous periapical radiolucencies, apparently healed at successive follow-ups, a vital pulp stump was present in the histological sections (cases 14, 35–37 and 45–48) (Fig. 4d, e; 7c, d).

Specimens taken with periapical lesions

In 16 cases periapical biopsies were performed in the presence of radiolucencies. In six of these vital pulp tissue was found coexisting with the periradicular lesion (cases 6, 7, 16, 21, 22 and 28). In the other 10 specimens, necrotic tissue was found in the most apical part of the canal, bordered towards the periodontal ligament by a

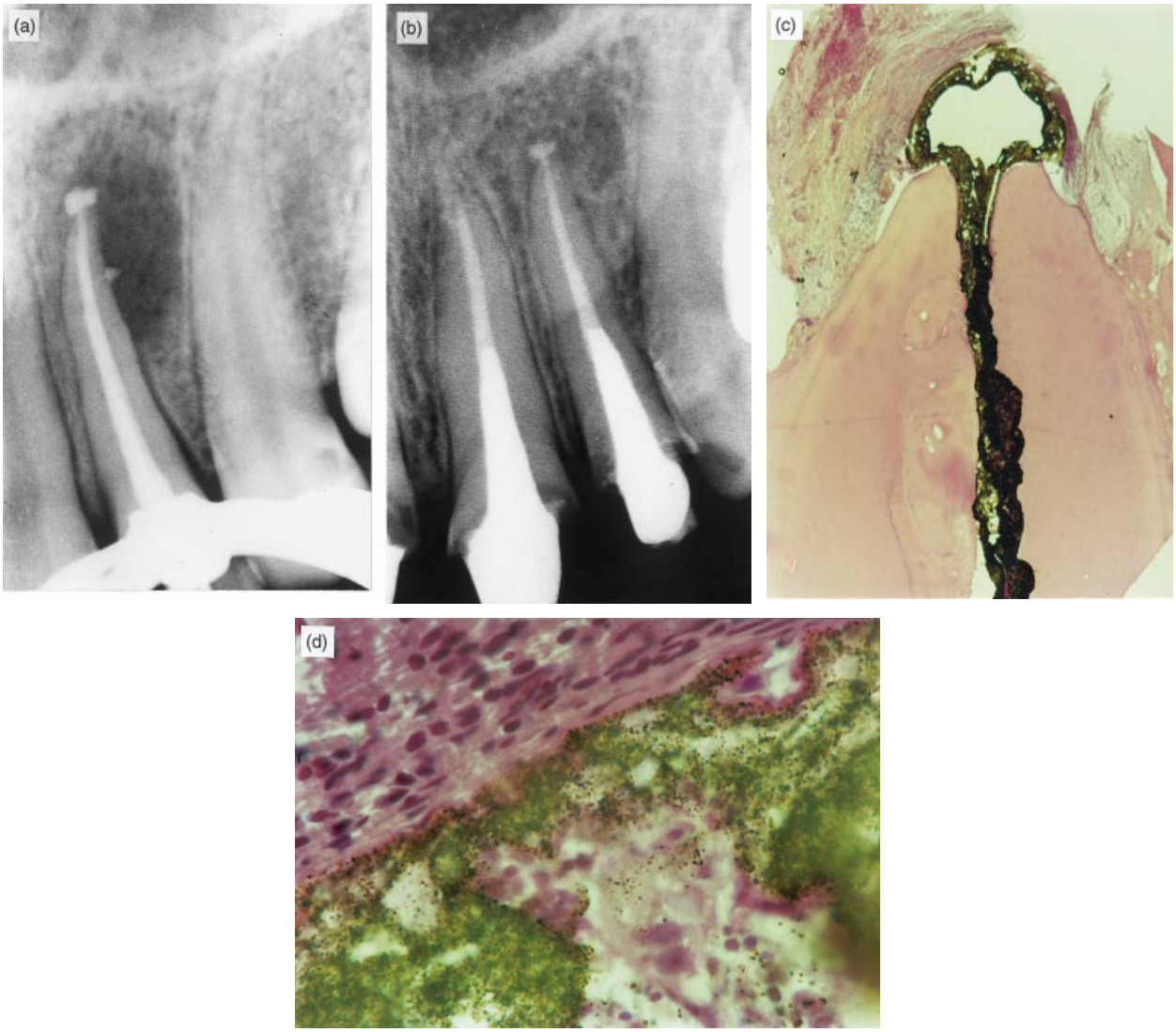

Fig. 3 Case 15 (Table 1). (a) A 22-year-old female. Tooth 22. Spontaneous flare-up with pain, swelling in the soft tissues and an elevated temperature. Incision and drainage undertaken and systemic antibiotics prescribed for 1 week. Returned after 2 weeks without symptoms. Sensitivity tests were negative but the tooth was slightly tender to percussion. The tooth was root canal treated. The post-operative radiograph showed a large periapical lesion and that the canal had been obturated beyond the foramen, into the periapical tissues, including a lateral canal. (b) At a 6-year follow-up no post-operative symptoms were found. There was no spontaneous pain and no pain to percussion or palpation. The radiograph showed regeneration of the periapical bone but a radiolucency remained around the excess material. The lateral canal was no longer distinguishable. (c) A 5.5 mm specimen; root canal with remaining filling material, excess material apically and adjacent inflammatory tissue around apparent empty space. H & E; $\times 12$. (d) From the area of radiolucency around the excess material, dense concentration of sealer particles can be seen bordered by fibrous tissue, with chronic inflammatory cells beyond. Amongst the sealer particles there is disintegrating periapical tissue with remaining lobes of nuclei of neutrophilic leucocytes. H & E; $\times 48$.

dense concentration of neutrophilic leucocytes (cases 3, 5, 12, 13, 23–27 and 29) (Fig. 5c).

Discussion

Despite careful preparation of optimal access and meticulous root canal cleaning and shaping, in all cases remnants of pulp tissue and dentine chips, often intermixed with sealer, were found at different levels of the root canals. The accidental presence of pulpal debris and dentine chips was limited mainly to the pulp wound surface, but in some cases chips and debris were displaced into the apical and periapical tissue. This confirmed previous observations indicating that a certain amount of debris may remain in the canal, regardless of the technique employed (Turek & Langeland 1982, Langeland

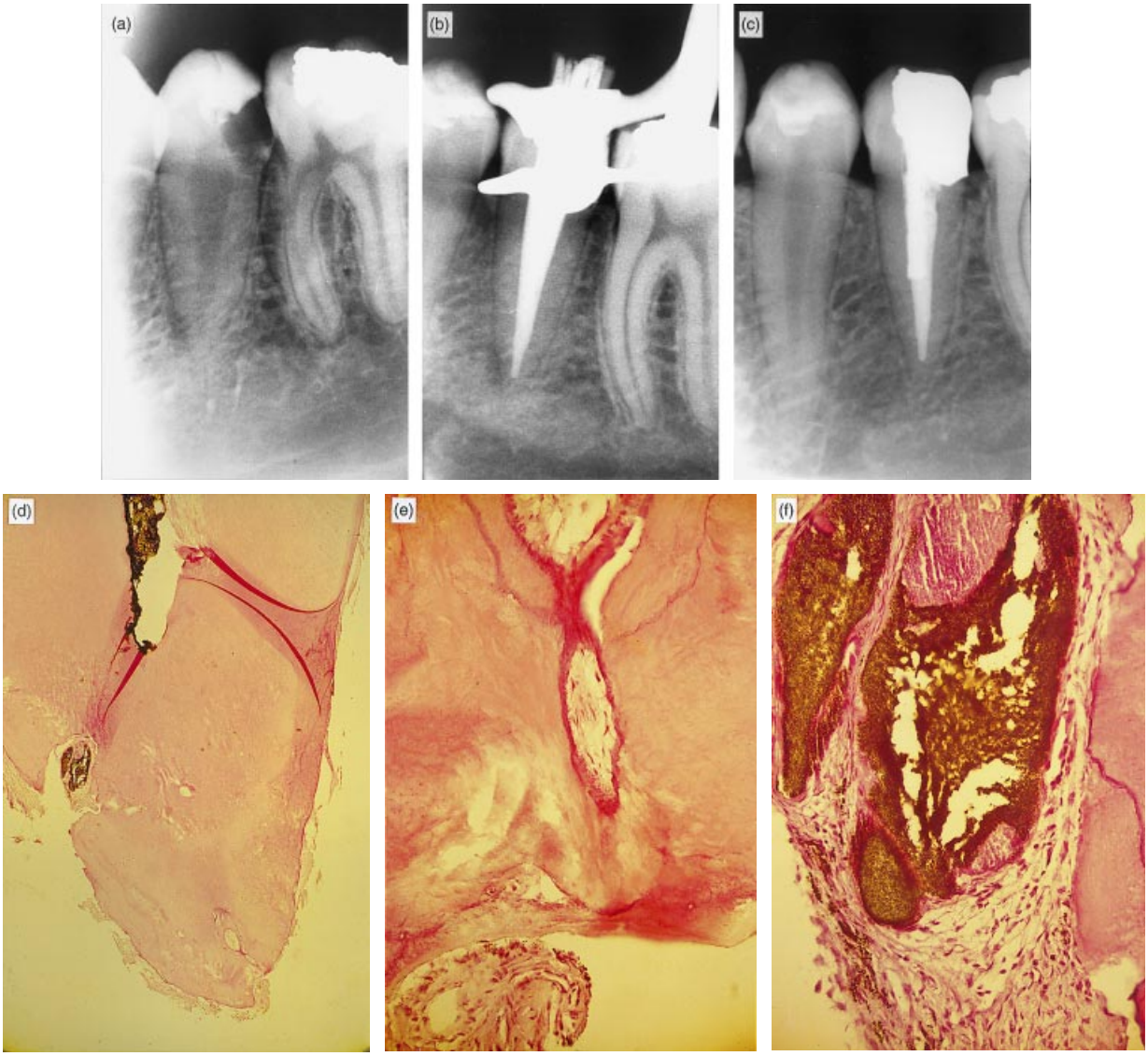

Fig. 4 Case 37 (Table 1). (a) A 16-year-old female. Tooth 35 had spontaneous pain with large distal carious cavity. The tooth was tender to percussion, with an exaggerated response to tests (hot, cold, electric). The radiograph shows a deep carious lesion close to the pulp chamber and a small radiolucency on the mesial aspect of the root. The diagnosis was irreversible pulpitis and orthograde routine root canal treatment was performed. (b) Laterally condensed gutta-percha and a zinc oxide-eugenol sealer, terminating 1 mm from the radiographic apex. Sealer in a lateral canal. (c) At 3-years and 1-month follow-up. The sealer in the lateral canal is no longer visible, and the lesion had disappeared. The tooth was comfortable and not tender to percussion. The case was recorded as success. The tooth was extracted for orthodontic reasons. (d) Root tip with main canal just short of the apex, and a lateral canal terminating 2.1 mm short of the apex. The lateral canal contains sealer. H & E; original magnification × 25. (e) Apical area with some attached periapical tissue. No inflammation in apical part of main canal and no inflammation in the periapical tissue. H & E; × 155. (f) Termination of lateral canal. Accumulations of sealer squeezed into the pulpal tissue of the lateral canal. An area of necrosis between the two accumulations of the sealer, and a number of inflammatory cells can be seen. H & E; × 155.

et al. 1985, Pascon *et al.* 1991). Root canal anatomy (straight, curved or ramified) and pathosis (resorption/apposition) were more important for the consequences of debridement than any particular method of instrumenting the canal (Langeland *et al.* 1985, Pascon *et al.* 1991).

The recommendation for the termination at the apical constriction is based on sound wound healing principles: the severance of the tissue in that area will create the smallest possible wound: the less tissue to heal the better the cure.

The patency technique as advocated by Buchanan (1989) violates this cure. 'Patency' means the use of 'a small flexible K-file which will passively move through the apical constricture without widening it'. This is a

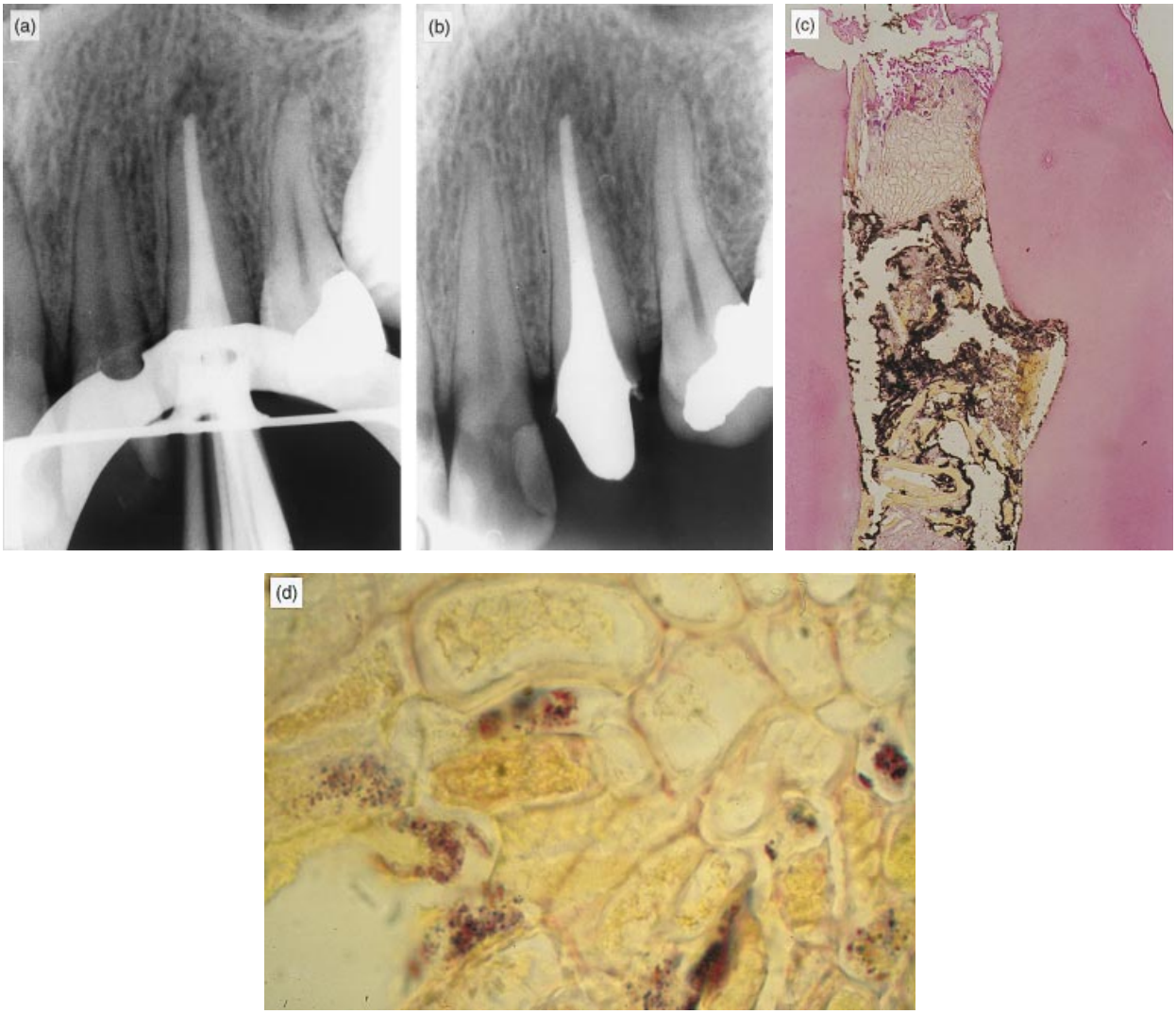

Fig. 5 Case 24 (Table 1). (a) A 20 year-old-female, tooth 23. The clinical crown was destroyed totally by caries. Small periapical radiolucency but no clinical symptoms. Root canal treatment performed in one visit. (b) At 1-year follow-up the tooth was tender to percussion and palpation. The radiograph showed a periapical radiolucency. The case was diagnosed as a failure and an apicectomy performed. (c) Part of root tip and canal. In coronal part of the canal there is debris mixed with sealer and in the foramen foreign material is present. Large vegetal cells bordered apically by an accumulation of neutrophilic leucocytes are present. H & E; $\times 45$. (d) Bacterial colonies in the vegetable cells. Brown & Brenn; $\times 450$.

total misconception of the problem. Moving a file through the foramen means cutting into undisturbed tissue (Fig. 1b; 2b) and causing a larger wound. Nygaard-Østby (1939 and 1944) stated that a better prognosis was obtained when that tissue was left undisturbed in vital cases, and Langeland (1987) has repeatedly demonstrated that undisturbed and uninflamed tissue also occurs in cases where there is necrosis in the canals. It is unfortunate that many American endodontic schools disregard the histopathological facts and 50% teach some form of patency in their graduate or undergraduate programs. However, only three schools require instrumentation and filling to the radiographic apex (Cailleteau & Mullaney 1997).

The irrigant used in the present study (1% NaOCl) was chosen because this concentration produces the lowest possible toxicity combined with the highest possible bactericidal effect (Spångberg *et al.* 1973).

The medicament used, $Ca(OH)_2$, was chosen because, despite its low solubility the $Ca(OH)_2$ ions raise the pH sufficiently to kill bacteria, and the large amount of $Ca(OH)_2$ can be packed into the root canal with little risk of periapical irritation over a long period (Safavi & Nichols 1993). The initial high pH of the $Ca(OH)_2$ is obviously

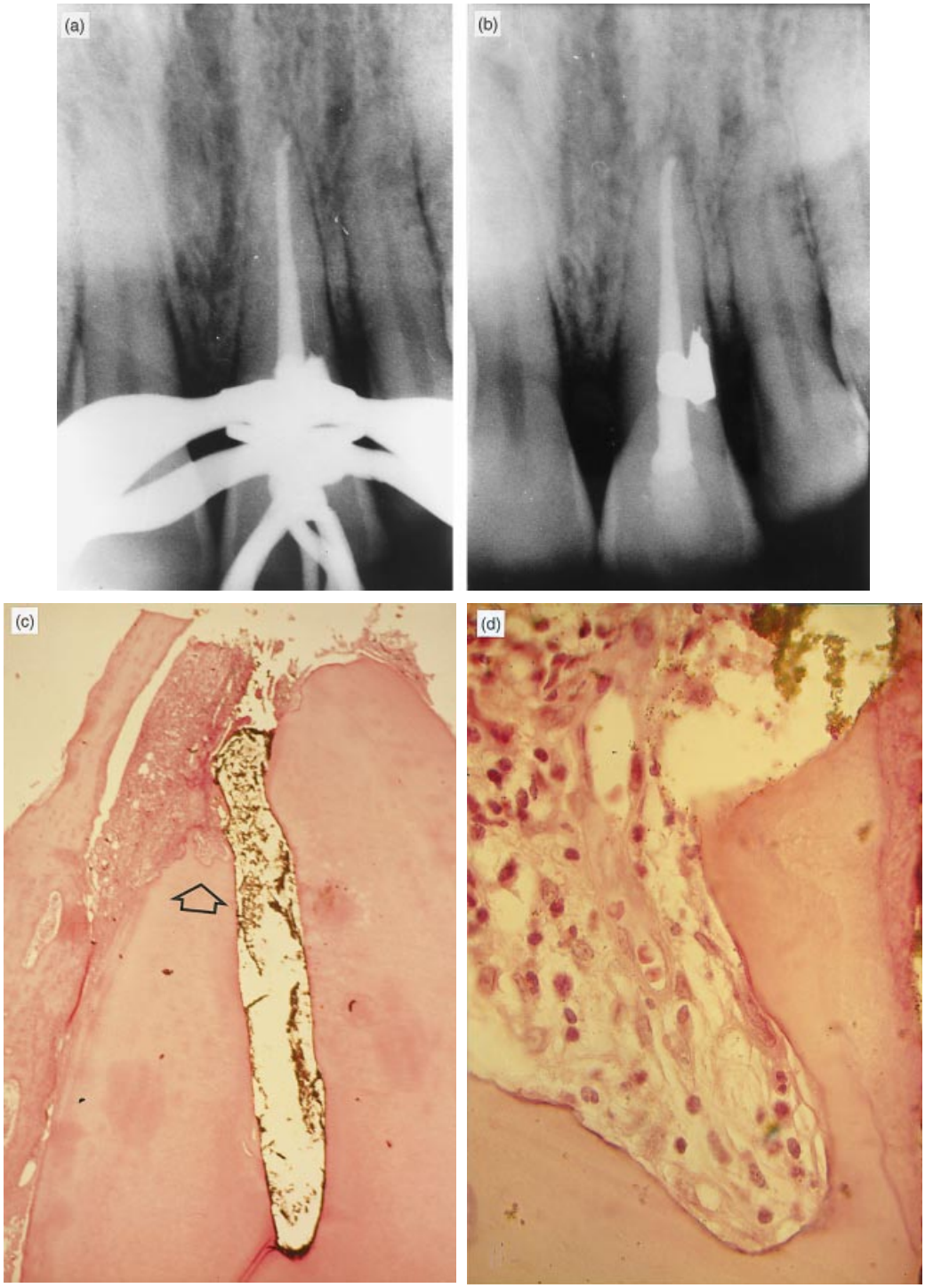

Fig. 6 Case 22 (Table 1). (a) A 23-year-old female, tooth 21. A diagnosis of irreversible pulpitis was made and root canal treatment undertaken. In the post-operative radiograph the obturation deviated distally but it seemed limited to the canal. No periapical radiolucency was present. (b) At 1-year follow-up a radiolucency was evident and the tooth was tender to percussion. The case was diagnosed as a failure and an apicectomy performed. (c) Section of root bucco-lingually. Obturation material protruding into the periodontal ligament through the foramen on the buccal aspect of the root. H & E; × 12. (d) From the area indicated by the arrow (in Fig. 6c), an area of resorption filled with inflamed tissue and resorbing cells, sealer particles in the most apical area. H & E; × 400.

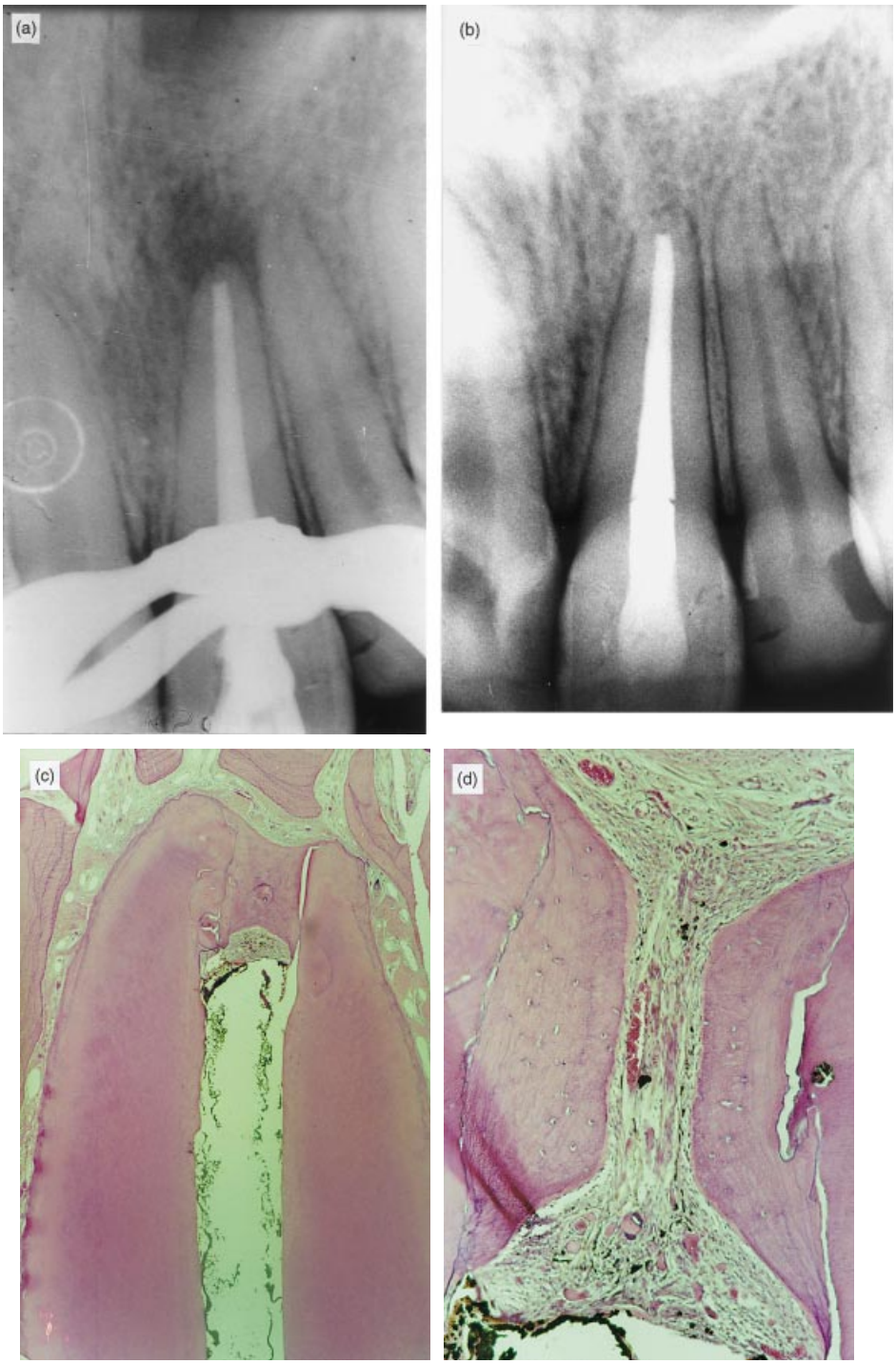

Fig. 7 Case 14 (Table 1). (a) A 18-year-old female, tooth 21. Spontaneous pain and pain to percussion, with swelling in the vestibule. The tooth had two composite restorations and did not respond to sensitivity tests. As emergency treatment incision and drainage of the abscess was made, followed a week later by root canal therapy. Instrumentation and obturation is about 1 mm from the radiographic apex. (b) A 3-year follow-up indicated the complete radiographic healing of the bone with the image of a continuous lamina dura along the entire root surface. At this time a biopsy was taken. (c) Apex with periapical tissue. Healthy periodontal ligament and trabecular periapical bone. Calcified tissue in this section 'obturated' the entire canal lumen. Soft tissue present coronal to the 'obturation'. H & E; × 15. (d) This section was taken numerous sections after that shown in Fig. 7c: tissue penetrating the apparent 'obturation' (in Fig. 7c). Vessels, nerves, numerous calcifications and sealer particles. H & E; × 60.

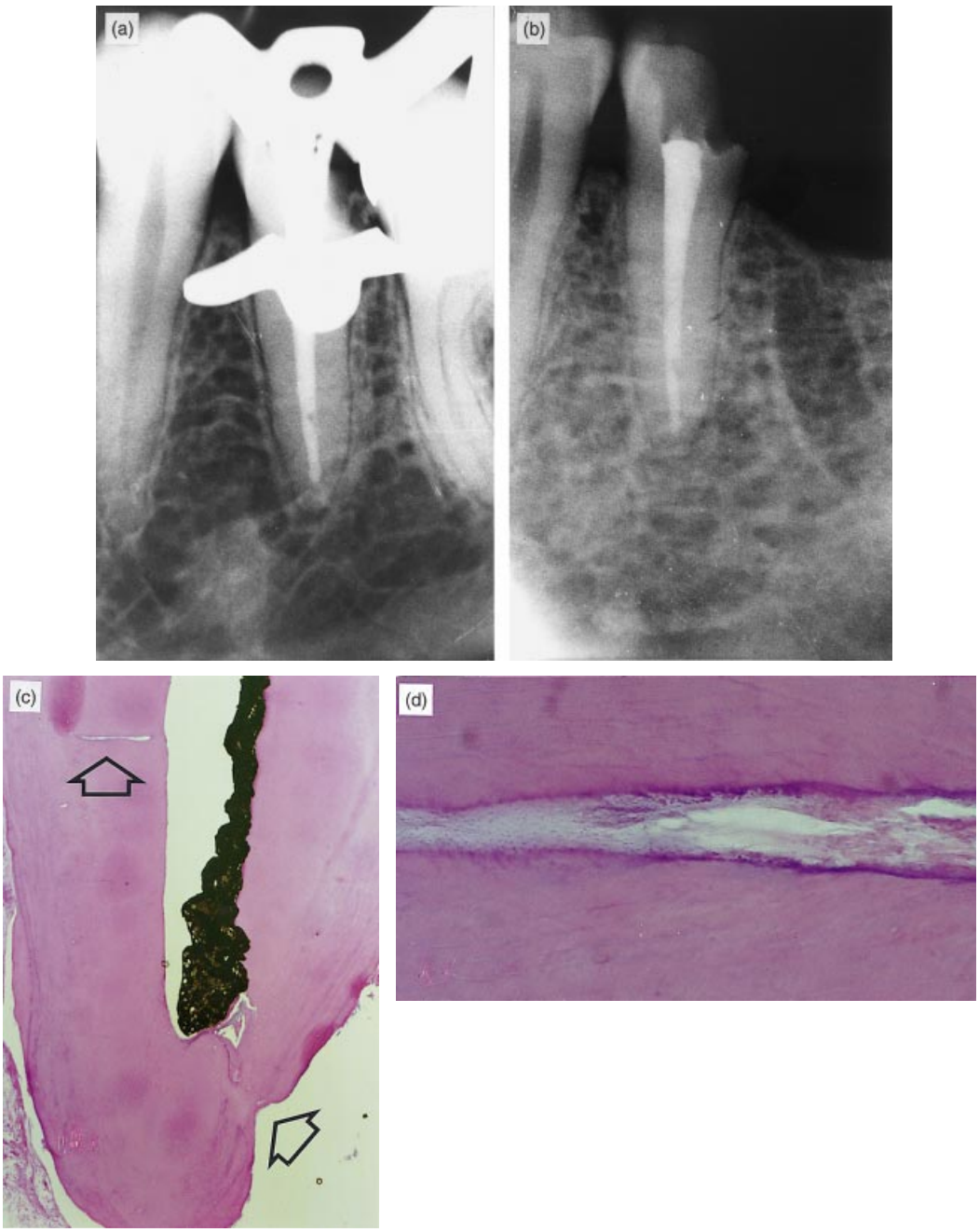

Fig. 8 Case 30 (Table 1). (a) A 25-year-old male, tooth 35. Irreversible pulpitis treated by root canal therapy in a single visit. Instrumentation and obturation about 1.5 mm from the radiographic apex using the cold lateral condensation technique. (b) After 6 years the patient returned following the loss of the coronal restoration. There had been no symptoms. The radiograph showed healthy periapical structures with an evident lamina dura along the root apex. Therefore, the case was recorded as an endodontic success. A longitudinal fracture was seen on the buccal aspect of the root and the tooth was extracted. (c) Root tip with periodontal ligament fragments attached. Foramen short of the apex (oblique arrow) and a lateral canal (vertical arrow). Sealer displaced in the canal. H & E; × 13. (d) Uninflamed tissue in the lateral canal. H & E; × 212.

partly responsible for the wound surface conditions (Fig. 1b, c), but as $Ca(OH)_2$ allegedly changes to $CaCO_3$ it is no longer irritant. Finally, the biological effect of LPS including toxicity, pyrogenicity and macrophage activation is lost by the modification of the lipid A structure by the calcium ions (Safavi & Nichols 1993). Whereas irrigants diffuse layer for layer into and through the pulp tissue, the concentration of the irrigant is diluted

as it mixes with tissue fluids. It must also be realised that the fluids and even $Ca(OH)_2$ particles are transported through vessels into the periapical tissues (Barnes & Langeland 1966).

The long heralded beneficial effect of termination at the cemento/dentine junction is totally irrelevant to healing. First, the junction is not where most practitioners believe it is, in fact it may be 3 mm higher in one area of the canal than in another area, and secondly it cannot be found clinically in any case (Langeland 1995).

The anatomical location of the apical constriction cannot be clinically determined with accuracy. It has been recorded as far as 3.8 mm from the anatomical apex in one SEM study (Gutierrez & Aguayo 1995). Therefore, although clinically desirable, no average millimetre setting may be made to secure a common constant distance from the apical constriction to the anatomical apex and certainly not from the radiographic apex. This fact should be remembered when determining the length of the root canal during root canal therapy. A measurement based on root length rather than canal length would carry the filling beyond the apical foramen and into the periapical tissue (Fig. 6c, d). Only the radiograph together with anatomical knowledge, tactile sense, and keen observation for tissue fluids and blood on instruments and paper points will help modifying the distance. Thus, the 1 mm distance of the obturation from the radiographic apex set by Strindberg (1956) as the standard for ultimate success is erroneous. Biologically and logically, according to all principles of wound healing, the best healing condition exists where the wound is smallest, and since the apical foramen is, more often than not, more than 1 mm short of the radiographic apex, obturations 1 mm short of the radiographic apex are in fact in the periapical tissue creating a larger wound (Ricucci *et al.* 1990). At this level, sealers and gutta-percha will cause tissue destruction, and inflammation, and a foreign body reaction, contrary to that which occurs when the obturation terminates at the apical constriction. This is clearly demonstrated frequently where there is an oblique apex and the filling radiographically ends short on one side and is over-extended on the opposite side of the apex (Fig. 6a–d). The severe periapical tissue reaction is obvious in the absence of bacteria (Fig. 3c, d; 6c, d). The more tissue destruction that takes place, the more time required for healing. The fact is that in none of the success/failure studies has the ever-changing, non-measurable distance, between the radiographic apex and the apical constriction been taken into consideration. Langeland (1957, 1967, 1987 and 1995) alone has stated that this can only be measured in the adequately cut histological section and that is the only method which allows a distinction between facts and speculation.

Although calcifications occur in dead and dying tissue (Robbins 1994a), natural closure of the foramen does not occur. The deposition of calcified tissue into the pulp stump could not be considered a physiological process of healing. Hence, the histological condition of the pulp stump represented (in Fig. 7c, d) even though the case was clinically successful, could not be considered histologically healed because of the presence of inflammatory cells including foreign body cells and calcifications, which are always indicative of pathology. In general pathology calcification, except the deposition of calcium salts during bone turn-over, is defined as 'the deposition of calcium salts in dying or dead tissue' (Robbins 1994a.). In the case illustrated (in Fig. 7c, d), even in the presence of a healthy periodontium, the pulp stump was not histologically healed, because of the deposition of calcified tissue, suggesting a metabolic disturbance. Terms like 'biological closure' or 'physiological closure' do not appear correct, since they are phenomena which occur in the presence of inflammation. Robbins (1994a) describes this under the heading 'Pathologic calcification', be it dystrophic or metastatic.

The claimed descriptions of the apical total closure by calcified tissue are caused by a misinterpretation of the histological sections. Images like that of Fig. 7c could easily be misinterpreted as total closure, whereas the presence of soft tissue and circulation coronally to the plug is evidence that there is an opening. The opening was found in the successive sections (Fig. 7c, d). For a histologic study to be valid, serial section through the entire canal system must be cut. Any study based on random sectioning is 'hit and miss' and invalid. However, in this particular case the inflammation was limited to the pulp stump, since no materials had been forced into the periapical tissue, and it did not interfere with the regeneration of a healthy periapical bone and periodontal ligament.

The filling beyond the apex is accompanied with the lowest prognosis. This is because the materials used are not biocompatible. Whenever such materials are inserted or injected into the connective tissue, tissue destruction, inflammation and a foreign body reaction occurs (Robbins 1994). Under such circumstances clinical failures could be observed even in the absence of bacteria (Fig. 3c, d; 6c, d).

The lateral canals which have received increased attention recently are not relevant to the success rate of endodontic therapy. Since they cannot be debrided (Fig. 2b; 8c, d) – neither mechanically nor chemically – when they appear filled radiographically (Fig. 4b) this is because primarily sealer has been pushed into and

possibly through them. In contact with vital tissue this will cause tissue disintegration, inflammation and a foreign body reaction in the same way as overfilling of the main canal, only at a reduced level, because there is less material (Fig. 4d–f).

Finally even in the cases in which necrosis and bacteria went beyond the foramen(ina) the apical limit of the procedure will be the apical constriction; once the origin of the canal infection is eliminated, the apical necrotic pulp will be removed by the periodontal circulation and by a foreign body reaction.

When cases fail this is because of failure to remove bacterial disintegration products from the apical space of the canal (Fig. 5c, d), or failure to avoid bacterial contamination of the root canal. When healing of the periapical lesion occurs, there is evidence of elimination of bacteria, cell walls of bacteria, and their disintegration products (Fig. 7c, d).

When a lesion reappears following partial healing, the reason is that bacteria that have been dormant or reduced in numbers, re-establish themselves and grow in the root canal (Sundqvist 1992) or the bacterial disintegration products, such as lipopolysaccharides, have not been eliminated fully (Safavi & Nichols 1993, Safavi & Rossomando 1991), or the canal system is contaminated by ingress of microorganisms coronally (Saunders & Saunders 1994).

Conclusions

The best prognosis for root canal treatment is: adequate instrumentation and homogeneous obturation to the apical constriction. The worst prognosis for root canal treatment is: instrumentation and filling beyond the apical constriction.

The second worst prognosis is: obturation more than 2 mm short of the apical constriction, combined with poor instrumentation and obturation. The distance between the foramen and the apical constriction is often more than 1 mm, e.g. 3 mm. Adequate radiographs, knowledge of anatomy, and tactile sense, and not 'apex locators' – will help to determine apical constriction.

Lateral canals and/or apical ramifications: (i) cannot be debrided mechanically or chemically (ii) when 'filled', the injected material causes tissue destruction and inflammation. Radiographic demonstration of them does not mean excellence in endodontics.

Since absence/presence of bacteria in one visit evaluation is only one – although important – aspect of many variables in prognosis, the treatment recommendation will need support of long term comparative studies.

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