Lesions of the oral mucosa may present originally as macules, papules, nodules, tumours, plaques, vesicles, bullae, pustules, erosions and ulcers. The most common of these clinical forms is the ulcer. Any lesion, irrespective of its original morphology, may become modified to present as an ulcer. This is because the oral mucosa is constantly subjected to the potentially damaging actions of chewing and swallowing hot and cold, rough and hard particles of food and to various chemicals contained in food or dissolved in the saliva. The clinical presentation and progress of ulcers will also be affected by oral debris, saliva and the many organisms which constitute the commensal flora of the mouth.

It seems remarkable that the oral mucosa manages to maintain its integrity so successfully in these circumstances and does not ulcerate more frequently. This integrity of the oral mucosa is maintained by a balance between the resistance of the tissues and damaging external and internal influences. Whenever there is an imbalance between these factors, an ulcer may result. Thus in leukaemia (Figure 8.1) or agranulocytosis, the ability of tissues to repair in the presence of traumatic or infective influences is severely impaired and an ulcer may occur.

The pathogenesis of any ulcer of the oral mucosa while dominated by a single aetiological factor, may be affected by a wide variety of influences. The investigation and treatment of oral ulcers must therefore take into account focal and systemic factors.

Oral ulcers may present the general practitioner with difficult diagnostic and therapeutic problems. This is because, although their aetiology may be diverse, clinically the lesions may have similar appearances. In many cases the aetiology is speculative, if not unknown, so that the treatment is only palliative and not curative. This has prompted the production of a spate of products which may be used for the treatment of these lesions. It is a mistake, however, to encourage self-treatment, as many potentially dangerous ulcers may go unrecognized.

Most ulcers are painful and may interfere with chewing, swallowing, nutrition and even speech.

For descriptive purposes oral ulcers may be divided into the following types:
1. Those in which the ulcers are confined to the oral mucosa.
2. Those in which similar lesions are found on the skin or other mucous membranes.
3. Those associated with general systemic disease.

Ulcers Confined to the Mouth

Recurrent Aphthous Ulceration

Canker sores, dyspeptic aphthosis, vesicular stomatitis and ulcerative stomatitis. This entity is by far the commonest form of oral ulceration. The aetiology is unknown but many interesting hypotheses have been advanced. Emotional factors, allergy, viral or bacterial influences, hormonal imbalance, trauma, anaemia, nutritional deficiencies, gastrointestinal disturbances and autoimmune reactions have all been incriminated.

Clinically these ulcers may be subdivided into three categories:
1. Minor aphthous ulcers (MiAU)
2. Major aphthous ulcers (MjAU)
3. Herpetiform ulcers

Minor aphthous ulceration. These ulcers affect women more than men—they may occur at any age but are more frequently seen between 10 and 20 years of age. They may involve any area of the oral mucosa but are normally confined to the lips and cheeks.

Typically the patient will give a history of an initial burning or itching sensation of the oral mucosa which is followed by the appearance of one to four shallow oral ulcers, 3 to 10 mm in diameter and surrounded by an area of erythema (Figure 8.2). The ulcers are exquisitely painful but usually heal within seven to ten days without scarring.
Major aphthous ulceration. Major aphthous ulcers are considered to be a more severe form of the minor variety. In addition to the lips and cheeks, these lesions may also be seen on the tongue, soft palate and pharynx. The ulcers are much larger (more than 10 mm in diameter) and deeper than the minor variety and may be accompanied by regional lymphadenopathy (Figure 8.3). They take up to six weeks to heal and often result in scarring. In a small percentage of cases scarring may be very severe, sufficient to cause deformities of the lips and tongue.

Herpetiform ulcers. These are described as herpetiform because they resemble in size and shape the ulcers seen in acute herpetic gingivostomatitis. However, a viral aetiology has not been established. The patient may present with crops of 5 to 100 shallow ulcers most of which are between 2 to 5 mm in diameter (Figure 8.4). Occasionally a crop of ulcers may coalesce so that a single ulcer more than 10 mm in diameter may be seen. The ulcers heal without scarring within seven to ten days.

All aphthous ulcers may precede or be part of Behcet’s or Reiter’s syndrome, which will be discussed later.

As the aetiology is unknown the treatment of these ulcers can only be palliative and empirical. Local steroid therapy in the form of either hydrocortisone–succinate or beta-methasone-17-valerate pellets or in a gel combined with an adhesive vehicle (Adcortyl A—in Orabase R), have been used for the treatment of MiAU and MjAU, while herpetiform ulcers appear to respond better to a two per cent suspension of tetracycline as a mouthwash. Rarely, severe episodes of major aphthous ulceration warrant the use of systemic steroids.