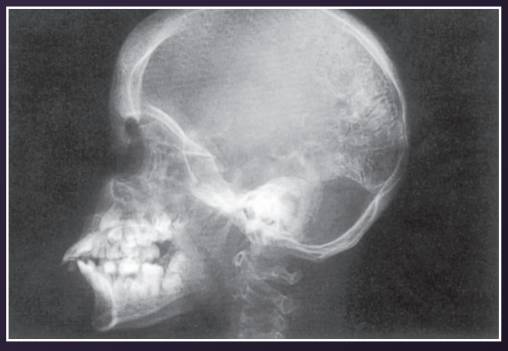
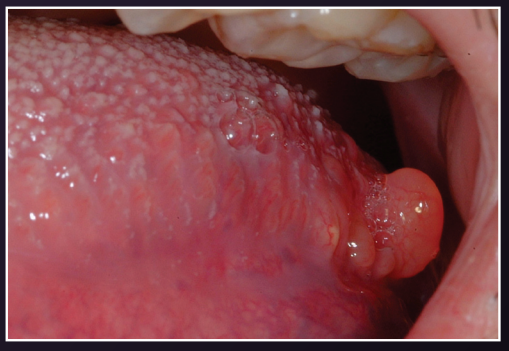
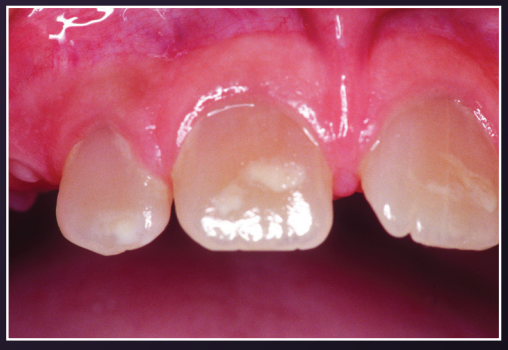
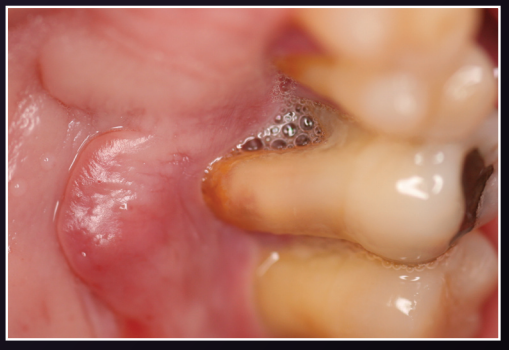
Oral and Maxillofacial Diseases

Fourth Edition

An illustrated guide to the diagnosis and management of diseases of the oral mucosa, gingivae, teeth, salivary glands, jaw bones and joints

Edited by
Crispian Scully, Stephen R Flint, Jose V Bagan, Stephen R Porter and Khursheed F Moos

informa
healthcare

ORAL AND MAXILLOFACIAL DISEASES

ORAL AND MAXILLOFACIAL DISEASES

An illustrated guide to the diagnosis and management of diseases of the oral mucosa, gingivae, teeth, salivary glands, jaw bones and joints

Fourth Edition

Crispian Scully
CBE, MD, PhD, MDS, MRCS, FDSRCPS, FFDRCSI, FDSRCS, FRCPath, FMedSci, FHEA, FUCL, DSc, DChD, DMed (HC), Dr HC
Professor of Oral Medicine, Pathology and Microbiology
University of London
Professor of Special Care Dentistry, Eastman Dental Institute
University College London
Visiting Professor
Universities of Bristol, Edinburgh, Granada and Helsinki
Honorary Consultant
University College London Hospitals, and Great Ormond Street Hospital for Children London, UK

Stephen R Flint
MA, PhD, MBBS, FFDRCSI, FDSRCS, FICD, FTCD
Professor and Consultant in Oral Medicine
Dublin Dental School and Hospital, Trinity College, Dublin, Ireland

Jose V Bagan
MD, DDS, PhD
Professor
Valencia University and Hospital General Universitario de Valencia, Valencia, Spain

Stephen R Porter
MD, PhD, FDSRCS, FDSRCSE, FHEA
Professsor of Oral Medicine
Eastman Dental Institute, University College London
Honorary Consultant
University College London Hospitals, UK

Khursheed F Moos
OBE, MB, BS, BDS, FRCS, FDSRCS, FDSRCSE, FDSRCPS
Emeritus Professor and Honorary Consultant
Glasgow Hospitals, University of Glasgow, UK

informa
healthcare

First published in 1989 as *An Atlas of Stomatology* by Martin Dunitz Ltd, London, UK
This edition published in 2010 by Informa Healthcare, Telephone House, 69-77 Paul Street, London EC2A 4LQ, UK.
Simultaneously published in the USA by Informa Healthcare, 52 Vanderbilt Avenue, 7th floor, New York, NY 10017, USA.

A CIP record for this book is available from the British Library.

ISBN-13: 9780415414944

Orders may be sent to: Informa Healthcare, Sheepen Place, Colchester, Essex CO3 3LP, UK
Telephone: +44 (0)20 7017 5540
Email: CSDhealthcarebooks@informa.com
Website: http://informahealthcarebooks.com/

For corporate sales please contact: CorporateBooksIHC@informa.com
For foreign rights please contact: RightsIHC@informa.com
For reprint permissions please contact: PermissionsIHC@informa.com

Typeset by Exeter Premedia Services
Printed and bound in the United Kingdom

CONTENTS

PREFACE TO THE FOURTH EDITION

This atlas of oral and maxillofacial pathology differs from other atlases by the inclusion of clinical detail on diseases of the oral mucosa, gingivae, teeth, salivary glands, jaw bones and joints, and of a wide range of the more obvious extraoral manifestations. It is intended primarily as a pictorial diagnostic aid, both for dental healthcare professionals, surgeons and physicians, with text that provides a concise synopsis of stomatology.

The previous editions over the past 20 years have been extremely successful and the Atlas has become increasingly popular because of the very wide coverage of oral and maxillofacial diseases and the depth of information contained. Versions have also been published in French, German and Portuguese.

This fourth edition welcomes a new author and provides one of the most comprehensively illustrated coverage of oral and maxillofacial diseases of which we are aware worldwide. The Atlas had also, however, become rather large and heavy and, therefore, it has been revised, updated and re-organized, and some new conditions included. It has been further improved to include better examples of many conditions, particularly additional examples of the more common orofacial conditions or where clinical diagnosis can be difficult because of varied presentations.

A major challenge with all books is how best to persuade publishers to afford enough pages without an excessively high price, and how best to organize and present the material. Our first edition presented conditions according to the International Classification of Diseases but this is incomplete and not always helpful in clinical diagnosis. We have here attempted to highlight the more common and/or important conditions by including more text and/or illustrations of these.

Chapter 1 summarizes the systemic disorders seen mainly in hospital practice, with some detail about the more important conditions but only a less detailed outline of the less common or less relevant disorders: further background can be found in *Medical Problems in Dentistry* (Scully C), Elsevier, London, 2010. Clearly, systemic factors may also influence conditions discussed in other chapters.

Chapters 2–11 cover the conditions which are more common and/or important in day-to-day primary care practice, and largely of local aetiology. Much more detail on aetiopathogenesis, clinical features, diagnosis and management of these conditions has been included. One can always argue as to which disease is best placed in which particular section but we trust readers will find the condition in which they are interested, somewhere.

The specific section on diagnosis and management has also been updated and continues to be presented in the clear and easy-to-use format and covers differential diagnoses by symptoms, signs and site, investigations and management of the various conditions covered in the book, the drugs used in the management of oral diseases and the oral and perioral adverse effects of drug treatment. The further reading has been fully updated.

We are grateful to our colleagues who have kindly provided some illustrations; particular thanks in addition to those acknowledged in the previous editions are to Antonio Azul (Lisbon), Drore Eisen (Cincinnatti), Catherine Flaitz (Houston), Florencio Monje Gil (Valencia), Rodney Grahame (London), Navdeep Kumar (London), Jane Luker (Bristol), Nick Rogers (Rochester), Richard Welbury (Glasgow) and Donald Winstock (London); to the publishers and our co-authors of *Dermatology of the Lips* (Scully C, Bagan JV, Eisen D, Porter S, Rogers RS) Isis Medical Media, Oxford, 2000; *A Colour Atlas of Orofacial Health and Disease* in *Children and Adolescents* (Scully C, Welbury R, Flaitz C, Almedia ODP) Martin Dunitz, London, 2001; *Orofacial Disease; an Update for the Dental Team (*Scully C, Porter SR), Elsevier Harcourt, London & Edinburgh, 2002; *Oral and Maxillofacial Medicine* (Scully C) Elsevier, London and Edinburgh, 2007; *Oral and Maxillofacial Medicine and Pathology* (Scully C, Almeida ODP, Bagan JS, Diz Dios P, Mosqueda A), Blackwell, Oxford, 2010; The oral cavity and lips (Scully C, Hegarty A) In *Rook's Textbook of Dermatology*. 8th edition. Eds: Burns DA, Breathnach SM, Cox N, Griffiths C, Blackwell Science, Oxford, 2010, and to the publishers of *British Dental Journal, British Journal of Dermatology, International Journal of Oral and Maxillofacial Surgery, Journal of Oral Pathology and Medicine, Medicina Oral, Oral Diseases, Oral Oncology* and *Oral Surgery, Oral Medicine and Oral Pathology*.

All typing and image preparation was carried out by Crispian Scully. Our thanks are to Paul Darkins and John Evans for assistance with the image scanning.

Crispian Scully
Jose-Vicente Bagan
Stephen R Flint
Stephen R Porter
Khursheed F Moos

1 MAXILLOFACIAL COMPLICATIONS IN SYSTEMIC CONDITIONS

A range of systemic conditions, treatments and complications can present with maxillofacial manifestations. Some of the more common or important conditions are summarised here, in alphabetic order, with their main maxillofacial manifestations. Others which also have maxillofacial manifestations are discussed elsewhere in the text.

1.1 CARDIOVASCULAR DISEASE

- angina pectoris
- angiomas
- anticoagulants
- congenital heart disease
- drugs
- giant cell arteritis
- hereditary haemorrhagic telangiectasia (HHT)
- hypertension
- polyarteritis nodosa
- transplantation
- Wegener granulomatosis
- Williams syndrome

ANGINA PECTORIS

Angina pectoris—pain related to cardiac ischaemia—may be referred to the jaw rarely, mainly to the mandible. Ulcers may result from use of nicorandil (chapter 1.6). Patients with angina or acute coronary syndrome appear more likely to have a positive interleukin-1 polymorphism and severe periodontitis.

ANGIOMAS

Angiomas (haemangiomas) may be hamartomas or acquired. Most are small but some haemangiomas are large and may result in enlargement of soft tissues and underlying bone (Klippel–Trenaunay or angio-osteohypertrophy syndrome), while others may be part of more widespread disease such as the von Hippel–Lindau syndrome (involving retina, cerebellum, spinal cord, kidneys, pancreas, liver but rarely the mouth), the Dandy–Walker syndrome (associated with posterior fossa brain abnormalities), Maffucci syndrome (multiple enchondromas) or Sturge–Weber syndrome. Haemangiomas may be extensive in Sturge–Weber syndrome (Figs 1.1.1 and 1.1.2) (encephalofacial angiomatosis)—a genetic condition in which a haemangioma affects the upper face and usually extends through the facial skeleton into the brain occipital lobe, producing epilepsy and often glaucoma, hemiplegia and learning impairment and neuralgia. Radiography shows calcification intracranially in the haemangioma. The haemangioma may extend intraorally and be associated with hypertrophy of the affected jaw, macrodontia and accelerated tooth eruption. Since the patients are often treated with phenytoin there is frequently also drug-related gingival enlargement.

Treatment for haemangiomas can include injection of sclerosants, or surgery (scalpel, laser, cryosurgery, photocoagulation or plasma knife).

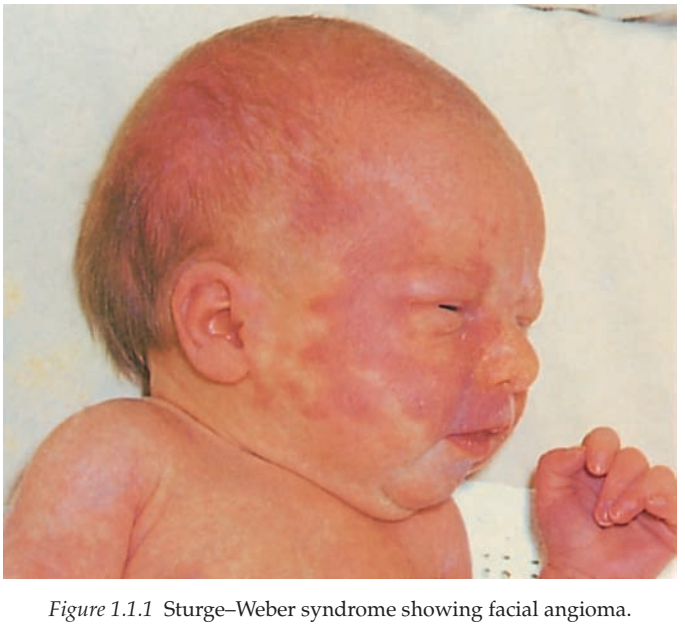


Figure 1.1.1 Sturge–Weber syndrome showing facial angioma.

ANTICOAGULANTS

Anticoagulants cause a bleeding tendency which may manifest with gingival bleeding or oral bruises.

CONGENITAL HEART DISEASE

Congenital heart disease may result in central cyanosis—seen especially in the lips, tongue and other mucosae which appear purple: the teeth appear milky white in contrast (Figs 1.1.3 and 1.1.4). Tetralogy of Fallot, one of the more common of the cyanotic congenital heart diseases, consists of ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy and an aorta that overrides both ventricles. Children with congenital heart disease in some studies have tended to have significantly more caries and an increased prevalence of fissured and geographic tongue.

DRUGS

Drugs such as cardioactive or antihypertensive agents can produce gingival swelling, dry mouth, ulcers, lichenoid and other lesions (see chapter 1.6). Calcium-channel blockers, particularly nifedipine, may lead to gingival swelling (Fig. 1.1.5) (chapter 1.6) and dry mouth. Nicorandil may cause oral (chapter 1.6) as well as anal, gastrointestinal and perioral ulceration. Beta-blockers may cause lichenoid lesions or dry mouth (chapter 1.6). Angiotensin converting enzyme inhibitors may cause

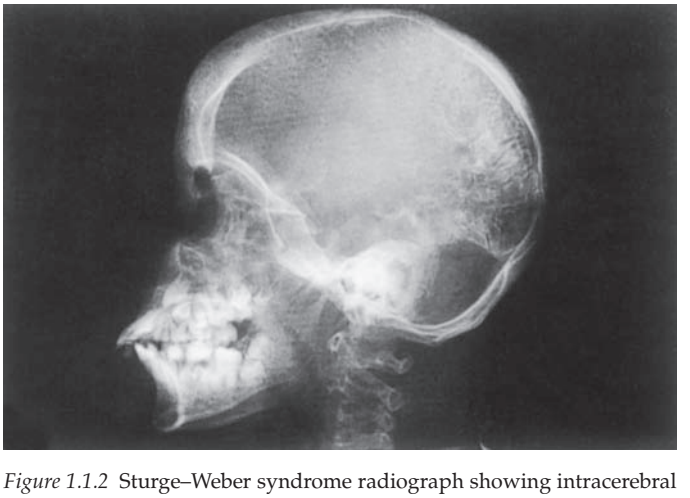


Figure 1.1.2 Sturge–Weber syndrome radiograph showing intracerebral angioma.

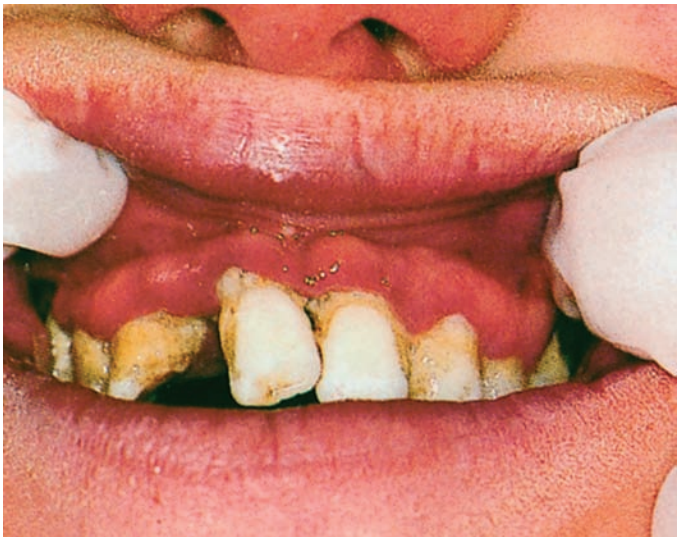


Figure 1.1.3 Central cyanosis in Down syndrome showing purple gingivae and lips.

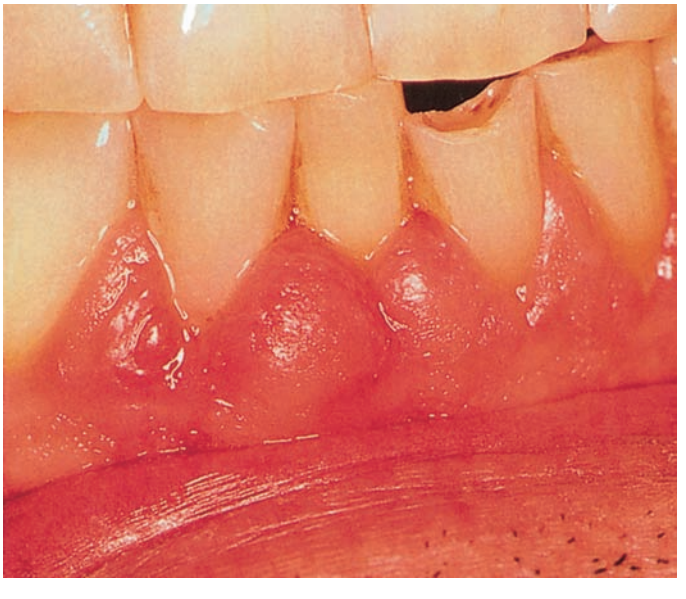


Figure 1.1.5 Nifedipine a common cause of drug-induced gingival swelling showing interdental swellings.

angioedema, or a burning sensation (chapter 1.6). Orolingual angioedema may be a complication of thrombolytics used in stroke victims.

GIANT CELL ARTERITIS

Giant cell arteritis (granulomatous, cranial or temporal arteritis)—a systemic vasculitis—may present with pain, usually over the temple, and can cause retinal artery spasm and blindness, or stroke. Rarely, there can be tongue or lip pain or 'jaw claudication', or ischaemic necrosis of tongue or lip (Fig. 1.1.6). Giant cell arteritis is sometimes associated with polymyalgia rheumatica and is a medical emergency; corticosteroids are indicated to avoid the retinal damage.

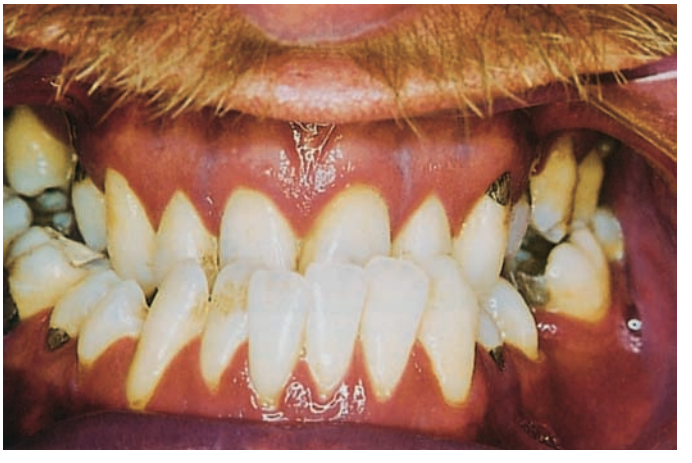


Figure 1.1.4 Central cyanosis in congenital heart disease showing teeth appearing white against the purple gingivae.

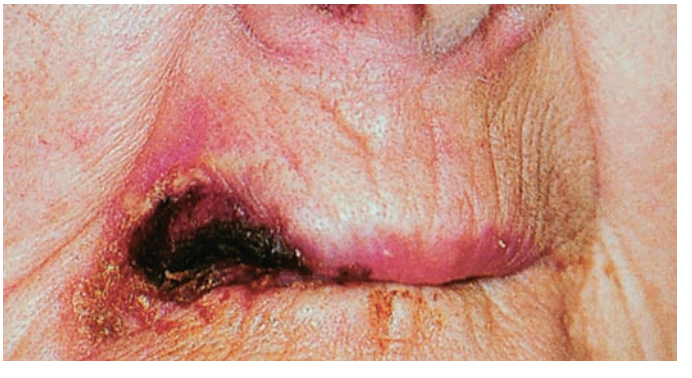


Figure 1.1.6 Giant cell arteritis—a rare cause of lip or tongue ischaemic necrosis.

HEREDITARY HAEMORRHAGIC TELANGIECTASIA (HHT)

HHT (Osler–Rendu–Weber syndrome)—an autosomal dominant genetic disorder caused by disruption in the transforming growth factor (TGF) signalling pathway, by mutations in endoglin gene (HHT1) and activin A receptor type II-like 1 gene (HHT2)—results in blood vessel abnormalities with deficient capillaries. Telangiectases and arteriovenous malformations are found primarily in the nose, skin of the face, hands and mouth and stomach, and also in the respiratory tract, intestines, kidneys, liver and brain. The telangiectatic lesions may bleed profusely (Figs 1.1.7 and 1.1.8) and laser or cryosurgery may be indicated.

HYPERTENSION

Hypertension—various antihypertensive drugs can cause oral complaints, especially dry mouth (chapter 1.6).

POLYARTERITIS NODOSA

Polyarteritis nodosa—a vasculitis affecting medium-sized arteries, possibly related to a reaction to hepatitis B virus, manifests with ischaemic damage mainly to skin, heart, kidneys and nervous system. Mouth ulcers may be seen (Fig. 1.1.9).

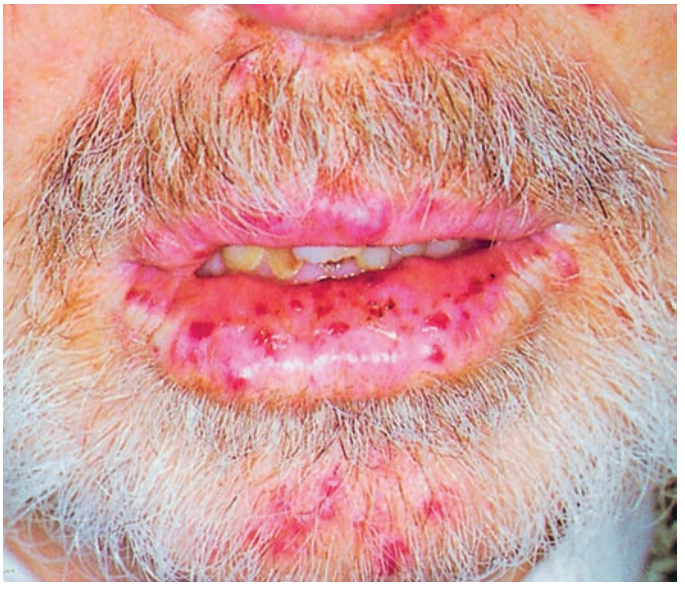


Figure 1.1.7 Hereditary haemorrhagic telangiectasia showing multiple telangiectases.

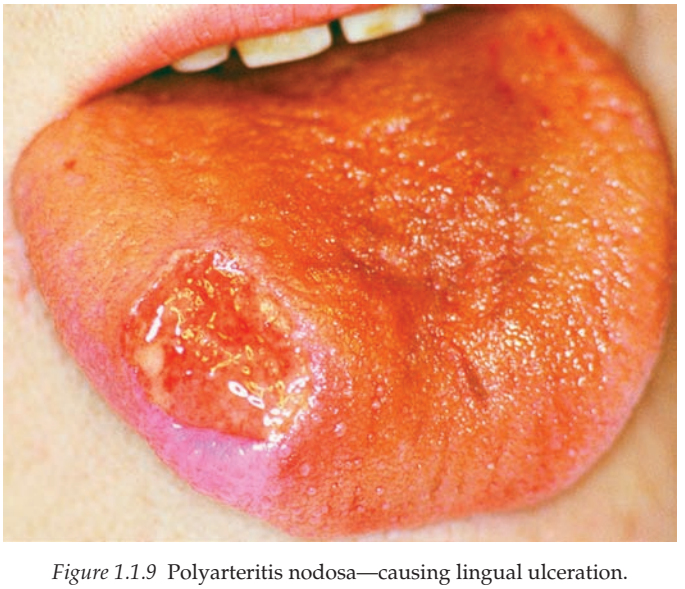


Figure 1.1.9 Polyarteritis nodosa—causing lingual ulceration.

TRANSPLANTATION

Transplantation necessitates that the patient is T-cell immunosuppressed to prevent graft rejection (chapters 1.6 and 1.7). Oral complications are mainly those related to the immunosuppression needed to prevent graft rejection, and may include infections and neoplasms. Infections such as candidosis, herpes simplex virus-related ulceration and hairy leukoplakia may be seen. An increased susceptibility to lip cancer after transplantation is related to exposure to immunosuppressive agents and UV light. Post-transplantation patients are also prone to graft-versus-host disease (GVH), lymphoproliferative diseases, Kaposi sarcoma and some carcinomas (chapter 1.6). Drugs used after cardiac transplantation may also produce other maxillofacial adverse side-effects; the most common is gingival swelling caused by ciclosporin or nifedepine (chapter 1.6).

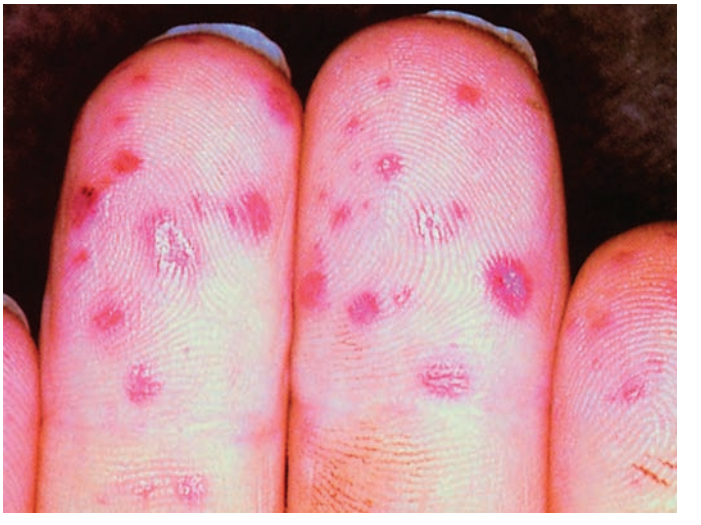


Figure 1.1.8 Hereditary haemorrhagic telangiectasia showing cutaneous telangiectases.

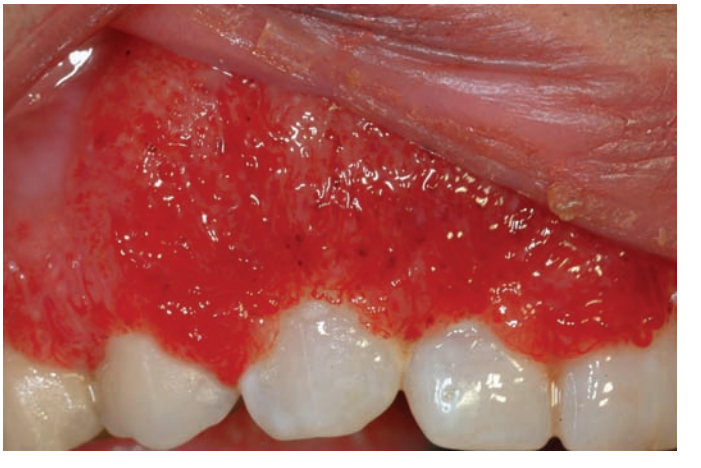


Figure 1.1.10 Wegener granulomatosis showing almost pathognomonic strawberry appearance of gingivae.

WEGENER GRANULOMATOSIS

Wegener granulomatosis—a rare vasculitis characterized by necrotizing granulomatous inflammation of small- and medium-sized blood vessels, with antineutrophil cytoplasmic antibodies directed at neutrophil proteinase 3—mainly affects the respiratory tract and kidneys. Symptoms include antral pain, discoloured or bloody nasal discharge and, occasionally, nasal or oral ulcers or palatal perforation, lingual infarction or sialadenitis. Characteristic, and almost pathognomonic, is the 'strawberry' appearing gingival swelling sometimes seen (Fig. 1.1.10).

Microscopic polyangiitis, a systemic necrotising vasculitis that clinically and histologically affects small-sized vessels but without granulomas, may present similarly.

WILLIAMS SYNDROME

Williams syndrome—a rare congenital disorder involving the cardiovascular system, connective tissue and central nervous system presents

characteristic facial features with full prominent cheeks, wide mouth, long philtrum, small nose with depressed nasal bridge, heavy orbital ridges and medial eyebrow flare. The bony chin is deficient and the mandibular plane angle is high. Microdontia is common and nearly one half have agenesis of one or more permanent teeth. Incisors tend to be tapered or screwdriver-shaped. Molars are often taurodont.

FURTHER READING

Angiero F, Benedicenti S, Romanos GE, Crippa R. Treatment of hemangioma of the head and neck with diode laser and forced dehydration with induced photocoagulation. Photomed Laser Surg 2008; 26: 113–18.

Axelsson S. Variability of the cranial and dental phenotype in Williams syndrome. Swed Dent J Suppl 2005; 170: 3–67.

Beil CM, Keberle M. Oral and oropharyngeal tumors. Eur J Radiol 2008; 66: 448–59.

Carter LM, Brizman E. Lingual infarction in Wegener's granulomatosis: a case report and review of the literature. Head Face Med 2008; 4: 19.

Comi AM. Pathophysiology of Sturge–Weber syndrome. J Child Neurol 2003; 18: 509–16.

Crinzi RA, Palm NV, Mostofi R, Indresano AT. Management of a dental infection in a patient with Sturge–Weber disease. J Am Dent Assoc 1980; 101: 798–800.

Dahan D, Fenichel GM, El-Said R. Neurocutaneous syndromes. Adolesc Med 2002; 13: 495–509.

Davé S, Van Dyke TE. The link between periodontal disease and cardiovascular disease is probably inflammation. Oral Dis 2008; 14: 95–101.

Egred M. Nicorandil-associated ulcerations. Eur J Gastroenterol Hepatol 2007; 19: 395–8.

Frantz MC, Frank H, von Weyhern C, Kiefer J. Unspecific parotitis can be the first indication of a developing Wegener's granulomatosis. Eur Arch Otorhinolaryngol 2008; 265: 131–4.

Friedlander AH, Yoshikawa T, Chang DS, Feliciano Z, Scully C. Atrial fibrillation: pathogenesis, medical-surgical management and dental implications. J Am Dent Assoc 2009; 140: 167–77.

Goteiner D, Ashmen R, Lehrman N, Janal MN, Eskin B. Presence and significance of interleukin-1 polymorphism in patients who present with acute coronary syndrome, angina, and chronic periodontitis: an epidemiologic pilot study. J Periodontol 2008; 79: 138–43.

Kutluhan A, Bozdemir K, Ugras S. The treatment of tongue haemangioma by plasma knife surgery. Singapore Med J 2008; 49: e312–14.

Miyazaki H, Kato J, Watanabe H, et al. Intralesional laser treatment of voluminous vascular lesions in the oral cavity. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009; 107: 164–72.

el-Mostehy MR, Stallard RE. The Sturge–Weber syndrome: its periodontal significance. J Periodontol 1969; 40: 243–6.

Mutalik SS, Bathi RJ, Naikmasur VG. Sturge–Weber syndrome: physician's dream; surgeon's enigma. N Y State Dent J 2009; 75: 44–5.

Ottomeyer C, Hennerici MG, Szabo K, et al. Raising awareness of orolingual angioedema as a complication of thrombolysis in acute stroke patients. Cerebrovasc Dis 2009; 27: 307–8.

Radford DJ, Thong YH. Facial and immunological anomalies associated with tetralogy of Fallot. Int J Cardiol 1989; 22: 229–39.

Rai K, Supriya S, Hegde AM. Oral health status of children with congenital heart disease and the awareness, attitude and knowledge of their parents. J Clin Pediatr Dent 2009; 33: 315–18.

Scully C, Azul AM, Crighton A, et al. Nicorandil can induce severe oral ulceration. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001; 91: 189–93.

Scully C, Roberts G, Shotts R. The mouth in heart disease. Practitioner 2001; 245: 432–7.

Scully C, Diz Dios P, Shotts R. Oral health care in patients with the most important medically compromising conditions; 4. Patients with cardiovascular problems. CPD Dent 2004; 5: 50–5.

Scully C, Diz Dios P, Shotts R. Oral health care in patients with the most important medically compromising conditions; 5. Patients at risk for endocarditis. CPD Dent 2004; 5: 75–9.

Selim H, Selim A, Khachemoune A, Metwally SA. Use of sclerosing agent in the management of oral and perioral hemangiomas: review and case reports. Med Sci Monit 2007; 13: CS114–19.

Sharathkumar AA, Shapiro A. Hereditary haemorrhagic telangiectasia. Haemophilia 2008; 14: 1269–80.

Shiboski CH, Regezi JA, Sanchez HC, Silverman Jr S. Oral lesions as the first clinical sign of microscopic polyangiitis: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002; 94: 707–11.

Shotts RH, Scully C, Avery CM, Porter SR. Nicorandil-induced severe oral ulceration: a newly recognized drug reaction. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999; 87: 706–7.

Stecksén-Blicks C, Rydberg A, Nyman L, Asplund S, Svanberg C. Dental caries experience in children with congenital heart disease: a case-control study. Int J Paediatr Dent 2004; 14: 94–100.

Tasioula V, Balmer R, Parsons J. Dental health and treatment in a group of children with congenital heart disease. Pediatr Dent 2008; 30: 323–8.

Thomas-Sohl KA, Vaslow DF, Maria BL. Sturge–Weber syndrome: a review. Pediatr Neurol 2004; 30: 303–10.

Turgeman Y, Atar S, Rosenfeld T. "Cyanotic blue tongue" in severe rheumatic tricuspid regurgitation. Isr Med Assoc J 2001; 3: 286–7.

Wakefield YS, Theaker ED, Pemberton MN. Angiotensin converting enzyme inhibitors and delayed onset, recurrent angioedema of the head and neck. Br Dent J 2008; 205: 553–6.

Weeda Jr LW, Coffey SA. Wegener's granulomatosis. Oral Maxillofac Surg Clin North Am 2008; 20: 643–9.

Yang Y, Sun M, Hou R, et al. Preliminary study of fibrin glue combined with pingyangmycin for the treatment of venous malformations in the oral and maxillofacial region. J Oral Maxillofac Surg 2008; 66: 2219–25.

1.2 ENDOCRINOLOGICAL AND METABOLIC CONDITIONS

- Addison disease
- congenital hypoparathyroidism
- congenital hypothyroidism
- Cushing syndrome
- diabetes insipidus
- diabetes mellitus
- gigantism/acromegaly
- hyperparathyroidism
- hyperthyroidism
- hypothyroidism
- multiple endocrine neoplasia (adenoma) (MEN or MEA) syndromes
- pituitary dwarfism
- precocious puberty
- pregnancy
- preterm children
- metabolic disorders

ADDISON DISEASE

Addison disease—hypoadrenocorticism—is associated with lowered plasma cortisol levels which cause feedback increased pituitary secretion of adrenocorticotrophic hormone (ACTH) and precursor hormones with melanocyte stimulating hormone-like activity, causing mucocutaneous hyperpigmentation. Similar hyperpigmentation is also seen in Nelson syndrome (increased ACTH production after bilateral adrenalectomy).

Classically hypoadrenocorticism is of autoimmune aetiology, but the same features may be caused by adrenal neoplasms or, rarely, infections such as tuberculosis, cytomegalovirus or histoplasmosis. There are occasional associations of Addison disease with other diseases, particularly autoimmune diseases, such as the probable TASS syndrome of thyroiditis, Addison disease, Sjögren syndrome and sarcoidosis.

Clinical features

Maxillofacial hyperpigmentation of a brown, grey or black colour, especially at sites of trauma such as the buccal mucosae or tongue, is typical (Figs 1.2.1 and 1.2.2), although most patients with oral hyperpigmentation prove to have causes other than Addison disease.

Generalised skin hyperpigmentation is also seen with particular pigmentation in the sun-exposed or traumatised sites, the areolae and the genitalia.

The patient with hypoadrenocorticism may also complain of weakness, anorexia and weight loss.

Diagnosis

Lowered blood pressure, reduced serum cortisol and sodium, raised serum potassium, reduced 24-hour urinary cortisol levels and impaired Synacthen (synthetic ACTH) test are confirmatory. Raised serum levels of adrenocorticotropin and renin confirm the diagnosis.

Management

Replacement hormone therapy and treatment of the underlying cause.

CONGENITAL HYPOPARATHYROIDISM

Congenital hypoparathyroidism—due to a rare chromosome 1 genetic defect—may cause seizures, retarded growth and mental development and unusual facies, and may manifest with dental hypoplasia and delayed tooth eruption and, if there is an associated immune defect, chronic candidosis (Figs 1.2.3 and 1.2.4). Autoimmune polyendocrinopathy-candidosis-ectodermal dystrophy and Shprintzen (velocardiofacial) syndrome are discussed in chapter 1.7.

CONGENITAL HYPOTHYROIDISM

Congenital hypothyroidism, due to thyroid deficiency at birth (sometimes because of dietary iodine deficiency), may cause retarded growth and mental development and unusual facies, and can present with macroglossia and delayed tooth eruption (Fig. 1.2.5) or salivary agenesis.

CUSHING SYNDROME

Cushing syndrome—hypercortisolism, from excess exogenous corticosteroids—or Cushing disease (hypercortisolism arising from an ACTH-producing pituitary adenoma), can cause facial swelling, hirsutism and erythema (Fig. 1.2.6).

DIABETES INSIPIDUS

Diabetes insipidus—a condition caused by lack of antidiuretic hormone, or resistance to it—is characterized by excessive thirst, excretion of large amounts of severely diluted urine and dry mouth. It may arise rarely from head injury, lithium treatment, Langerhans histiocytosis or neurological involvement in Behçet disease.

DIABETES MELLITUS

Diabetes mellitus—caused by lack of insulin, or resistance to it—is characterized by excessive thirst, excretion of large amounts of severely diluted urine, dry mouth, liability to arteriosclerosis and an immune defect. It can present with periodontal disease (Figs 1.2.7 and 1.2.8), xerostomia (Fig. 1.2.8), candidosis, sialosis, burning mouth sensation or lichen planus. Oral changes may be seen mainly in severe insulin-dependent diabetics. Diabetes is characterised by hyperglycaemia and microvascular changes and phagocyte defects.

Parodontal abscesses, infections and rapid periodontal breakdown are the most obvious maxillofacial features. Dry mouth may be caused by dehydration. Other oral lesions may include candidosis such as angular stomatitis or median rhomboid glossitis, burning sensation of the tongue and other oral mucosal surfaces, and lichenoid lesions induced by hypoglycaemic drugs. Autonomic neuropathy may cause sialosis, or gustatory sweating and, with dehydration, contributes to xerostomia. Hyperglycaemic ketoacidosis may cause halitosis. The incidence of birth defects in newborns of women with diabetes including deformities of the face and palate is approximately three to five times higher than among nondiabetics.

Rare complications include rhinocerebral mucormycosis (zygomycosis; see chapter 1.8), squamous carcinoma, diabetic angiopathy presenting

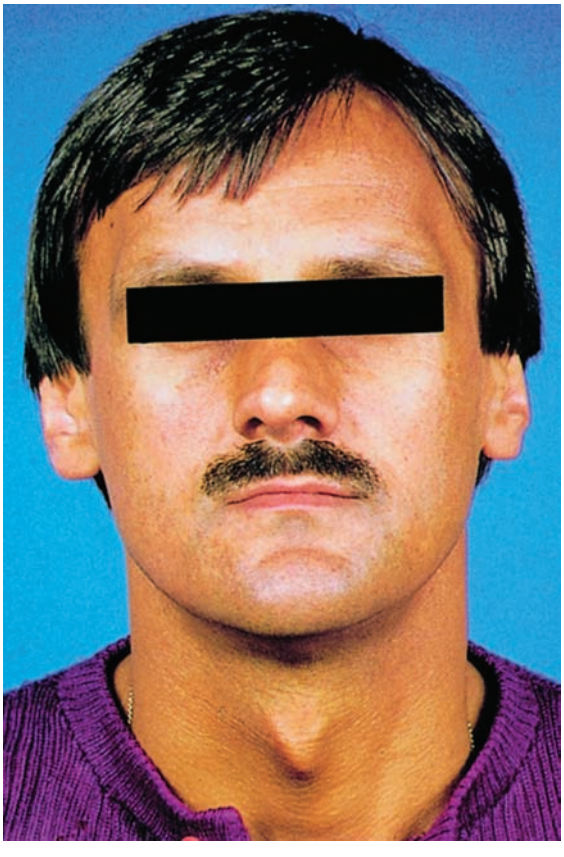


Figure 1.2.1 Hypoadrenocorticism showing cutaneous hyperpigmentation.

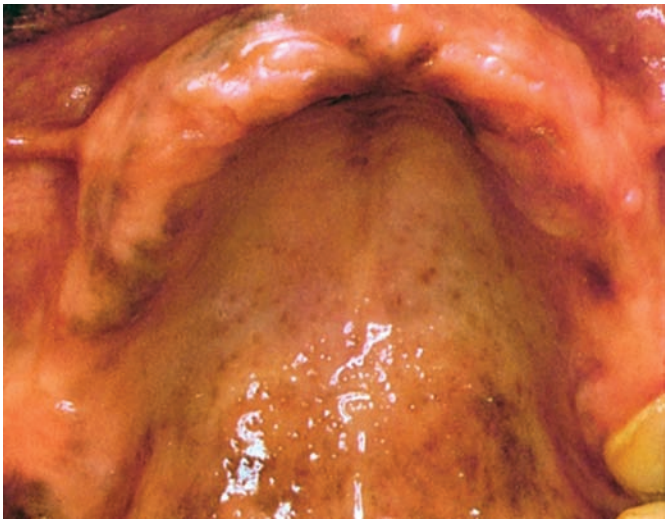


Figure 1.2.2 Hypoadrenocorticism showing oral hyperpigmentation.

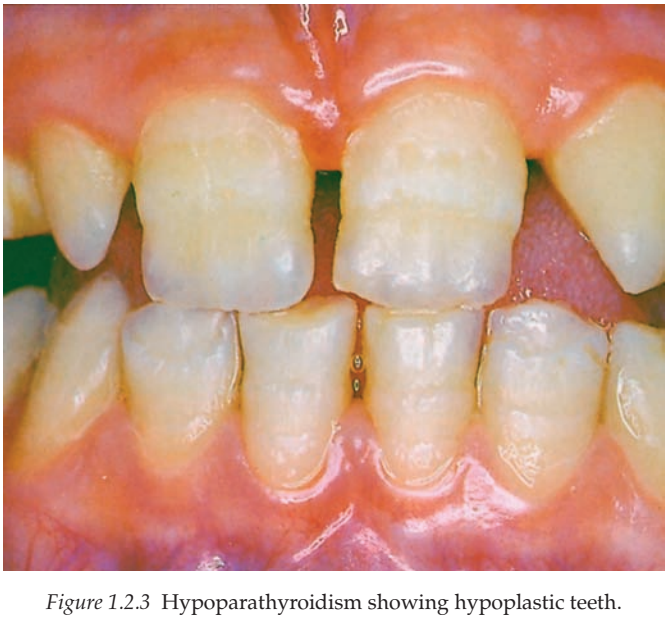


Figure 1.2.3 Hypoparathyroidism showing hypoplastic teeth.

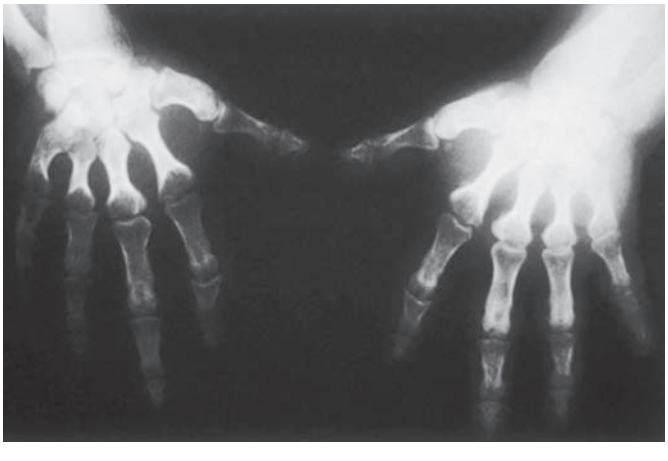


Figure 1.2.4 Hypoparathyroidism showing shortened metacarpal bones.

with palatal petechiae and acanthosis nigricans. Mononeuritis multiplex may cause facial palsy.

GIGANTISM/ACROMEGALY

Gigantism/acromegaly—caused by excessive growth hormone, from a pituitary adenoma—lead to excessive weight and height, plus pressure effects from the tumour such as headache and visual defects. Gigantism can also manifest with spaced teeth, mandibular prognathism, macroglossia and megadontia (Fig. 1.2.9).

HYPERPARATHYROIDISM

Hyperparathyroidism—overactivity of the parathyroid glands resulting in excess production of parathyroid hormone—disturbs calcium and bone homeostasis. This primary type causes skeletal lesions in virtually

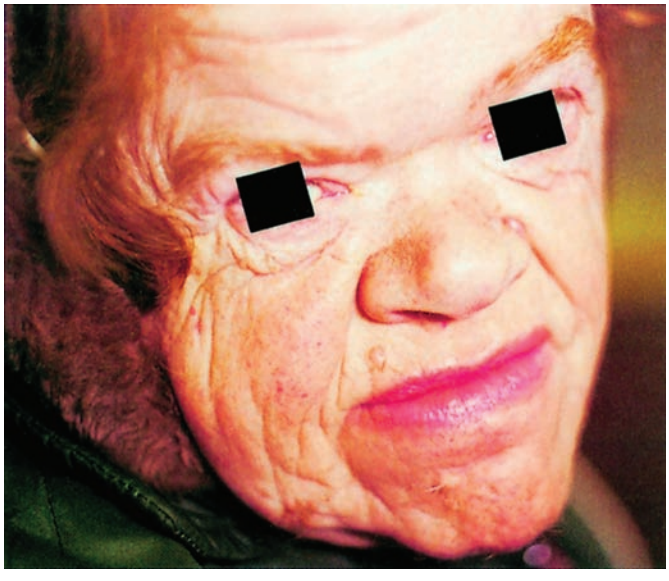


Figure 1.2.5 Hypothyroidism—facies of congenital hypothyroidism.

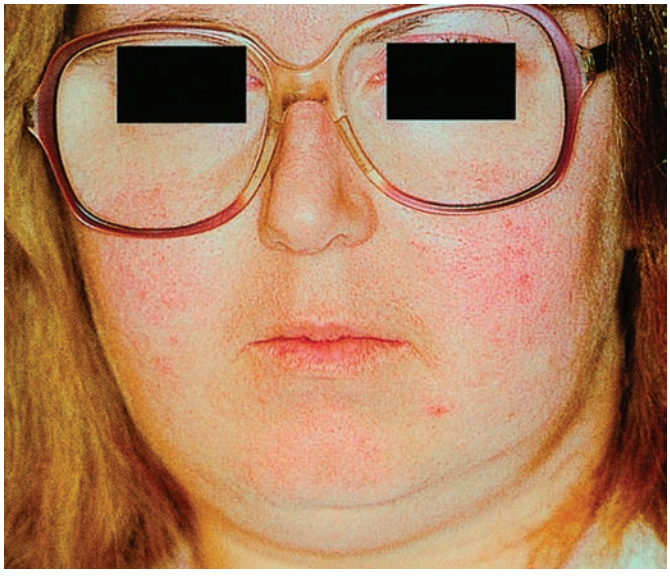


Figure 1.2.6 Cushingoid facies (moon face).

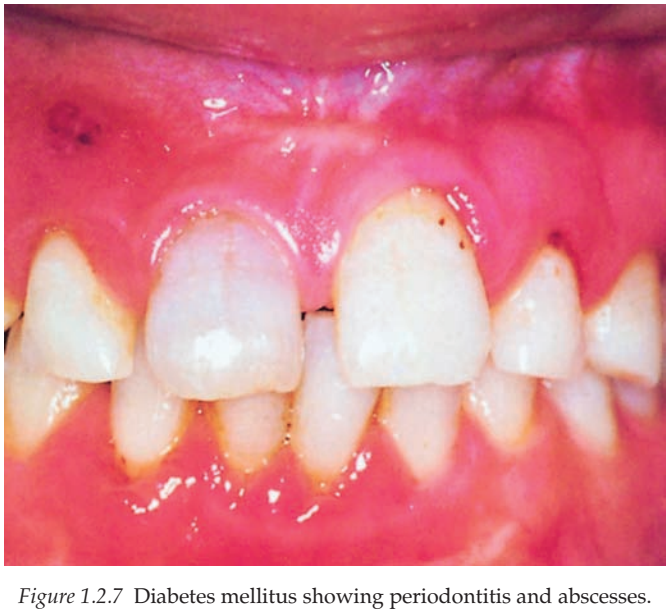


Figure 1.2.7 Diabetes mellitus showing periodontitis and abscesses.

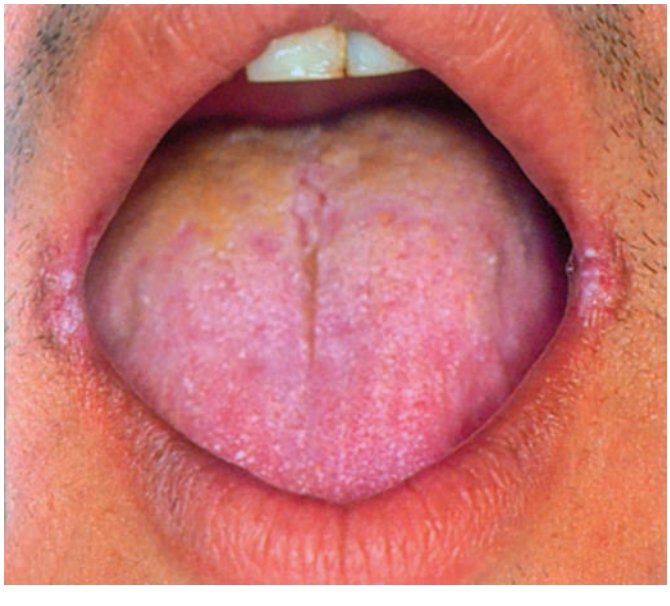


Figure 1.2.8 Diabetes showing dry mouth and angular stomatitis.

all patients, microscopically indistinguishable from the central giant cell granuloma of bone (brown tumours).

Clinical features

Skeletal changes in primary hyperparathyroidism typically include generalised rarefaction, and sometimes lytic lesions (osteitis fibrosa cystica), but an almost pathognomonic oral change is the loss of the lamina dura (Figs 1.2.10–1.2.12). The characteristic radiographic sign of the condition is subperiosteal bone resorption, and 'tufting' of terminal phalanges. Skull and jaw involvement are late complications.

Giant-cell granulomas are not always associated with hyperparathyroidism.