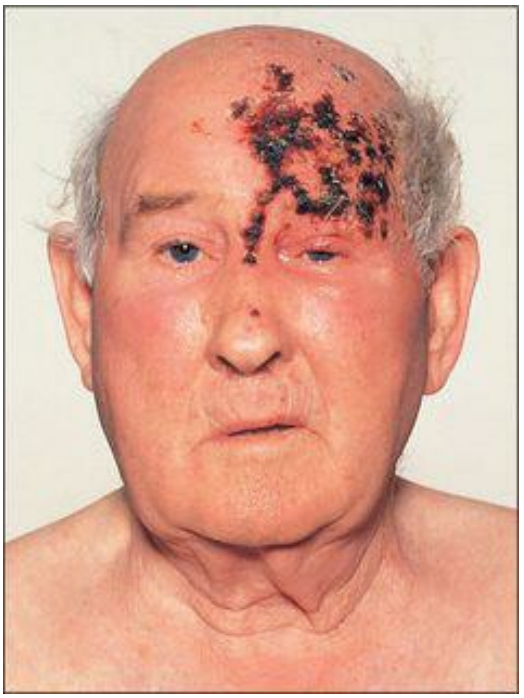


**Figure 13.7** Herpes zoster, involving the eye, along the distribution of the ophthalmic branch of the fifth cranial nerve

*From Mir MA, Atlas of Clinical Diagnosis, 2nd edn. Edinburgh: Saunders, 2003, with permission.*

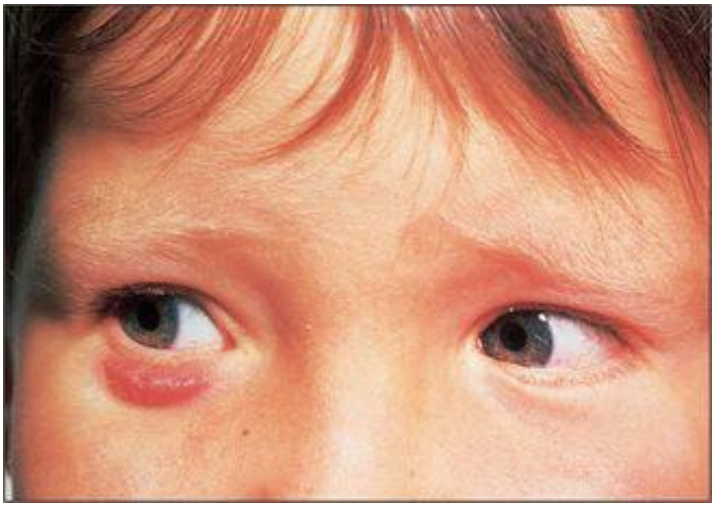


**Figure 13.8** A chalazion; unlike styes, chalazions are not usually tender or painful.

## The ears

## Examination anatomy

The pinna, external auditory canal and ear drum are easily assessed with simple equipment (Figure 13.9). Tests of hearing can also provide information about the severity and anatomical site of hearing loss.

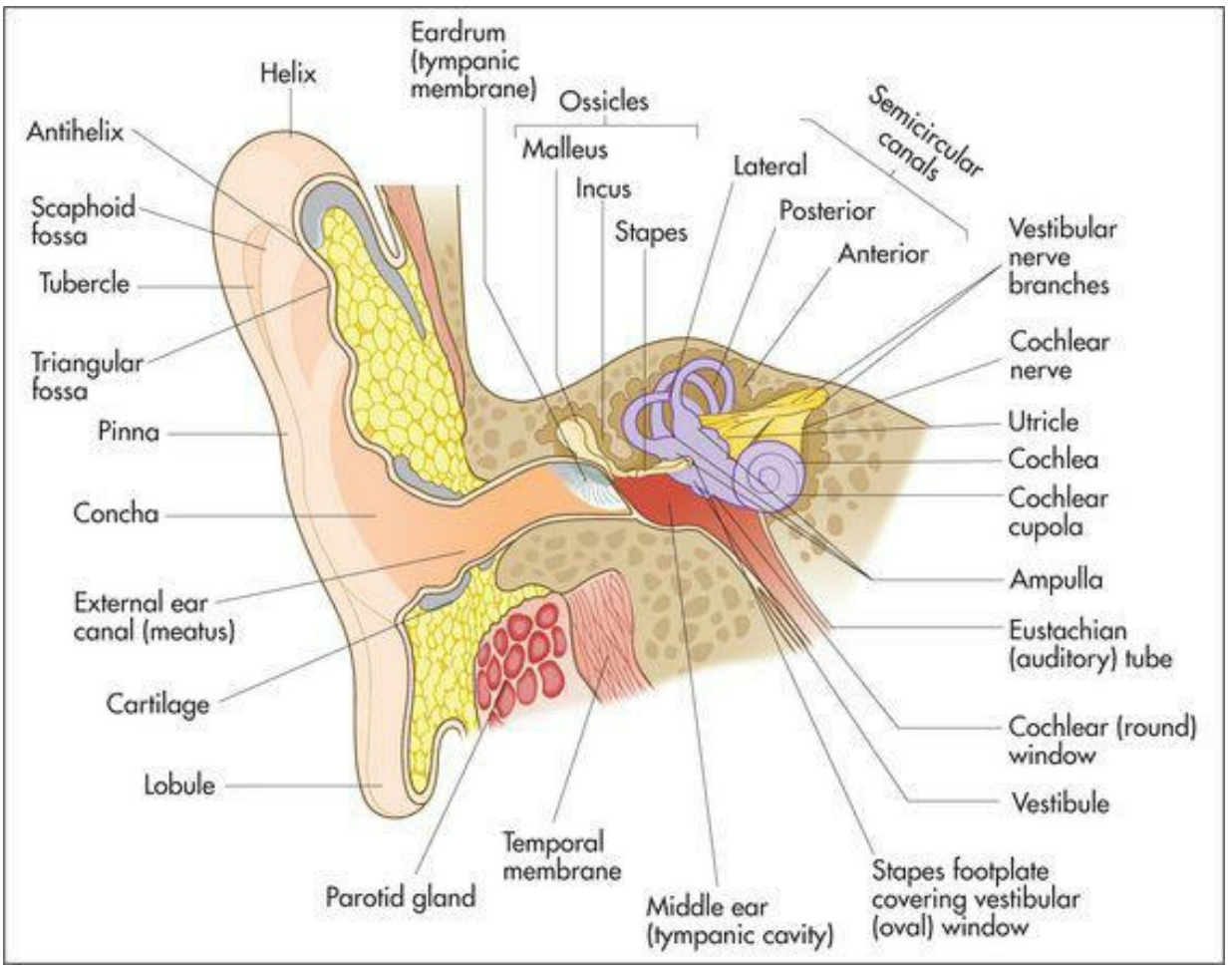



**Figure 13.9** Cross-sectional anatomy of the ear showing the pinna, external auditory canal, middle and inner ear

## Examination method

Ear examination consists of inspection and palpation, auriscopic examination and testing hearing.

**Inspect** the position of the **pinna** and note its size and shape. Note any scars or swelling around the ears. Look for an obvious accessory auricle (separate piece of cartilage away from the pinna), cauliflower ears (haematomas from recurrent trauma, which fill in the hollows of the ear) and bat ears (protrusion of the ears from the side of the head).

Look for **inflammation externally** and any obvious ear *discharge*. Otitis externa (swimmer's ear) is redness of the external canal. Necrotising malignant otitis is usually due to *Pseudomonas* infection and damages the deep tissues, sometimes to the bone.

Look for signs of **gouty tophi** (nodular, firm, pale and non-tender chalky depositions of urate in the cartilage of the ear, specific but not sensitive for gout).

**Palpate the pinna** for swelling or nodules. Pull down the pinna gently; infection of the external canal often causes tenderness of the pinna.

**Auriscopic examination** of the ears requires use of an earpiece that fits comfortably in the ear canal to allow inspection of the ear canal and tympanic membrane (Figure 13.10). This examination is essential if there is a history of recent deafness or a painful ear. Examination is also necessary in the patient who has had a head injury. Always examine both ears!

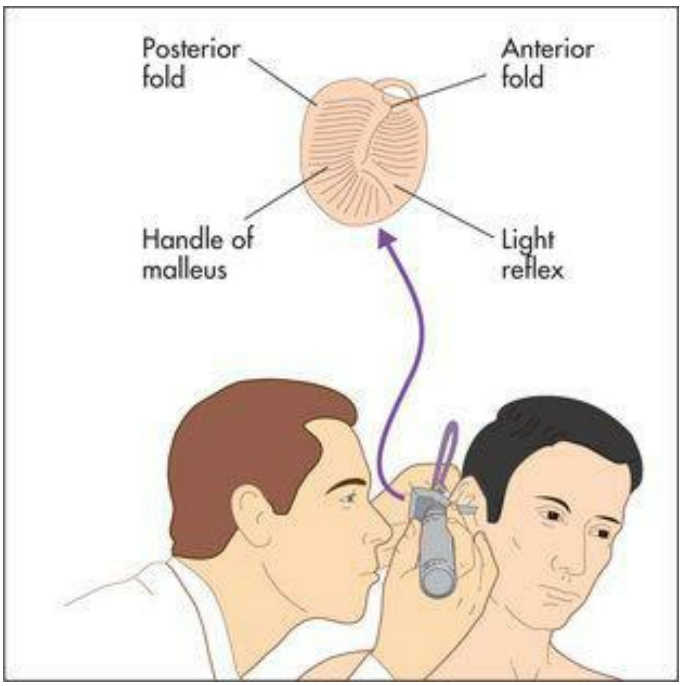



**Figure 13.10** Use of the auriscope

The correct technique is as follows. Ask the patient to turn his or her head slightly to the side, then pull the pinna up, out and back to straighten the ear canal and provide optimal vision. Stretch out the fingers of the hand holding the auriscope to touch the patient's cheek, to steady the instrument and prevent sudden movements of the patient's head. When examining the patient's right ear, the auriscope is preferably held in a *downward position* with the right hand, while using the left hand to pull the pinna. An alternative position involves holding the auriscope upward, but there is a risk that if the patient moves suddenly injury is more likely to occur.

Look at the **external canal** for any evidence of inflammation (e.g. redness or swelling) or discharge. There should be no tenderness unless there is inflammation. *Ear wax* is white or yellowish, and translucent and shiny; it may obscure the view of the tympanic membrane. *Blood* or *cerebrospinal fluid* (watery, clear fluid) may be seen in the canal if there is a fracture at the base of the skull. In patients with herpes zoster, there may be *vesicles* (fluid-filled blisters) on the posterior wall around the external auditory meatus.

Inspect the **tympanic membrane** (ear drum) by introducing the speculum further into the canal in a forward but downward direction. The normal tympanic membrane is greyish and reflects light from the centre at approximately 5 or 7 o'clock (Figures 13.11 and 13.12). Note the colour, transparency and any evidence of *dilated blood vessels* (hyperaemia—a sign of otitis media) (Figure 13.13). Look for *bulging* or *retraction* of the tympanic membrane. Bulging can suggest underlying fluid or pus in the middle ear. Perforation of the tympanic membrane should be noted (Figure 13.14).

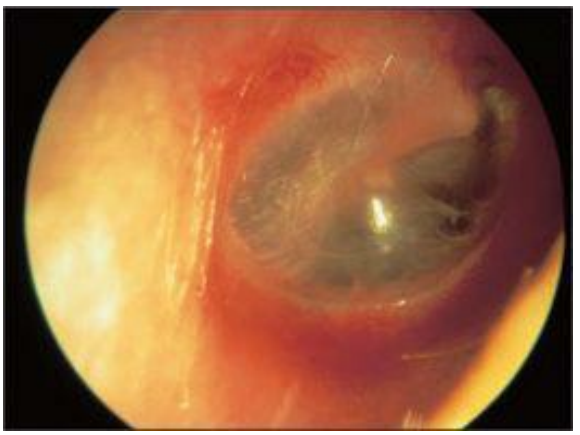
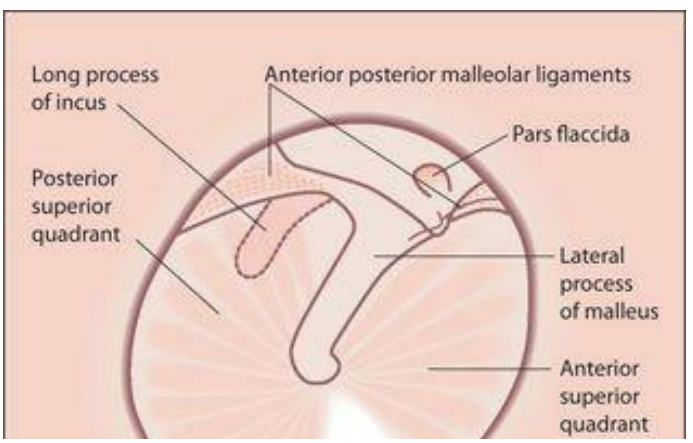


**Figure 13.11** The tympanic membrane as viewed through an otoscope

*From Mir MA, Atlas of Clinical Diagnosis, 2nd edn. Edinburgh: Saunders, 2003, with permission.*



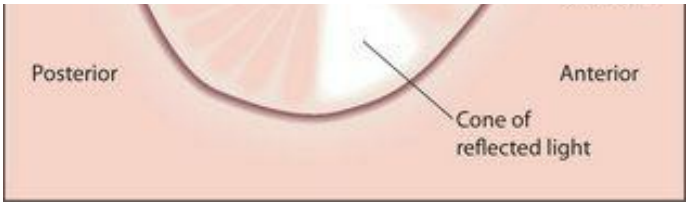



**Figure 13.12** The detail of the tympanic membrane

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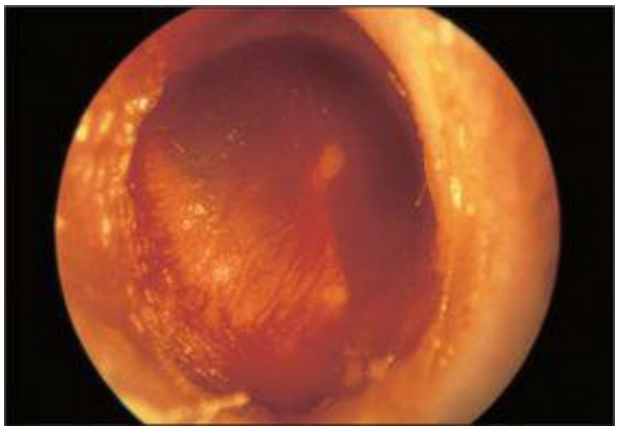


**Figure 13.13** Otitis media with hyperaemia of the tympanic membrane

*From Mir MA, Atlas of Clinical Diagnosis, 2nd edn. Edinburgh: Saunders, 2003, with permission.*

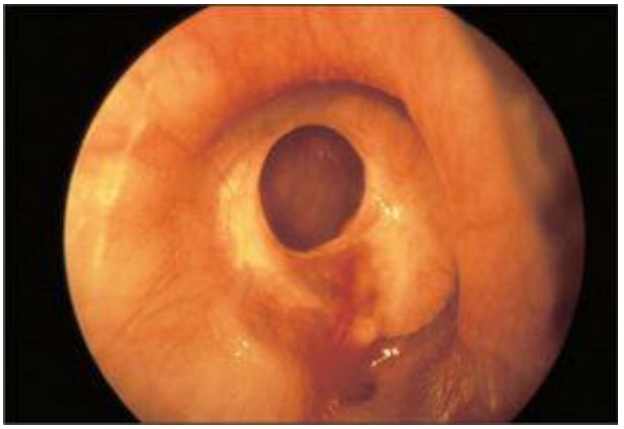


**Figure 13.14** Perforated tympanic membrane

*From Mir MA, Atlas of Clinical Diagnosis, 2nd edn.Edinburgh: Saunders, 2003, with permission.*

If a middle ear infection is suspected, **pneumatic auriscopy** can be useful. Use a speculum large enough to occlude the external canal snugly. Attach a rubber squeeze bulb to the otoscope. When the bulb is squeezed gently, air pressure in the canal is increased and the tympanic membrane

gently, air pressure in the canal is increased and the tympanic membrane should move promptly inward. Absence of, or a decrease in, movement is a sign of fluid in the middle ear.

To test hearing, whisper numbers 60 cm away from one of the patient's ears while the other ear is distracted by movement of the examiner's finger in the auditory canal. Then repeat the process with the other ear. With practice the normal range of hearing is appreciated. Next perform **Rinné's** and **Weber's** tests (page 347):

1. **Rinné's test**: place a vibrating 256 Hz tuning fork on the mastoid process. When the sound is no longer heard move the fork close to the auditory meatus where, if air conduction is (as is normal) better than bone conduction, it will again be audible.

2. **Weber's test**: place a vibrating 256 Hz fork at the centre of the patient's forehead. Nerve deafness causes the sound to be heard better in the normal ear, but with conduction deafness the sound is heard better in the abnormal ear.

## The nose

### Examination method

Nose examination consists of **inspection**, **palpation** and **testing the sense of smell**.

Look at the skin. Note any nasal deviation (best seen from behind the patient and looking down). Note any periorbital swelling (e.g. from sinusitis). Inspect the nares by pressing the tip of the nose upwards with the thumb.

Palpate the nasal bones. Then feel for facial swelling or signs of inflammation. Block each nostril to assess any obstruction by asking the patient to inhale. If there is a history of anosmia (loss of smell), test smell as described in Chapter 11 (cranial nerve I).

A saddle-nose deformity (collapse of the nasal septum) can occur in Wegener's granulomatosis and relapsing polychondritis.

### Sinusitis

Sinusitis is inflammation of the paranasal sinuses. Pain and tenderness over the sinuses occurs, which in adults is classified as acute if less than 4 weeks in duration, subacute if duration 4–12 weeks and chronic if greater than 12

weeks in duration. Most acute sinusitis is secondary to viral infection.

Acute bacterial sinusitis can occur after viral infection or in the setting of allergic rhinitis, in patients with anatomical abnormalities such as nasal septal deformity or polyps in the nose, or in immunocompromised patients. The commonest bacterial causes of sinusitis are *Streptococcus pneumoniae* and *Haemophilus influenzae*. The four key clinical features suggesting that sinusitis may be bacterial are: (i) worsening symptoms after early improvement (a biphasic illness pattern); (ii) purulent discharge from the nose; (iii) tooth or facial pain over the maxillary sinus (especially if unilateral); and (iv) tenderness over the maxillary sinus (unilaterally). Fever can occur but is rare.

Complications of acute bacterial sinusitis can include orbital cellulitis, meningitis, cavernous sinus thrombosis, brain abscess and osteolitis of the sinus bones. Therefore, if patients present with any of the following warning signs—periorbital oedema, visual changes, or changes in mental status—one should be concerned about complicated bacterial sinusitis. Orbital cellulitis typically presents with erythema of the eyelid, oedema of the eyelid and proptosis.

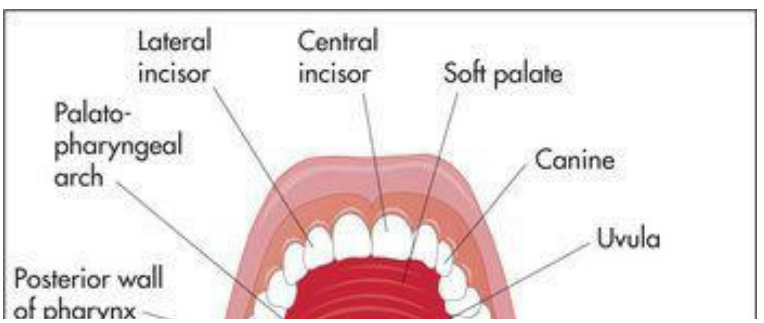
Potential mimickers of acute bacterial sinusitis include Wegner's granulomatosis, carcinoma or lymphoma, sarcoidosis, and in immunocompromised or diabetic patients, fungal sinusitis. Chronic sinusitis presents with chronic sinus congestion, postnasal drip, cough, headache and bad breath.

Rhinocerebral mucormycosis is a fungal infection that destroys the sinuses. A black eschar may be seen on the nasal mucosa or palate.

## The throat

### Examination anatomy

See Figure 13.15.



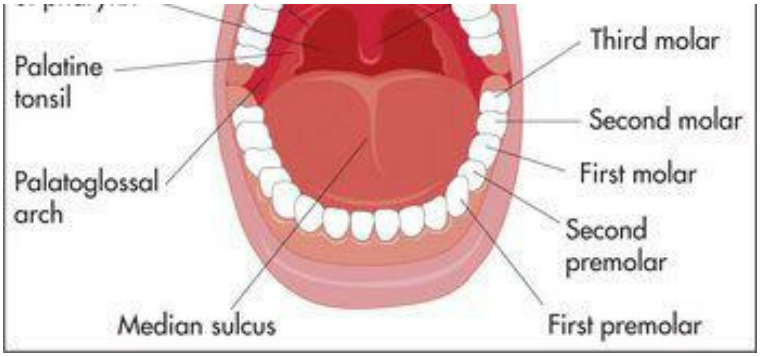



**Figure 13.15** The mouth and throat

## Examination method

Throat examination consists of **inspection** and **palpation**.

Look in turn at the lips, buccal mucosa, gums, palate and teeth. Note any signs of inflammation (e.g. redness, swelling). Inspect the tongue first in the mouth, then ask the patient to poke it out, and then ask the patient to touch it to the roof of the mouth (so that the examiner may look at the floor of the mouth).

Ask the patient to say 'Ah', then inspect the oropharynx and uvula (there is often a need to press a tongue depressor on the posterior tongue to see properly). Inspect the tonsils (note the size, shape, colour, and note any discharge or membrane—they involute in adults and may not be seen).

Palpate the tongue for lumps (wear gloves). Palpate the salivary glands. Examine the cervical lymph nodes.

## Pharyngitis

A sore throat due to an exudative pharyngitis in adults is usually secondary to infection. The specific causes of pharyngitis include viruses in about half the cases, while about 10% are due to the group A beta-haemolytic streptococci. *Neisseriae gonorrhea* is a rare cause of pharyngitis in adults; typically there are sexual risk factors present in the history. The two most important viruses are herpes simplex and adenovirus. Many cases are of unknown cause. Clinically, there is redness of the pharynx with or without ulceration.

Clinical criteria are available for determining whether the pharyngitis is likely due to beta-haemolytic streptococcus or not. The *absence* of cough, along with a history of fever, presence of a pharyngeal exudate on examination and anterior cervical adenopathy together strongly predict the presence of this infection, while the absence of the last three strongly suggests that the infection is not due to beta-haemolytic streptococcus. It is

suggests that the infection is not due to beta-haemolytic streptococcus. It is important to recognize this because beta-haemolytic streptococcus infection of the pharynx can lead to direct infectious complications (otitis media, sinusitis, peritonsillar abscess [quinsy] and submandibular space infection [Ludwig's[e] angina]) and to indirect complications (acute rheumatic fever and glomerulonephritis). Glomerulonephritis is not prevented by antibiotic therapy.

## Epiglottitis

A rare cause of sore throat is epiglottitis. This disease classically presents with a triad of sore throat, painful swallowing (odynophagia) and fever. The patient may uncommonly have stridor which may be misdiagnosed as asthma; here there is inspiratory wheeze due to the inflammation of the epiglottis. Pooling of secretions is another clue to the diagnosis. Urgent medical attention is indicated to prevent airway obstruction.

## Reference

1. McGee S. *Evidence-based clinical diagnosis*, 2nd edn. St Louis: Saunders; 2007. 694

[a] Johann Friedrich Horner (1831–1886), professor of ophthalmology, Zürich, described this in 1869.

[b] Enophthalmos or retraction of the eye, which is often mentioned as a feature of Horner's syndrome, probably does not occur in humans. It may occur in cats. Horner's original paper was very specific about miosis and ptosis, but only casually mentioned that 'the position of the eye seemed very slightly inward'. Apparent enophthalmos results from a combination of ptosis and an elevated lower lid (upside-down ptosis).

[c] Occlusion of any of the following vessels may result in this syndrome: vertebral; posterior inferior cerebellar; superior, middle or inferior lateral medullary arteries.

[d] Sir Jonathon Hutchinson (1828–1913). Among other appointments he was surgeon to Moorfields Eye Hospital. He was president of the Royal College of Surgeons in 1889, elected to the Royal Society in 1882 and knighted in 1908.

[e] Wilhelm Ludwig (1790–1865) was an army surgeon during the Napoleonic wars and a Russian prisoner of war for 2 years. He became court doctor to King Frederick II and was professor of surgery and midwifery at Tübingen. He described submandibular cellulitis in his first paper, published 20 years after he became a professor. The patient described was Queen Catherine of Würtenberg.

# Chapter 14

# The breasts

> Blessed is the physician who takes a good history, looks keenly at his patient and thinks a bit.
> *Walter C Alvarez (1976)*

Breast examination is a vitally important part of the general physical examination. Examinations for breast cancer should be done monthly by the patient and yearly by the doctor in those over the age 40 of years.

## History

The history is important. Important questions to ask include the length of time any mass has been present, presence of pain, change in size or texture over time, relationship to menstrual cycle, and any nipple discharge. Ask about previous cyst aspirations.

Find out about risk factors for breast cancer including any family history of breast or ovarian cancer (and age affected), previous personal history of breast cancer, late menopause, late first pregnancy, mantle radiation, heavy alcohol use, and use of oestrogens post-menopausally. A personal history of atypical hyperplasia (ductal or lobular) increases the risk of breast cancer 3 to 5 times. However, three-quarters of patients presenting with a breast cancer have *no* known risk factors.

The breast cancer genes BRCA1 and BRCA2 are associated with a strong risk of breast (and ovarian) cancer, as well as breast cancer in men. For all women from the age of 50 years, screening mammography[1] is generally recommended.

## Examination

When it is done properly, the examination takes some time to perform (about 3 minutes per breast).[2] This must obviously be explained to the patient at the start. The patient should be offered a chaperone for the examination.

The examination is only just over 50% sensitive for carcinoma but specificity is as high as 90%. The likelihood ratio of a positive examination is 14.1 and the LR of a negative examination is 0.47.[3]

## Inspection

Ask the patient to sit up with her chest fully exposed. There is controversy about the value of inspection of the breasts as part of the examination, but advanced cancers may be obvious at this stage. Look at the *nipples* for retraction (due to cancer or fibrosis; in some patients retraction may be normal) and Paget's disease of the breast (where underlying breast cancer causes a unilateral red, bleeding skin).

Next inspect the rest of the *skin*. Look for visible veins (which if unilateral suggest a cancer), skin dimpling, and for peau d'orange skin (where advanced breast cancer causes oedematous skin pitted by the sweat glands).

A persistent erythematous plaque in the areola area may be contact dermatitis or skin irrigation, but if asymmetric or it has not responded to treatment this may be the malignancy *Paget disease of the breast*.

Ask the patient to *raise her arms above her head* and then lower them slowly. Look for tethering of the nipples or skin, a shift in the relative position of the nipples or a fixed mass distorting the breast (Figure 14.1).

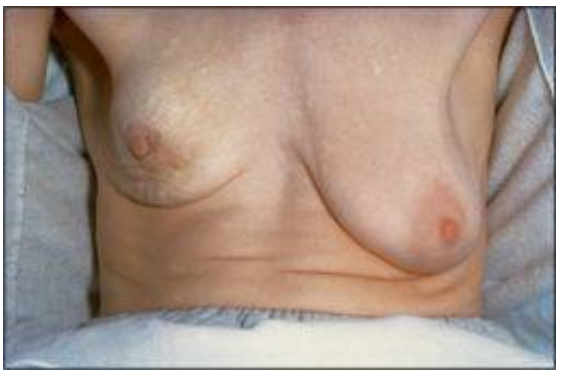


**Figure 14.1** Carcinoma of the right breast, showing elevation of the breast, dimpling of skin, and retraction of the nipple

Note whether there are any obvious visible masses in the axillae.

Next ask her to rest her hands on her hips and then press her hands against her hips (the pectoral contraction manoeuvre). This accentuates areas of dimpling or fixation.

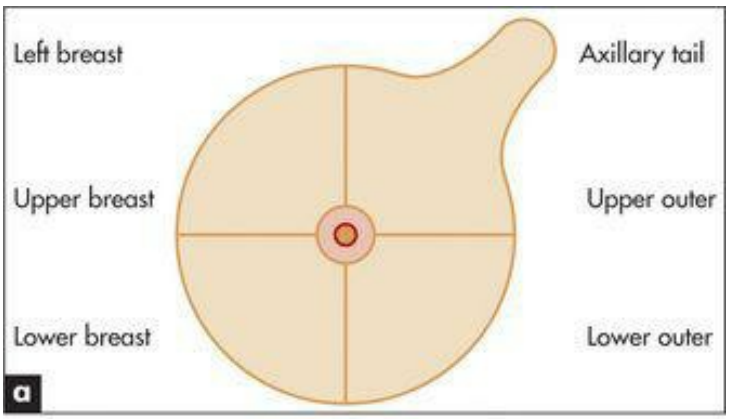
of dimpling or fixation.

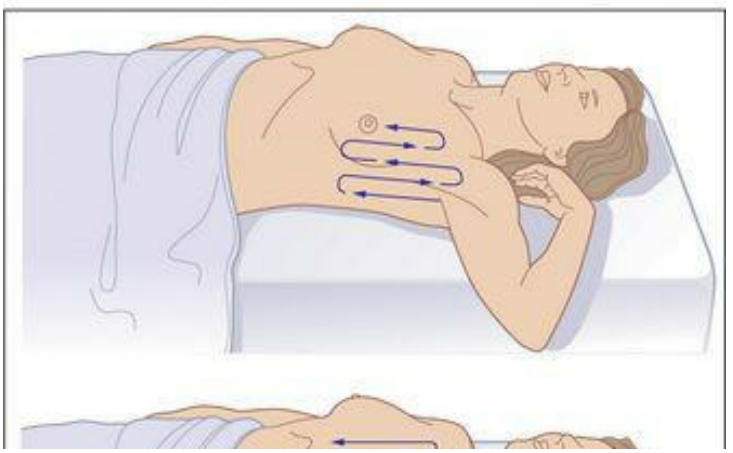
## Palpation

Examine both the supraclavicular and axillary regions for lymphadenopathy. It may be difficult, however, to distinguish an axillary fat pad from an enlarged lymph node.

Then ask the patient to lie down. The examination can be performed only if the breast tissue is flattened against the chest wall. If the breasts are large, it can be helpful to have the patient place her hand on her forehead for the palpation of the lateral aspect of the breast and bring her elbow up level with the shoulder for the palpation of the medial side of the breast.

Palpation is performed gently with the pulps of the middle three fingers parallel to the contour of the breast. Feel the four quadrants of each breast systematically (Figure 14.2a). Don't pinch the breast as you may think you then feel a mass. The total examination should involve a rectangular area bordered by the clavicle, sternum, mid-axillary line and the bra line. Start in the axilla and palpate in a line down to the bra line inferiorly. The pattern of palpation is like that of mowing a lawn, a series of vertical strips that cover the whole of the rectangle (Figure 14.2b).



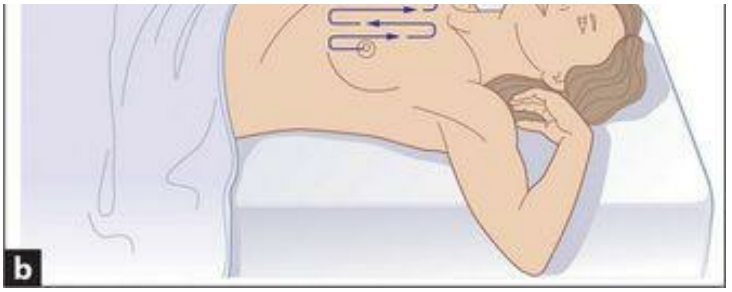



**Figure 14.2** Examination of the breast: (a) quadrants; (b) systematic examination

Each area is palpated three times, using small circular movements and slightly increasing pressure. Palpation is more difficult when a breast implant is present. It is probably best to examine such a patient in a supine position and to keep the ipsilateral arm down at her side.

Next feel behind the nipple for lumps and note if any fluid can be expressed: bright blood (from a duct papilloma, fibroadenosis or carcinoma), yellow serous (fibroadenosis) or serous (early pregnancy) fluid, milky (lactation) or green (mammary duct ectasia) fluid.

Don't mistake normal breast structures for a mass![4] You may feel a rib or costochondral junction normally on deep palpation. The inferior ridge of breast tissue (inframammary fold) may be felt and is symmetrical. You may feel normal rubbery-type plaques (fibroglandular tissue), especially in the upper outer quadrant. It is normal to feel firm breast tissue at the areola border.

## Evaluation of a breast lump

The following five points need to be carefully elucidated if a lump is detected.

1. Position—the breast quadrant involved and proximity to the nipple.

2. Size, shape and consistency—a hard, irregular nodule is characteristic of carcinoma.

3. Tenderness—suggests an inflammatory or cystic lesion; breast cancer is usually not tender.

4. Fixation—mobility is determined by taking the breast between the hands and moving it over the chest wall; in advanced carcinoma the lump may be fixed to the chest wall.

5. Single or multiple lesions present—multiple nodules suggest benign

cystic disease or fibroadenosis.

A palpable breast mass is likely to be significant (called a dominant mass) if it is:[4]

1. Clearly 3-dimensional.
2. Distinct from the surrounding tissue.
3. Asymmetrical compared with the other breast.
4. Persistent throughout a menstrual cycle.
5. *Not* smooth, well-demarcated and mobile.

A palpable breast mass is more likely to be malignant if it has the following characteristics:[4]

1. Very firm.
2. Margins seem poorly defined or have an irregular edge.
3. Immobile or fixed.
4. Associated skin dimpling.
5. Associated retraction of the nipple, or nipple scaling.
6. A bloody nipple discharge.
7. Draining lymph nodes are palpable.

Remember that many normal breasts have palpable lumps and that although benign lumps tend to be soft, moveable and regular, they can also have the characteristics of malignant lumps (*Good signs guide 14.1*). Causes of a lump in the breast are listed in Table 14.1.

**TABLE 14.1** Causes of a breast lump

| Non-tender | Tender |
|---|---|
| Cyst | Cyst |
| Carcinoma | Breast abscess |
| Fibroadenosis (chronic mastitis) | Fibroadenosis |
| Fibroadenoma (benign highly mobile 'breast mouse') | Costal cartilage chondritis |
| Uncommon causes | Inflammatory breast cancer |
| • Trauma, fat necrosis | |
| • Other cysts—e.g. galactocele | |
| • Other neoplasms—e.g. duct papilloma | |
| • Chest wall—e.g. lipoma, costal cartilage chondritis (causes tenderness but not a lump) (Tietze's* disease) | |

* Alexander Tietze (1864–1927), Chief Surgeon, Allerheiligen Hospital, Breslau, Poland. He described the condition in 1921.

In men with true gynaecomastia, a disc of breast tissue can be palpated under the areola. This is not present in men who are merely obese. Causes of breast enlargement in men are presented on page 316.

**GOOD SIGNS GUIDE 14.1** Breast lump characteristics and likelihood of cancer in a woman of average risk[3]

| Sign | Positive LR |
|---|---|
| Mass | 2.1 |
| Fixed | 2.4 |
| Hard | 1.6 |
| Irregular | 1.8 |
| >2 cm diameter | 1.9 |

*From McGee S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.*

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1. Kerlikowske K, Smith-Bindman R, Ljung BM, et al. Evaluation of abnormal mammography results and palpable breast abnormalities. *Ann Intern Med*. 2003;139:274-284.

2. Fenton JJ, Rolnick SJ, Harris EL, Barton MB, Barlow WE, Reisch LM, Herrinton LJ, Geiger AM, Fletcher SW, Elmore JG. Specificity of clinical breast examination in community practice. *J Gen Intern Med*. 22(3), 2007. Mar 332–327

3. Barton MB, Harris R, Fletcher SW. Does this patient have breast cancer? *JAMA*. 1999;282:1270-1280. The clinical breast examination may have an overall specificity which is high (94%) but the sensitivity is poor (54%). Unfortunately interobserver variation seems to be high

4. Pruthi S. Detection and evaluation of a palpable breast mass. *Mayo Clin Proc*. 2001;76:641-647.

# Chapter 15

# The skin, nails, and lumps

> For one mistake made for not knowing, ten mistakes are made for not looking.
> *JA Lindsay*

## The dermatological history

With any rash or skin condition, it is important to determine when and where it began, its distribution, whether it has changed over time, its relationship to sun exposure or heat or cold, and any response to treatment[1] (see *Questions box 15.1*). Ask if pruritus is associated; localised pruritus is usually due to dermatological disease. Determine if pain or disturbed sensation has occurred; for example, inflammation and oedema can produce pain in the skin, while disease involving neurovascular bundles or nerves can produce anaesthesia (e.g. leprosy, syphilis). Constitutional symptoms such as fever, headache, fatigue, anorexia and weight loss also need to be documented.

---

**Questions box 15.1**

## Questions to ask the patient with a rash

1. How long have you had the problem?

2. Have you ever had it before?

3. Is it getting worse?

4. What parts of your skin are affected (e.g. sun-exposed areas, areas in contact with clothing or chemicals)?

5. Was the rash flat or raised to begin with, or was it blistered?

6. Is the area itchy?

7. Does anything seem to make it better?

8. Has your diet changed recently?

9. What treatment have you tried for it?

10. Have you had a fever or any joint pains?

11. Have you had problems with allergies?

12. Are you taking any tablets or medicines? Are any of these new (last 2 weeks)?

13. Have you changed your soap, shampoo, deodorant or washing powder recently?

14. What sort of work do you do? Do you come into contact with chemicals at work or with your hobbies?

15. Have you travelled recently? Where to?

16. Has anyone you know got a similar rash?

17. Have you any other problems with your health?

---

It is important to obtain a past history of rashes or allergic reactions. A past history of asthma, eczema or hay fever suggests atopy. Similarly, evidence of systemic disease in the past may be important in a patient with a rash (e.g. diabetes mellitus, connective tissue disease, inflammatory bowel disease).

A detailed social history needs to be obtained regarding occupation and hobbies, as chemical exposure and contact with animals or plants can all induce dermatitis. All medications that have been taken must be documented. Orally ingested or parenteral medications can cause a whole host of cutaneous lesions and can mimic many skin diseases (Table 15.1). Similarly, a family history of atopic dermatitis, hay fever or skin infestation can be helpful.

**TABLE 15.1** Types of cutaneous drug reactions

| |
|---|
| **1** Acne, e.g. steroids |
| **2** Hair loss (alopecia), e.g. cancer chemotherapy |
| **3** Pigment alterations: hypomelanosis (e.g. hydroxyquinone, chloroquine, topical steroids), hypermelanosis (page 449) |
| **4** Exfoliative dermatitis or erythroderma (page 448) |
| **5** Urticaria (hives), e.g. non-steroidal anti-inflammatory drugs, radiographic dyes, penicillin |
| **6** Maculopapular (morbilliform) eruptions, e.g. ampicillin, allopurinol |
| **7** Photosensitive eruptions, e.g. sulfonamides, sulfonylureas, chlorothiazides, phenothiazines, tetracycline, nalidixic acid, anticonvulsants |
| **8** Drug-induced lupus erythematosus, e.g. procainamide, hydralazine |
| **9** Vasculitis, e.g. propylthiouracil, allopurinol, thiazides, penicillin, phenytoin |
| **10** Skin necrosis, e.g. warfarin |
| **11** Drug-precipitated porphyria, e.g. alcohol, barbiturates, sulfonamides, contraceptive pill |
| **12** Lichenoid eruptions, e.g. gold, antimalarials, beta-blockers |
| **13** Fixed drug eruption, e.g. sulfonamides, tetracycline, phenylbutazone |
| **14** Bullous eruptions, e.g. frusemide, nalidixic acid, penicillamine, clonidine |
| **15** Erythema nodosum or erythema multiforme (page 448) |
| **16** Toxic epidermal necrolysis, e.g. allopurinol, phenytoin |

| |
|---|
| **16** Toxic epidermal necrolysis, e.g. allopurinol, phenytoin, sulfonamides, non-steroidal anti-inflammatory drugs |
| **17** Pruritus (page 445) |

## Examination anatomy

Figure 15.1 shows the three main layers of the skin—epidermis, dermis and subcutaneous fat. These layers can all be involved in skin diseases in varying combinations. For example, most skin tumours arise in the epidermis (Figure 15.2a&b), some bullous eruptions occur at the epidermo-dermal junction, and lipomas are tumours of subcutaneous fat. The skin appendages which include the sweat (eccrine and apocrine) glands, hair follicles (Figure 15.3) and the nails are common sites of infection.

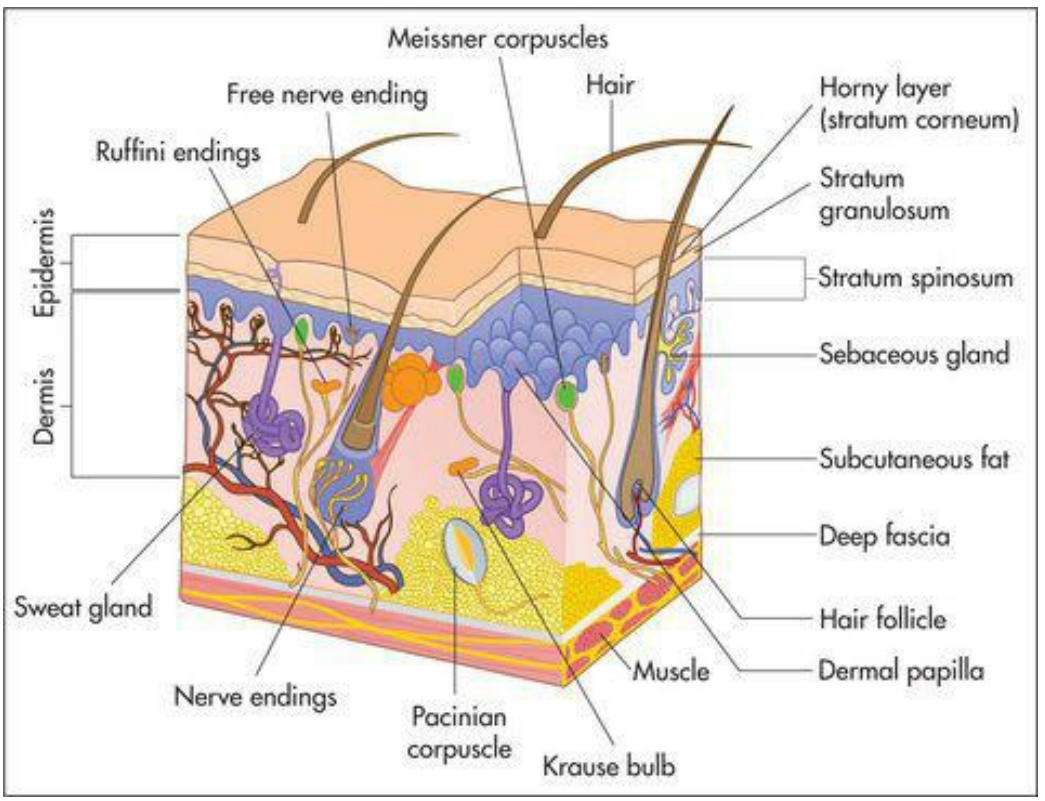



**Figure 15.1** The layers of the skin

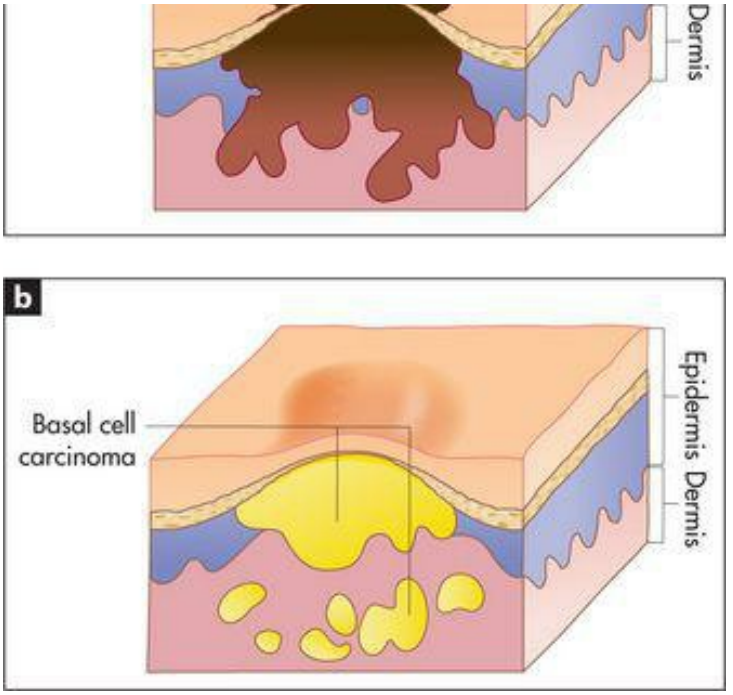



**Figure 15.2** (a) Melanoma; (b) Basal cell carcinoma

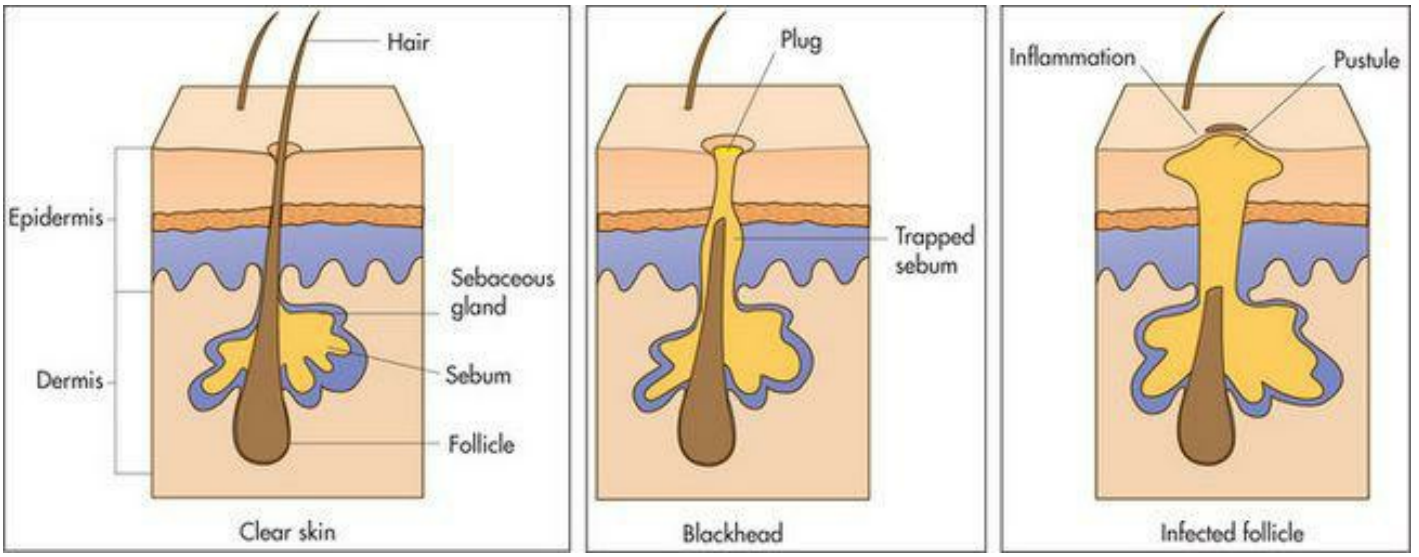



**Figure 15.3** Common sites of infection in the skin

The eccrine glands are present everywhere except in the nail beds and on some mucosal surfaces. They are able to secrete over 5 litres of sweat per day. The apocrine glands are found in association with hair follicles but are confined to certain areas of the body which include the axillae, the pubis, perineum and nipples. They secrete a viscous fluid whose function is unclear in humans.

The nails are formed from heavily keratinised cells that grow from the nail matrix. The matrix grows in a semilunar shape and appears as the lunules

in normal finger and toe nails. Hair is also the product of specialised epithelial cells and grows from the hair matrix within the hair follicle.

## General principles of physical examination of the skin

The aim of this chapter is to provide an approach to the diagnosis of skin diseases.[2,3] Particular emphasis will be placed on cutaneous signs as indications of systemic disease. Other chapters have included the usual clues that can be used to arrive at a particular diagnosis. This chapter tries to unify the concept of 'inspection' as a valuable starting point in the examination of the patient.

Ask the patient to undress. The whole surface of the skin and its appendages should be carefully inspected (Table 15.2).

**TABLE 15.2** Considerations when examining the skin

| |
|---|
| **1** Hair |
| **2** Nails |
| **3** Sebaceous glands—oil-producing and present on the head, neck and back |
| **4** Eccrine glands—sweat-producing and present all over the body |
| **5** Apocrine glands—sweat-producing and present in the axillae and groin |
| **6** Mucosa |

When one is examining actual skin lesions, a number of features should be documented. First, each lesion should be *described* precisely, including colour and shape. Use the appropriate dermatological terminology (Table 15.3), even though this may seem to make dermatological diseases more, rather than less, mysterious. As many dermatological diagnoses are purely descriptive, a good description will often be of considerable help in making the diagnosis. Second, the *distribution* of the lesions should be noted, as certain distributions suggest specific diagnoses. Third, the *pattern* of the

certain distributions suggest specific diagnoses. Third, the *pattern* of the lesions—such as linear, annular (ring-shaped), reticulated (net-like), serpiginous (snake-like) or grouped—also helps establish the diagnosis. Then *palpate* the lesions, noting consistency, tenderness, temperature, depth and mobility. Types of skin lesions are shown in Figure 15.4 and a clinical algorithm for diagnosis is presented in Figure 15.5.

**TABLE 15.3** Dermatological terms

| Term | Definition | Descriptive terms | |
|---|---|---|---|
| Atrophy | Thinning of epidermis with loss of normal skin markings | Annular | Ring-shaped (hollow centre), e.g. tinea infection |
| Bulla | A large collection of fluid below the epidermis | Arcuate | Curved, e.g. secondary syphilis |
| Crust | Dried serum and exudate | Circinate | Circular |
| Ecchymoses | Bruises | Confluent | Lesions that have run together, e.g. measles |
| Excoriations | Lesions caused by scratching that results in loss of the epidermis | Discoid | Circular without a hollow centre, e.g. lupus |
| Keloid | Hypertrophic scarring | Eczematous | Inflamed and crusted, e.g. allergic eczema |
| Macule | A circumscribed alteration of skin colour | Keratotic | Thickened from increased keratin, e.g. psoriasis |
| Nodule | A circumscribed palpable mass, greater than 1 cm diameter | Lichenified | Thickening and roughening of the epidermis associated with accentuated skin markings |
| Papule | A circumscribed palpable elevation, less than 1 cm diameter | Linear | In lines, e.g. contact dermatitis |
| Petechiae | Red, non-blanching spots <5 mm | Nodule | Raised solid lesion >10 mm, e.g. erythema nodosum |
| Pigment alterations | Increased (hyperpigmentation) or decreased (hypopigmentation) | Papule | Raised solid lesion <10 mm, e.g. wart |
| Plaque | A palpable disc-shaped lesion | Papulosquamous | Plaques associated with scaling |
| Purpura | Red, non-blanching spots >5 mm | Reticulated | In a network pattern, e.g. cutaneous parasite |
| Pustule | A visible collection of pus | Serpiginous | Sinuous |
| Scales | An accumulation of excess keratin | Zosteriform | Following a nerve distribution |
| Sclerosis | Induration of subcutaneous tissues, which may involve the dermis | | |
| Ulcer | A circumscribed loss of tissue | | |
| Vesicle | A small collection of fluid below the epidermis | | |
| Wheal | An area of dermal oedema | | |

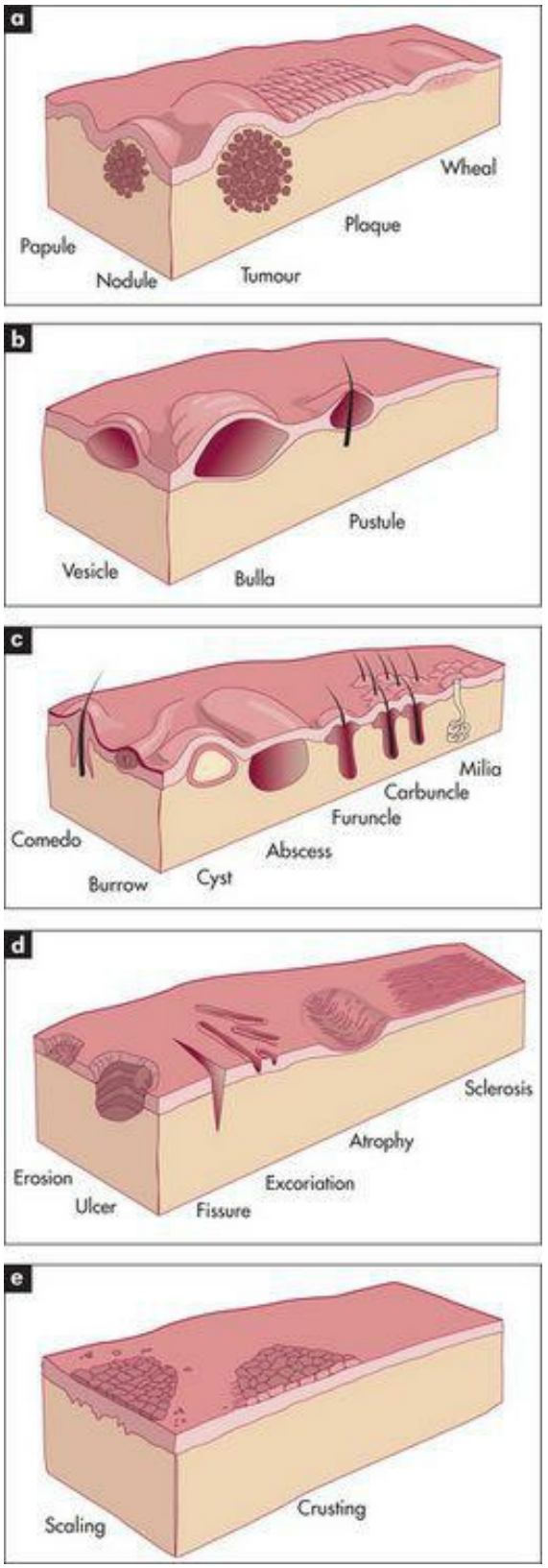



**Figure 15.4** Types of skin lesions
(a) Primary skin lesions, palpable with solid mass.
(b) Primary skin lesions, palpable and fluid-filled.
(c) Special primary skin lesions.

(d) Secondary skin lesions, below the skin plane.
(e) Secondary skin lesions, above the skin plane.

*Adapted from Schwartz M. Textbook of physical diagnosis, 4th edn. Philadelphia: Saunders, 2002.*

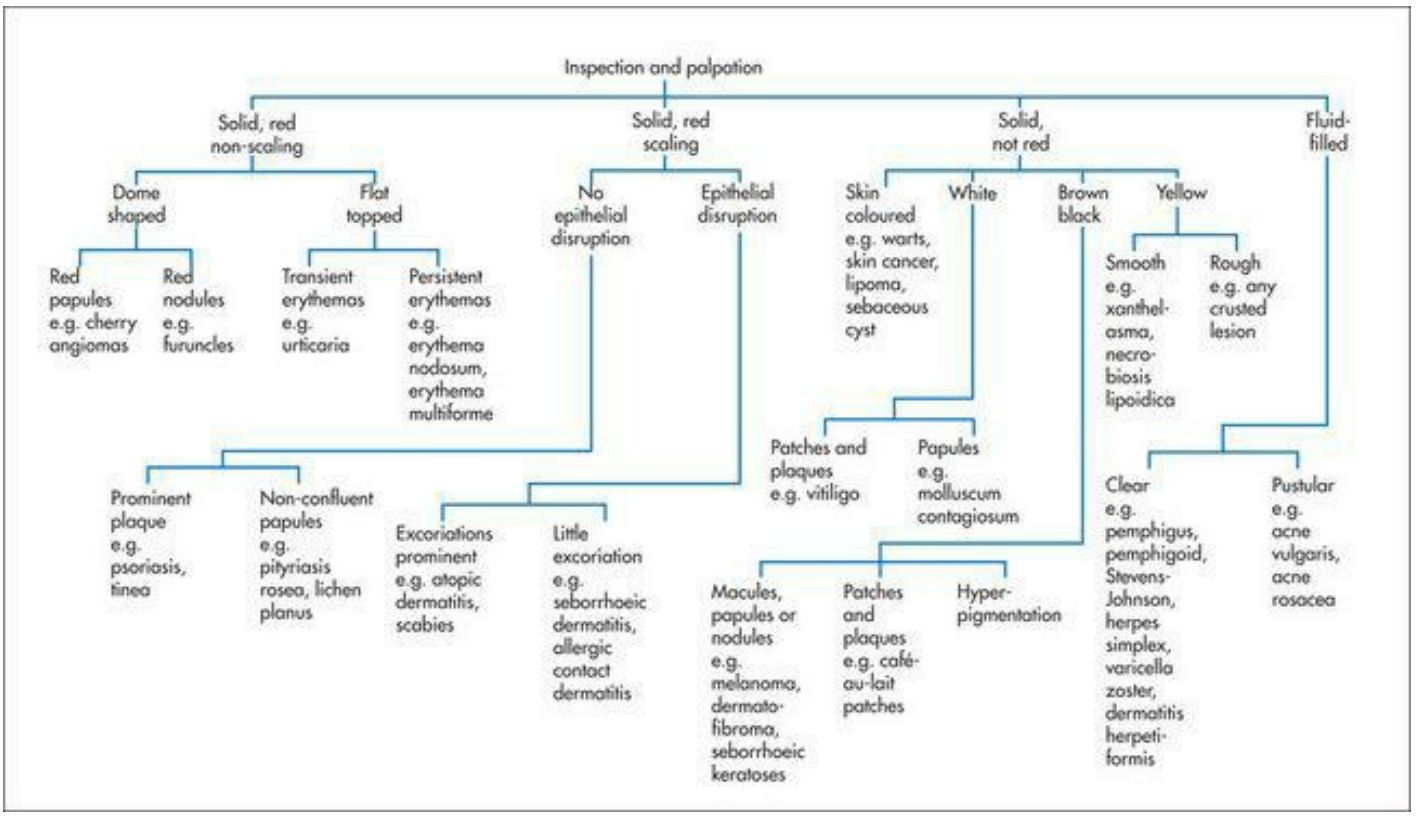



**Figure 15.5** Diagnosis of skin disease: an algorithm

*Adapted from Lynch P.J. Dermatology for the house officer, 2nd edn. Baltimore: Williams & Wilkins, 1987.*

## How to approach the clinical diagnosis of a lump

First, determine the lump's site, size, shape, consistency and tenderness. Next, evaluate in what tissue layer the lump is situated. If it is in the *skin* (e.g. sebaceous cyst, epidermoid cyst, papilloma), it should move when the skin is moved, but if it is in the s*ubcutaneous tissue* (e.g. neurofibroma, lipoma), the skin can be moved over the lump. If it is in the *muscle* or *tendon* (e.g. tumour), then contraction of the muscle or tendon will limit the lump's mobility. If it is in a *nerve*, pressing on the lump may result in pins and needles being felt in the distribution of the nerve, and the lump cannot be moved in the longitudinal axis but can be moved in the transverse axis. If it is in *bone*, the lump will be immobile.

Determine if the lump is *fluctuant* (i.e. contains fluid). Place one forefinger (the 'watch' finger) halfway between the centre and periphery of the lump. The forefinger from the other hand (the 'displacing' finger) is placed diagonally opposite the 'watch' finger at an equal distance from the centre of the lump. Press with the displacing finger and keep the watching finger still. If the lump contains fluid, the watching finger will be displaced in *both* axes of the lump (i.e. fluctuation is present).

Place a small torch behind the lump to determine whether it can be *transilluminated*.

Note any associated signs of *inflammation* (i.e. heat, redness, tenderness and swelling[a]).

Look for similar lumps elsewhere, such as multiple subcutaneous swellings from neurofibromas or lipomas. Neurofibromas are smaller than lipomas. They look hard but are remarkably soft; they occur in neurofibromatosis Type 1 (von Recklinghausen's[b] disease). They continue to increase in number throughout life and are associated with café-au-lait spots and sometimes spinal neurofibromas.

If an inflammatory or neoplastic lump is suspected, remember always to examine the regional lymphatic field and the other lymph node groups.

## Correlation of physical signs and skin disease

There are many different skin diseases with varied physical signs. With each major sign the groups of common important diseases that should be considered will be listed.

### Pruritus

Pruritus simply means itching. It may be either generalised or localised. Scratch marks are usually present. Localised pruritus is usually caused by a dermatological condition such as dermatitis or eczema. Generalised pruritus may be caused by primary skin disease, systemic disease or psychogenic factors.

To determine the cause of the pruritus it is essential to examine the skin in detail (Table 15.4). Excoriations are caused by scratching, regardless of the underlying cause. Specific features of cutaneous diseases such as dermatitis, scabies (Figure 15.6) or the blisters of dermatitis herpetiformis should be looked for.

**TABLE 15.4** Primary skin disorders causing pruritus

| |
|---|
| **1** Asteatosis (dry skin) |
| **2** Atopic dermatitis (erythematous, oedematous papular patches on head, neck, flexural surfaces) |
| **3** Urticaria |
| **4** Scabies |
| **5** Dermatitis herpetiformis |

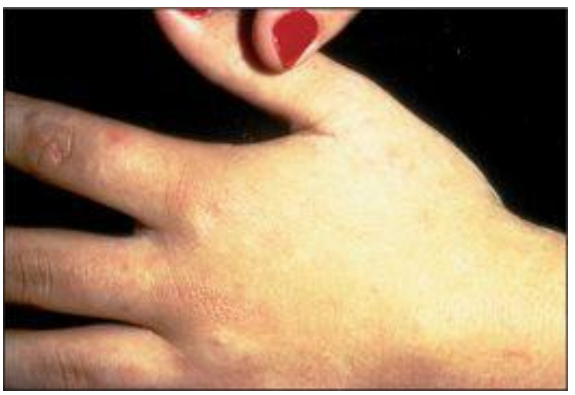


**Figure 15.6** Scabies
Scattered fine papules with severe itching. Finger web involvement is common.

When primary skin diseases have been excluded, a detailed history and examination should be undertaken to consider the various systemic diseases listed in Table 15.5.

**TABLE 15.5** Systemic conditions causing pruritus

| |
|---|
| **1** Cholestasis, e.g. primary biliary cirrhosis |
| **2** Chronic renal failure |

| |
|---|
| **2** Chronic renal failure |
| **3** Pregnancy |
| **4** Lymphoma and other internal malignancies |
| **5** Iron deficiency, polycythaemia rubra vera |
| **6** Endocrine diseases, e.g. diabetes mellitus, hypothyroidism, hyperthyroidism, carcinoid syndrome |

## Erythrosquamous eruptions

Erythrosquamous eruptions are made up of lesions that are red and scaly. They may be well demarcated or have diffuse borders. They may be itchy or asymptomatic.

When one is attempting to establish a diagnosis of an erythrosquamous eruption, the history is very important. First ask about the time course of the eruption, about a family history of similar skin diseases and whether or not there is a family history of atopy.

The presence or absence of itching and the distribution of the lesions (often on the extensor surfaces of the limbs) also give clues about the diagnosis.

Asymptomatic lesions on the palms and soles are suggestive of secondary syphilis, whereas itchy lesions in the same location would be more suggestive of lichen planus (Figures 15.7 and 15.8). Lichen planus is occasionally associated with primary biliary cirrhosis and other liver diseases, chronic graft-versus-host disease and drugs (e.g. gold, penicillamine). Scattered lesions of recent origin on the trunk would be more suggestive of pityriasis rosea (Figure 15.9), whereas more widespread, diffuse and intensely itchy lesions would be more suggestive of nummular eczema (Figure 15.10) (Table 15.6).

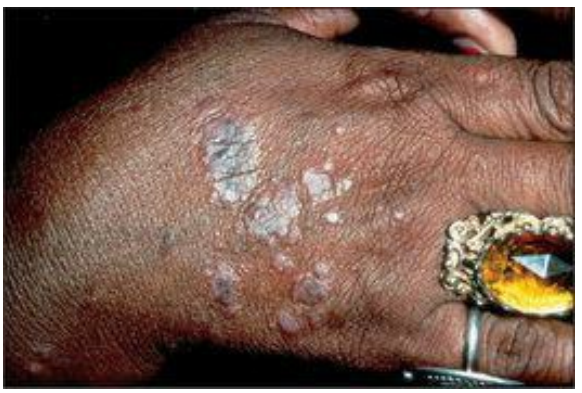


**Figure 15.7** Lichen planus

**Figure 15.7** Lichen planus
With polygonal flat-topped violaceous lesions.

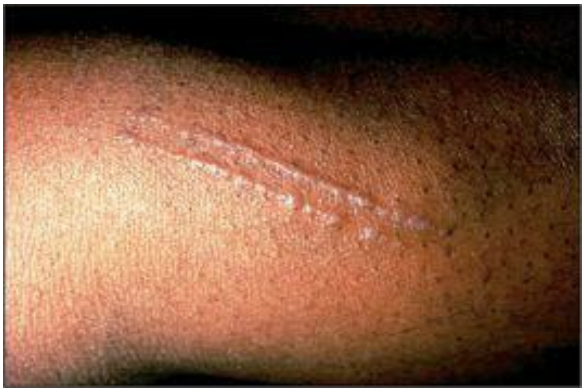


**Figure 15.8** Lichen planus
With development of lesions in an area of trauma—the 'Koebner' phenomenon.

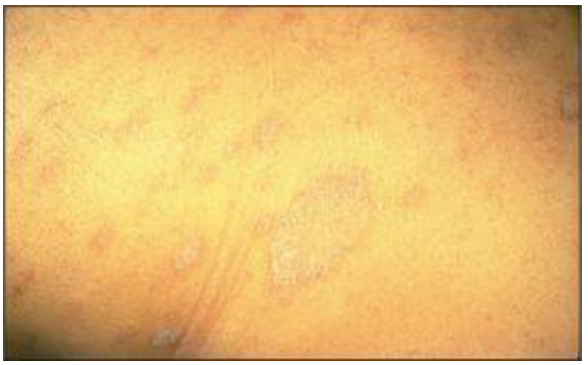
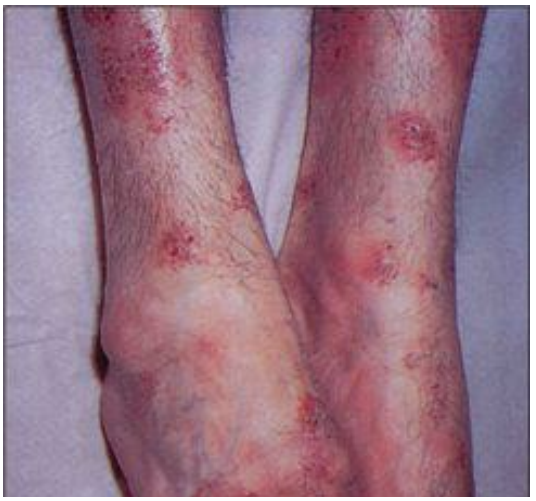


**Figure 15.9** Pityriasis rosea
With scattered scaly oval lesions on the trunk and a larger 'herald' patch.



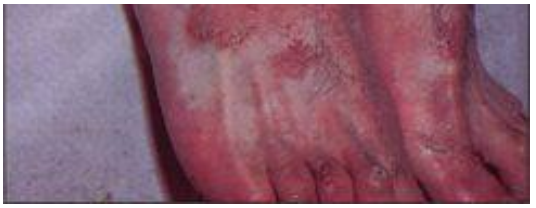
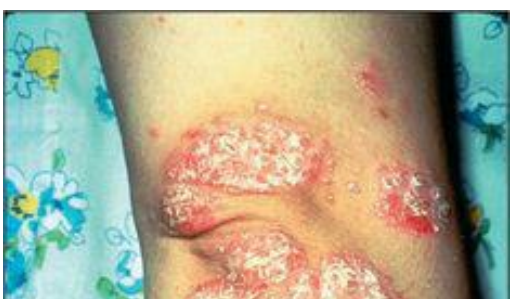


**Figure 15.10** Nummular eczema
Typical scattered coin-like lesions of indolent dermatitis.

**TABLE 15.6** Causes of erythrosquamous eruptions

| |
|---|
| **1** Psoriasis (bright pink plaques with silvery scale) |
| **2** Atopic eczema (diffuse erythema with fine scaling) |
| **3** Pityriasis rosea (paler pink, scaly, macular lesions in a Christmas tree pattern; herald patch; self-limited) |
| **4** Nummular eczema (round patches of subacute dermatitis) |
| **5** Contact dermatitis (irritant or allergic) |
| **6** Dermatophyte infections (ringworm) |
| **7** Lichen planus (violet-coloured, small, polygonal papules) |
| **8** Secondary syphilis (flat, red, hyperkeratotic lesions) |

Scaly lesions with a well-demarcated edge over the extensor surfaces are usually due to psoriasis (Figures 15.11 and 15.12).



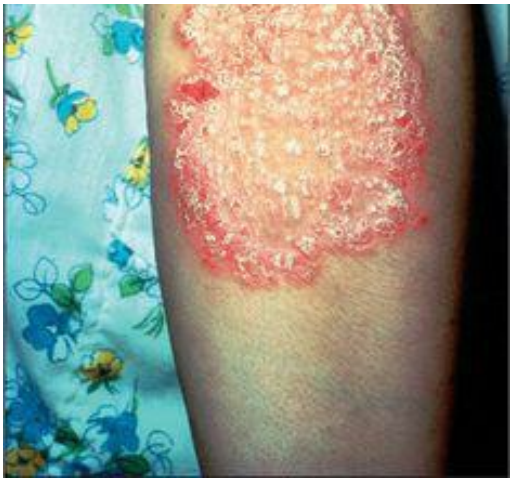


**Figure 15.11** Psoriasis
Typical bright red, scaly plaque with silvery scale over a joint.

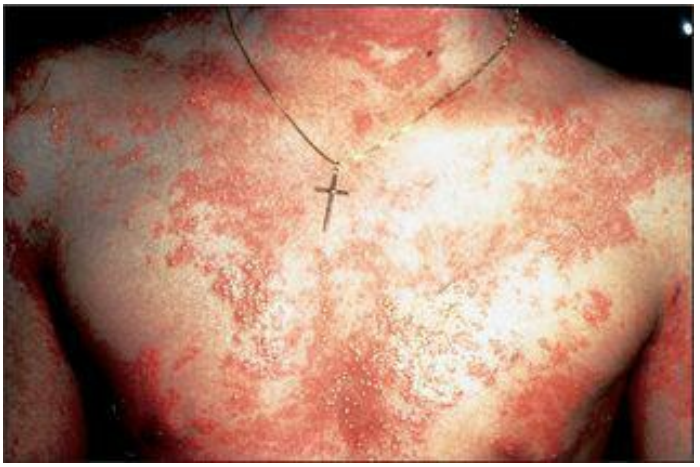


**Figure 15.12** Acute widespread pustular psoriasis
Often the eruption is bright red with bizarre patterns and pustules predominantly at the margins.

## Blistering eruptions

There are a number of different diseases which will present with either vesicles or blisters (Table 15.7). Dermatitis can present as a blistering eruption, particularly acute contact dermatitis (Figure 15.13). See *Questions box 15.2*.

**TABLE 15.7** Causes of blistering eruptions

| |
|---|
| **1** Traumatic blisters and burns |
| **2** Bullous impetigo |
| **3** Viral blisters (e.g. herpes simplex, varicella) |
| **4** Bullous erythema multiforme |
| **5** Bullous pemphigoid |
| **6** Dermatitis herpetiformis |
| **7** Pemphigus |
| **8** Porphyria |
| **9** Epidermolysis bullosa |
| **10** Dermatophyte infections |
| **11** Acute contact dermatitis |

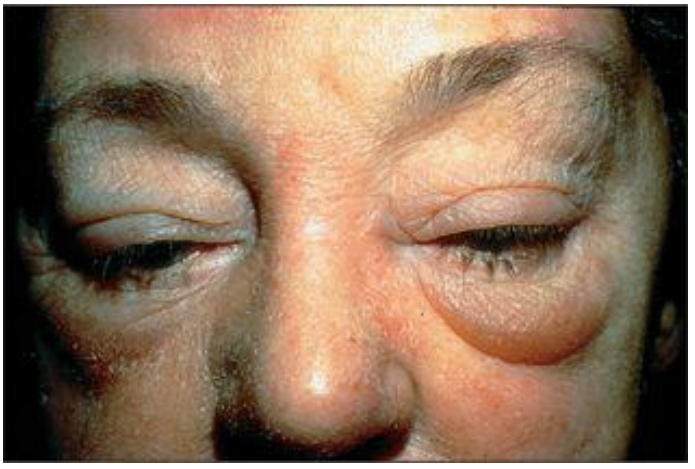


**Figure 15.13** Allergic contact dermatitis
From over-the-counter topical medication rubbed over congested sinuses.

**Questions box 15.2**

## Questions to ask the patient with a blistering eruption

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. Have you had blisters on the backs of your hands which break easily and are worse if you have been in the sun?—Porphyria cutanea tarda

2. Have you had sores or blisters in your mouth that came on before the skin blisters?—Pemphigus vulgaris

3. Did the inside of your mouth become ulcerated and painful suddenly?—Stevens-Johnson syndrome

4. Was the blister on your lip or genitals, and was it preceded by itching or burning?—Herpes simplex

5. Were the blisters preceded by some days of severe pain and burning in the areas where the blisters have broken out?—Herpes zoster

6. Did you notice pink spots on the skin that were itchy before the blisters appeared?—Bullous pemphigoid

## Clinical features of bullous eruptions

**Viral blisters** such as those of herpes simplex virus infection (Figure 15.14) have a distinctive morphology (grouped vesicles on an erythematous background).

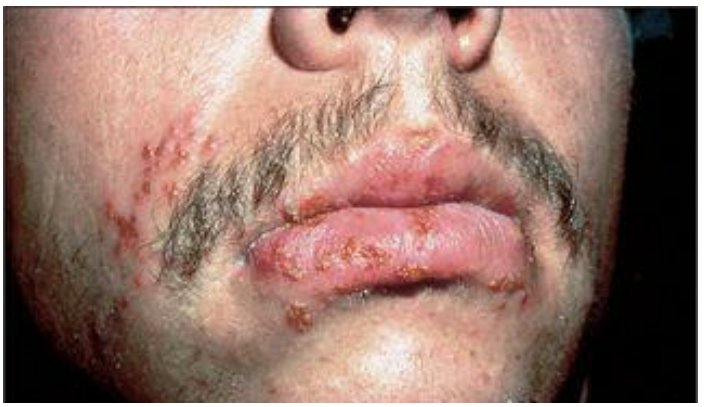


**Figure 15.14** Primary herpes simplex virus infection in an adult
Shows typical widespread distribution around the mouth.

**Bullous pemphigoid** is a rare disease usually affecting older patients. Blisters are widespread, have a thick roof and tend not to rupture easily.

**Pemphigus vulgaris** is much more severe. It has thin-roofed blisters that readily rupture and form crusts. The affected superficial skin can be moved over the deeper layer (Nikolsky's[c] sign). Oral ulcers are common.

**Dermatitis herpetiformis** is characterised by a very itchy widespread vesicular or bullous eruption.

**Porphyria cutanea tarda** is characterised by clear or haemorrhagic tense blisters on the hands and other sun-exposed areas, hyperpigmentation and increased facial hair; many patients have hepatitis C and alcohol can induce symptoms (due to decreased uroporphyrinogen decarboxylase).

## Erythroderma

Erythroderma is best thought of as the end-stage of numerous skin conditions (Table 15.8). The erythrodermic patient has involvement of nearly all the skin with an erythematous inflammatory process, often with exfoliation. There is usually associated oedema and loss of muscle mass. This represents that most unusual occurrence, a dermatological emergency.

**TABLE 15.8** Causes of erythroderma

| |
|---|
| **1** Eczema |
| **2** Psoriasis |
| **3** Drugs, e.g. phenytoin, allopurinol |
| **4** Pityriasis rubra pilaris |

| |
|---|
| **5** Mycosis fungoides, leukaemia, lymphoma |
| **6** Lichen planus |
| 7 Pemphigus foliaceus |
| **8** Hereditary disorders |
| **9** Dermatophytosis |

An attempt should be made to determine the underlying cause of the erythroderma, and this is best done based on history and examination. Specific treatment can then be directed at the underlying cause. Some patients with erythroderma will develop profound metabolic changes (including hypoalbuminaemia and extrarenal water loss), and these patients require constant supervision and monitoring until they have recovered from the acute phase of their illness.

The most common cause is eczema, which is usually of the atopic variety. These patients often have an intense pruritus. Some of them will develop a chronic unremitting erythroderma.

## Pustular and crusted lesions

The clinical appearance of a *pustular* lesion results from accumulation of neutrophils. Such collections usually indicate an infective process; however, sterile pustules may form as part of a number of skin diseases due to the release of chemotactic factors following an immunological reaction.

A *crust* is a yellowish crystalline material that is found on the skin; it is made up of desiccated serum.

It is essential to determine whether or not a pustular lesion (or a group of pustular lesions) represents a primarily infectious process or an inflammatory dermatological condition. For example, pustular lesions on the hands and feet may either be due to tinea infection or be a primary pustular psoriasis or palmoplantar pustulosis (Table 15.9). See *Questions box 15.3*.

**TABLE 15.9** Causes of pustular and crusted lesions

| |
|---|
| **1** Acne vulgaris (comedones, papules, pustules, cystic lesions, ice pick scars—no telangiectasiae) |
| **2** Acne rosacea (acne-like lesions, erythema and telangiectasia on central face) |
| **3** Impetigo |
| **4** Folliculitis |
| **5** Viral lesions |
| **6** Pustular psoriasis |
| **7** Drug eruptions |
| **8** Dermatophyte infections |

**Questions box 15.3**

### Questions to ask the patient with pustular lesions

1. Are you taking cortisone tablets?—Steroid acne

2. Has the skin been painful or have you had a fever?—Pustular psoriasis

3. Have you had psoriasis in the past?

4. Do you find your face becomes flushed easily, for example if you drink hot drinks?—Acne rosacea

5. Are you a diabetic?—Cutaneous candidiasis

6. Do you sweat excessively?—Folliculitis

## Dermal plaques

Plaques are localised thickenings of the skin which are usually caused by changes in the dermis or subcutaneous fat. These may be due to chronic inflammatory processes or scarring sclerotic processes (Table 15.10).

**TABLE 15.10** Causes of dermal plaques

| |
|---|
| **1** Granuloma annulare |
| **2** Necrobiosis lipoidica |
| **3** Sarcoidosis |
| **4** Erythema nodosum |
| **5** Lupus erythematosus |
| **6** Morphoea and scleroderma |
| **7** Tuberculosis |
| **8** Leprosy |

The pattern of involvement of the plaques, the age of the patient and other clinical features should enable a diagnosis to be established.

In *Sweet's syndrome* there are painful red plaques and a high fever (acute febrile neutrophilic dermatosis); 10% have leukaemia.

## Erythema nodosum

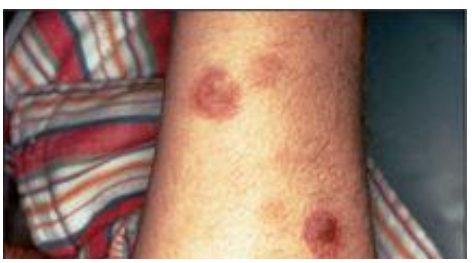
This is the best known of the group of diseases classified as nodular vasculitis. The lesions of erythema nodosum are usually found below the knee in the pretibial area and are erythematous, palpable and tender (Figure 6.36, page 191). There may be an associated fever (Table 15.11). Sarcoidosis is a common cause, but the skin changes in sarcoid can mimic almost any skin disease (except vesicles).

**TABLE 15.11** Causes of erythema nodosum

| |
|---|
| **1** Sarcoidosis |
| **2** Streptococcal infections (β-haemolytic) |
| **3** Inflammatory bowel disease |
| **4** Drugs, e.g. sulfonamides, penicillin, sulfonylurea, oestrogen, iodides, bromides |
| **5** Tuberculosis |
| **6** Other infections, e.g. lepromatous leprosy, toxoplasmosis, histoplasmosis, *Yersinia*, *Chlamydia* |
| **7** Systemic lupus erythematosus |
| **8** Behçet's syndrome |

### Erythema multiforme

This is a distinctive inflammatory reaction of skin and mucosa. It is not a systemic disease. Characteristic discrete target lesions occur, particularly on the distal extremities (Figure 15.15). The periphery of these lesions is red, whereas the centre becomes bluish or even purpuric. The lesions can become bullous, and severe cases of this syndrome involve widespread desquamation of the mucosal surfaces (the Stevens-Johnson[d] syndrome). In many cases the condition is precipitated by clinical or subclinical herpes simplex virus infection. Other causes include *Mycoplasma pneumoniae*, histoplasmosis, malignancy, sarcoidosis and drugs (including those that can cause toxic epidermal necrolysis). Sometimes no underlying cause of the erythema multiforme will be established.



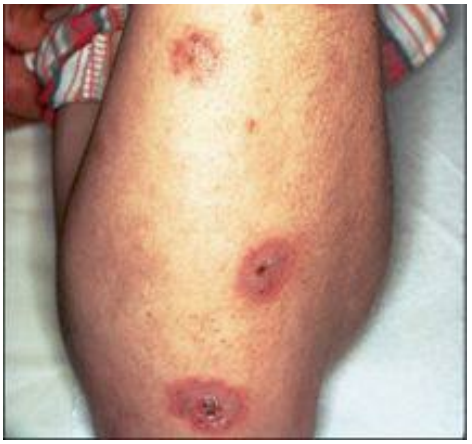


**Figure 15.15** Erythema multiforme
Shows classic iris or target lesions, secondary to herpes simplex virus infection of the lips.

*Toxic epidermal necrolysis*, on the other hand, is a systemic condition and usually secondary to a drug reaction. It results in a peeling of large skin areas. The major causes include penicillin, sulfonamides, phenytoin and non-steroidal anti-inflammatory drugs.

## Hyperpigmentation

The presence of hyperpigmentation can be a clue to underlying systemic disease (Table 15.12).

**TABLE 15.12** Causes of diffuse hyperpigmentation

| **Endocrine disease** |
| --- |
| Addison's disease (excess ACTH) |

| |
|---|
| Addison's disease (excess ACTH) |
| Ectopic ACTH secretion (e.g. carcinoma) |
| The contraceptive pill or pregnancy |
| Thyrotoxicosis, acromegaly, phaeochromocytoma |
| **Metabolic** |
| Malabsorption or malnutrition |
| Liver diseases, e.g. haemochromatosis, primary biliary cirrhosis, Wilson's disease |
| Chronic renal failure |
| Porphyria |
| Chronic infection, e.g. bacterial endocarditis |
| Connective tissue disease, e.g. systemic lupus, scleroderma, dermatomyositis |
| **Racial or genetic** |
| **Other** |
| Drugs, e.g. chlorpromazine, busulphan, arsenicals |
| Radiation |

ACTH = adrenocorticotrophic hormone.

## Flushing and sweating

Flushing of the skin may sometimes be observed, especially on the face, by the examiner. Some of the causes of this phenomenon are presented in Table 15.13.

**TABLE 15.13** Causes of facial flushing

| |
|---|
| **1** Menopause |
| **2** Drugs and foods, e.g. nifedipine, monosodium glutamate (MSG) |
| **3** Alcohol after taking the drug disulfiram (or alcohol alone in some people) |
| **4** Systemic mastocytosis |
| **5** Rosacea |
| **6** Carcinoid syndrome (secretion of serotonin and other mediators by a tumour may produce flushing, diarrhoea and valvular heart disease) |
| **7** Autonomic dysfunction |
| **8** Medullary carcinoma of the thyroid |

Excessive sweating (hyperhidrosis) can occur with thyrotoxicosis, phaeochromocytoma, acromegaly, hypoglycaemia, autonomic dysfunction, stress, fever and menopause.

### Skin tumours

Skin tumours are very common and are usually benign (Table 15.14).[4] Most malignant skin tumours can be cured if they are detected early and treated appropriately (Table 15.15).

**TABLE 15.14** Benign skin tumours

| |
|---|
| **1** Warts |
| **2** Molluscum contagiosum |
| **3** Sebaceous keratosis |

| |
|---|
| **3** Seborrhoeic keratoses |
| **4** Dermatofibroma |
| **5** Neurofibroma |
| **6** Angioma |
| **7** Xanthoma |

**TABLE 15.15** Malignant skin tumours

| |
|---|
| **1** Basal cell carcinoma |
| **2** Squamous cell carcinoma |
| **3** Bowen's* disease (squamous cell carcinoma confined to the epithelial layer of the skin—carcinoma in situ) |
| **4** Malignant melanoma |
| **5** Secondary deposits |

* John Templeton Bowen (1857–1941), Boston dermatologist.

Skin cancer often occurs in those predisposed individuals (with the fair skin of Celtic or Northern European origin) who undergo chronic exposure to ultraviolet light.

Skin cancers may present as flat scaly lesions or as raised scaly or smooth lesions. They may be large or small and they may eventually ulcerate. All non-healing ulcers should be considered to be skin cancer, until proven otherwise.

The earliest lesions are actinic (solar) keratoses, which are pink macules or papules surmounted by adherent scale (Figure 15.16). Basal cell carcinoma is characteristically a translucent papule with a depressed centre and a rolled border with ectatic capillaries (Figure 15.17). Squamous cell carcinoma is typically an opaque papule or plaque which is often eroded or scaly (Figure 15.18).

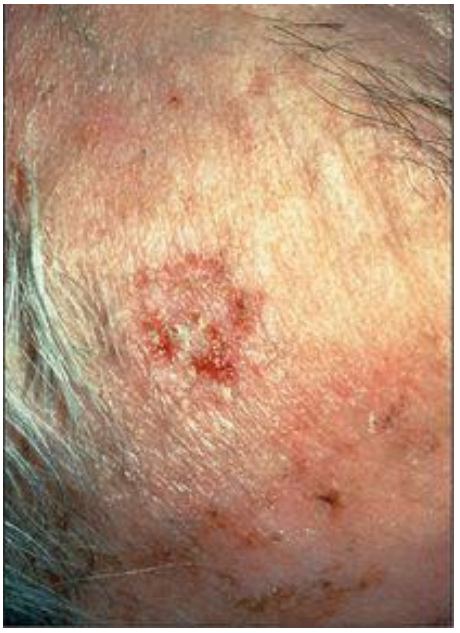


**Figure 15.16** Actinic keratosis, slightly eroded and scaly
Higher on the forehead additional granular keratosis could be easily palpated.

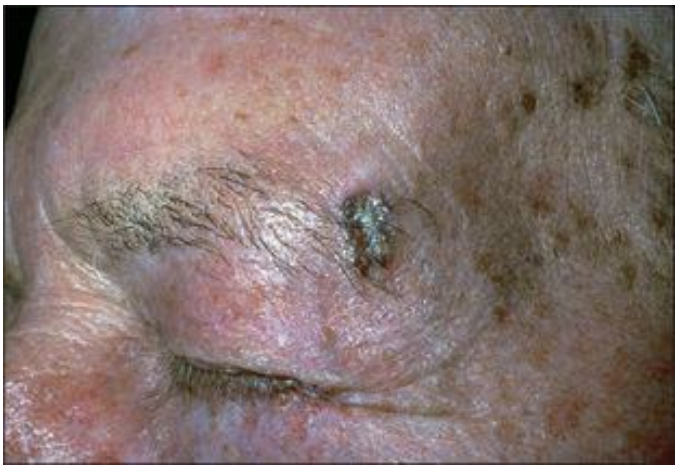
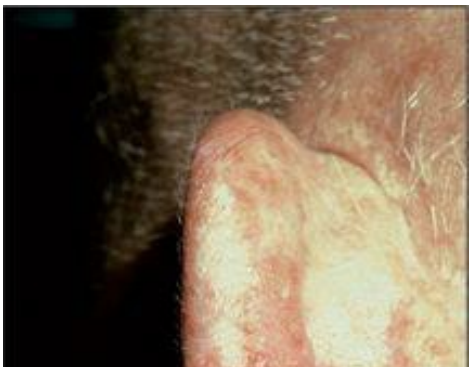


**Figure 15.17** Pigmented basal cell carcinoma
With pearly quality and depressed centre, in a patient with sun-damaged skin.



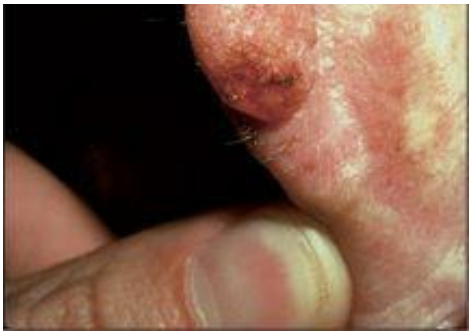


**Figure 15.18** Squamous cell carcinoma

Malignant melanomas are usually deeply pigmented lesions that are enlarging and have an irregular notched border (Figure 15.19). There is often variation of pigment within the lesion. Malignant melanoma is likely if the lesion is ***A****symmetrical*, has an irregular ***B****order*, has an irregular ***C****olour* and is large (***D****iameter* >6 mm) and may be ***E****levated*, referred to as the ABCDE checklist.[5,6] Patients with numerous large and unusual pigmented naevi (dysplastic naevus syndrome) are at an increased risk of developing malignant melanoma.

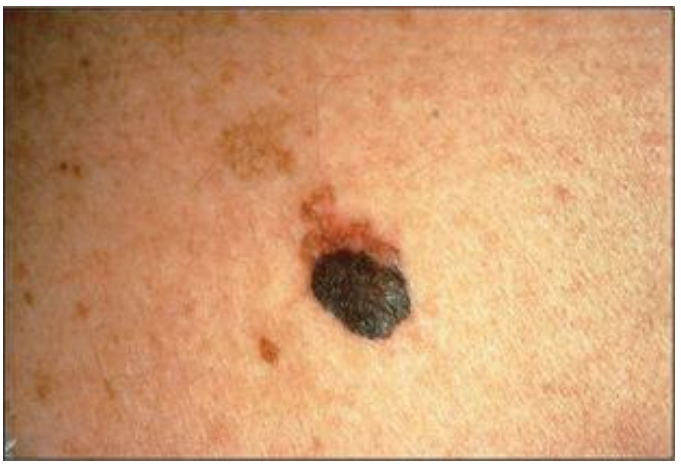


**Figure 15.19** Superficial spreading melanoma, still confined to the upper dermis

## The nails

Systemic disease is commonly associated with changes in the patient's finger (and toe) nails and in the nail beds. The slow growth of the nails means that the temporal course of an illness may be seen in nail changes. Many of these findings have been described in other chapters but important features of nail changes are dealt with here.

Fungal infection of the nails (onychomycosis) (Figure 15.20) is their

most common abnormality. It makes up 40% of all nail disorders and 30% of all cutaneous fungal infections. The characteristic findings are pitting, thickening, ridging and deformity. The changes can be indistinguishable from those of psoriasis. Candidal nail infections are less common than those due to dermatophytes. Candidal nail infection (diagnosed by microscopy and culture) suggests the possibility of chronic mucocutaneous candidiasis, which is a rare condition associated with polyendocrinopathies.

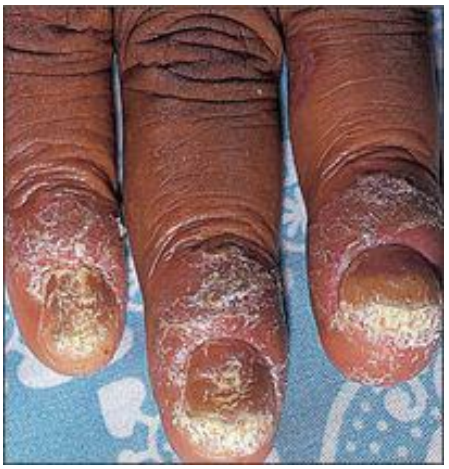


**Figure 15.20** Onychomycosis: fungal infection of the nails

Nail involvement occurs in about 25% of patients with psoriasis (Figure 15.21). The characteristic abnormality is pitting. This can also occur in fungal infections, chronic paronychia, lichen planus and alopecia areata. Psoriasis is also the most common cause of onycholysis. Rarer changes in psoriatic nails include longitudinal ridging (onychorrhexis), proximal transverse ridging, subungual hyper-keratosis and yellow-brown discoloration.

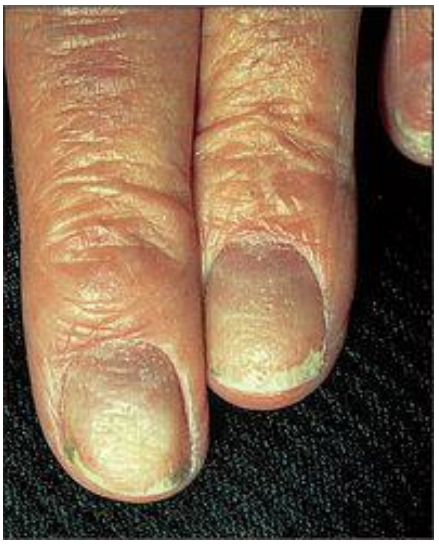


**Figure 15.21** Nail involvement occurs in about 25% of patients with psoriasis

Nailfold telangiectasia is an important sign in a number of systemic disorders, including systemic lupus erythematosus, scleroderma and Raynaud's phenomenon. These changes are not very specific and considerable variation in nailfold capillary shape is present in normal people. In patients with dermatomyositis, nailfold telangiectasiae are associated with hypertrophy of the cuticle and small haemorrhagic infarcts.

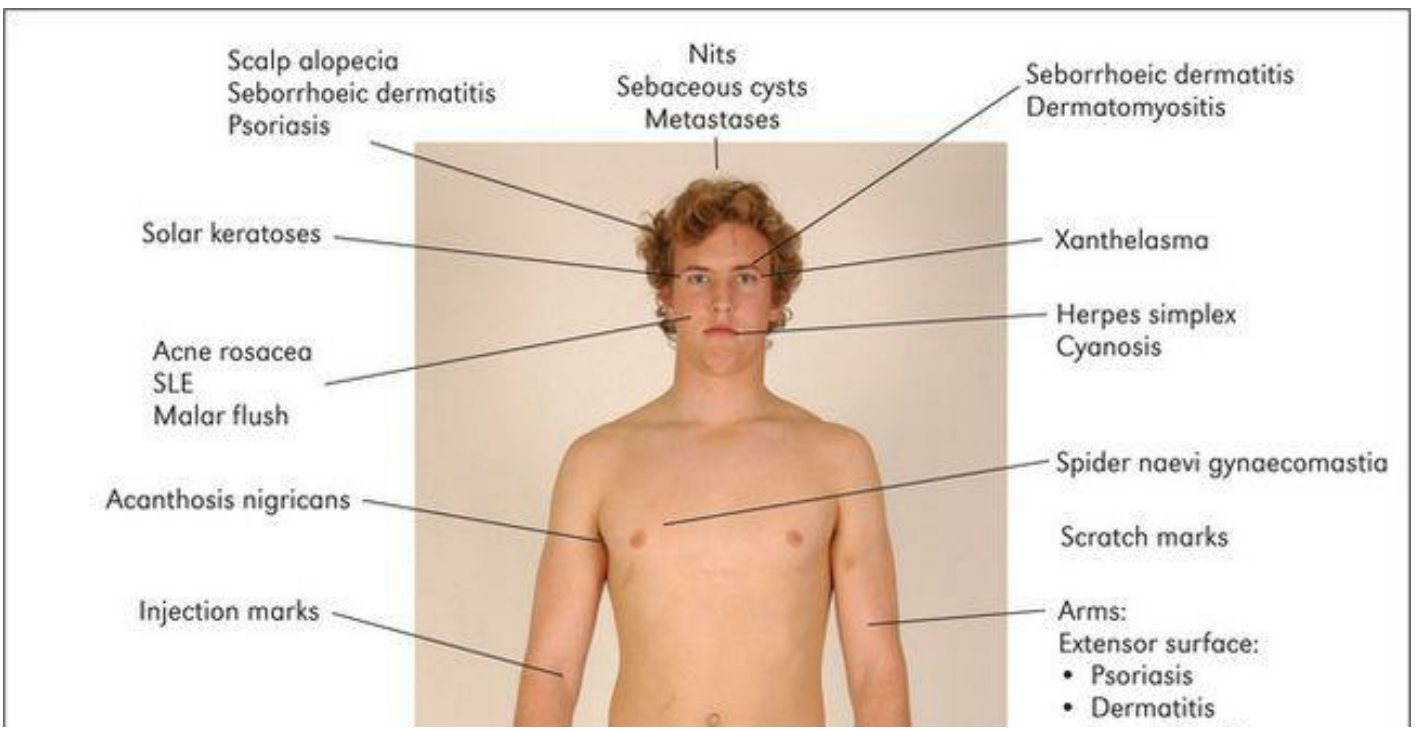
Raynaud's is also associated with nail changes caused by the inadequate blood supply. These include brittleness, longitudinal ridging, splitting, flattening, onycholysis, koilonychia and a redder than normal nail bed.

Clubbing is an important nail abnormality. It has also been described in patients with HIV infection, and its severity seems proportional to the degree of immunosuppression. HIV infection is also associated with onychomycosis and longitudinal melonychia (dark line in the nail), secondary to treatment with zidovudine.

## Summary

### The dermatological examination in internal medicine: a suggested method

(Figure 15.22)



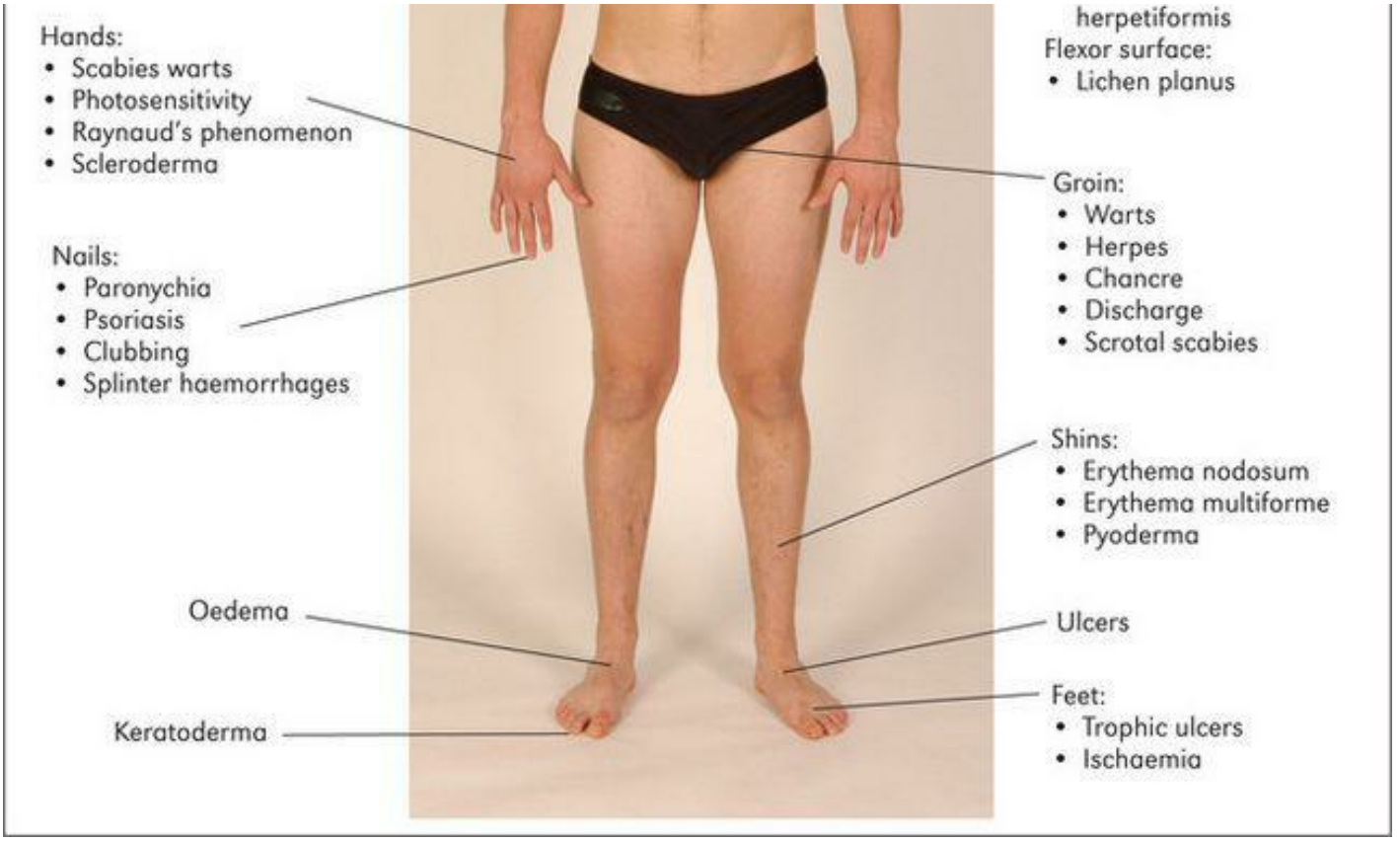



**Figure 15.22 Sites of some important skin lesions of the limbs, face and trunk** SLE = systemic lupus erythematosus.

Even if the patient shows the examiner only a small single area of abnormality, proceed to examine all the skin.

After obtaining good lighting conditions and asking the patient to disrobe, begin by looking at the **nails and hands**. Paronychia is an infection of the skin surrounding the nails. Other changes to note include pitting (psoriasis, fungal infections) and onycholysis (e.g. thyrotoxicosis, psoriasis). Dark staining under the nail may indicate a subungual melanoma. Linear splinter haemorrhages (e.g. vasculitis) or telangiectasiae (e.g. systemic lupus erythematosus) may be seen in the nail bed.

A purplish discoloration in streaks over the knuckles may indicate dermatomyositis. Also look at the backs of the hands and forearms for the characteristic blisters of porphyria, which occur on the exposed skin. Papules and scratch marks on the backs of the hands, between the fingers and around the wrists may indicate scabies. Viral warts are common on the hands.

Look at the palms for Dupuytren's contracture, pigmented flat junctional moles (which have a high risk of becoming malignant) and xanthomata in the palmar creases.

Next look at the **forearms**, where lichen planus may occur on the flexor surfaces (characterised by small shiny, purple-coloured papules) and psoriasis may be present on the extensor surfaces. Palpable purpura—raised bruising

that indicates bleeding into the skin—may be seen on the arms, and indicates vasculitis. Acanthosis nigricans can occur in the axillae.

Inspect the patient's **hair and scalp**. Decide whether or not the hair is dry and whether the distribution is normal. Alopecia may indicate male pattern baldness, recent severe illness, hypothyroidism or thyrotoxicosis. Patches of alopecia occur in the disease alopecia areata. Short broken-off hairs occur typically in systemic lupus erythematosus. In psoriasis there are silvery scales, which may be seen on the skin of the scalp. Metastatic deposits may rarely be felt as firm nodules within the skin of the scalp. Sebaceous cysts are common. The unfortunate examiner may find nits sticking to the head hairs.

Move down now to the **eyebrows** and look for scaling and greasiness, which are found in seborrhoeic dermatitis. A purplish erythema occurs around the eyelids in dermatomyositis. Xanthelasmata are seen near the eyelid.

Look at the **face** for rosacea, which causes bright erythema of the nose, cheeks, forehead and chin, and occasionally pustules and rhinophyma (disfiguring swelling of the nose). Acne causes papules, pustules and scars involving the face, neck and upper trunk. The butterfly rash of systemic lupus erythematosus occurs across the cheeks but is rare. Spider naevi may be present. Ulcerating lesions on the face may include basal cell carcinoma, squamous cell carcinoma or rarely tuberculosis (lupus vulgaris).

Benign tumours of the face include keratoacanthoma (a volcano-like lesion from a sebaceous gland) and congenital haemangiomas.

Look for the blisters of herpes zoster, which may occur strictly in the distribution of one of the divisions of the trigeminal nerve.

Inspect the **neck**, which is prone to many of the lesions that occur on the face. Rarely, the redundant loose skin of pseudoxanthoma elasticum will be seen around the neck.

Go on to inspect the **trunk**, where any of the childhood exanthems produce their characteristic rashes. Look for spider naevi. Campbell de Morgan spots are commonly found on the abdomen (and chest), as are flat, greasy, yellow-coloured seborrhoeic warts. Erythema marginatum (rheumatic fever) occurs on the chest and abdomen. Herpes zoster may be seen overlying any of the dermatome distributions.

Metastases from internal malignancies may rarely occur anywhere on the skin. Neurofibromas are soft flesh-coloured tumours; when associated with more than five 'café-au-lait' spots (brownish, irregular lesions), they suggest neurofibromatosis (von Recklinghausen's disease). Pigmented moles are seen on the trunk and evidence of malignancy must be looked for with these. The patient's buttocks and sacrum must be examined for bedsores, and the abdomen and thighs may have areas of fat atrophy or hypertrophy from insulin injections.

from insulin injections.

Go to the **legs**, where erythema nodosum or erythema multiforme may be seen on the shins. Necrobiosis lipoidica diabeticorum affects the skin over the tibia in diabetics. Pretibial myxoedema also occurs over the shins. Look for ulcers on either side of the lower part of the leg. Livedo reticularis is a net-like, red reticular rash that occurs in vasculitis, the anti-phospholipid syndrome and with atheroembolism.

Inspect the **feet** for the characteristic lesion of Reiter's disease called keratoderma blennorrhagica, where crusted lesions spread across the sole because of the fusion of vesicles and pustules. Look at the foot for signs of ischaemia, associated with wasting of the skin and skin appendages. Trophic ulcers may be seen in patients with peripheral neuropathy (e.g. diabetes mellitus). Always separate the toes to look for melanomas.

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[a] These four cardinal signs were described by Celsus in the 8th volume of his medical book which taught those who were interested surgical techniques. After performing surgery readers were warned to look out for the four cardinal signs of post-surgical inflammation—*calor, rubor, dolor* and *tumor*. Modern surgeons have added *loss of function* to these signs.

[b] Frederich von Recklinghausen (1833–1910). He was Virchow's assistant in Berlin and then professor of pathology in Strasbourg from 1872. He described this disease in 1882 and haemochromatosis in 1889.

[c] Pyotr Vasilyevich Nikolsky (1855–1940), Kiev and Warsaw dermatologist. Nikolsky's sign also occurs in staphylococcal scalded skin syndrome and toxic epidermal necrolysis.

[d] Albert Mason Stevens (1884–1945), New York paediatrician, and Frank C Johnson (1894–1934), American physician.

# Chapter 16

## A system for the infectious diseases examination

> As it takes two to make a quarrel, so it takes two to make a disease, the microbe and its host.
> *Charles Chaplain (1856–1941)*

We have selected two important presentations to be covered in this chapter to show how infectious diseases can be approached in a systematic manner.

### Pyrexia of unknown origin (PUO)

This condition is defined as documented fever (>38 °C) of more than 3 weeks' duration, where no cause is found despite basic investigations.[1,2] The