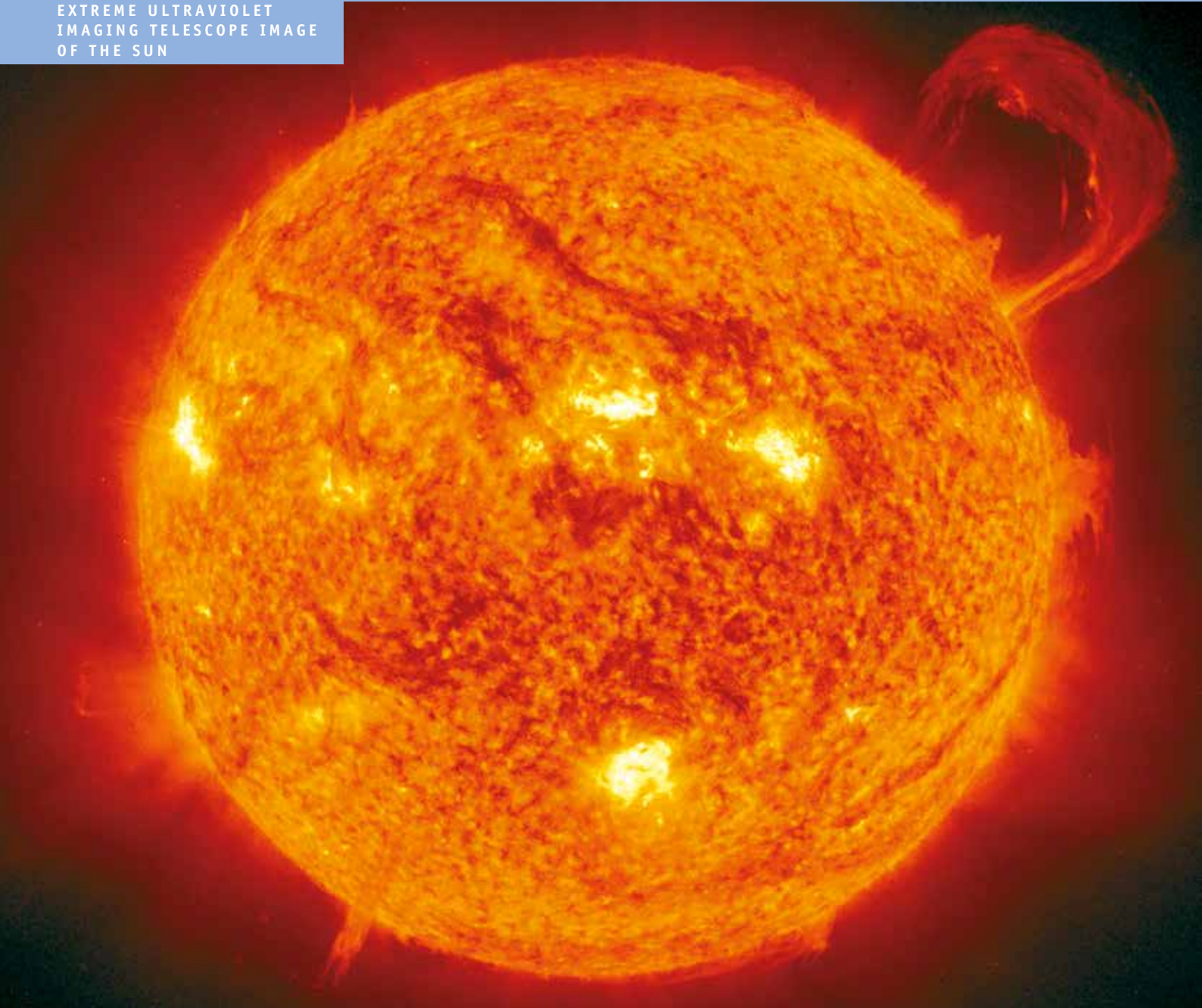


*Number 4 in a series of 7*

CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

## SKIN MALIGNANCY

EXTREME ULTRAVIOLET  
IMAGING TELESCOPE IMAGE  
OF THE SUN



UPDATED CHAPTER  
OCTOBER 2020

‘Solar’ of the sun

## CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

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CHAPTER TWO: PSORIASIS

CHAPTER THREE: SKIN INFECTION AND INFESTATION

**CHAPTER FOUR: SKIN MALIGNANCY**

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TESTS AND TECHNIQUES – WHICH, WHEN AND WHY?  
A SEQUEL TO THE CORE TUTORIALS IN DERMATOLOGY  
FOR PRIMARY CARE

*Front cover image:* Extreme Ultraviolet Imaging Telescope (EIT) image of a huge, handle-shaped prominence taken on September 14th 1999 taken in the 304 angstrom wavelength – Prominences are huge clouds of relatively cool dense plasma suspended in the Sun's hot, thin corona. At times, they can erupt, escaping the Sun's atmosphere. Emission in this spectral line shows the upper chromosphere at a temperature of about 60,000 degrees K. Every feature in the images traces magnetic field structure. The hottest areas appear almost white, while the darker red areas indicate cooler temperatures.  
*Courtesy of SOHO/Extreme Ultraviolet Imaging Telescope (EIT) consortium. SOHO is a project of international cooperation between ESA and NASA.*



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As a consequence of the well established and indeed alarming epidemiological trends in the increasing incidence of all skin cancers in the United Kingdom,<sup>1</sup> there is an enormous amount of literature available to the primary care physician. In this article I plan to review the present situation regarding the common forms of skin malignancy and pre-malignancy.

## SKIN CANCER

Let us look at the relatively small number of conditions that compose the vast majority of cutaneous malignancy and pre-malignancy in ascending order of importance:

**ACTINIC/SOLAR KERATOSES** – These comprise by far the most common of conditions considered by some to be pre-malignant. Their presence acts more as a marker of significant cumulative exposure to ultraviolet radiation and thus a higher risk profile for the affected patient. The exact individual risk of progression from a solar keratosis to squamous cell carcinoma (SCC) is unknown but is considered to be in the ‘ball park’ of 1:1000<sup>2</sup> per annum and 1% in a lifetime. Prevalence in the UK is between 6-15% of the population aged over 40, rising to 23% over the age of 60. Males are most commonly affected. The presence of multiple AK’s increases the risk of progression to SCC in an individual to 14% in 5 years. The clinical appearances are classically those of a textured, circumscribed, scaly, keratotic patch or plaque usually less than 1 cm in diameter. Colouration is varied from frankly erythematous through to yellow, pigmented or flesh-coloured. They always occur on sun exposed sites and particularly the scalp, ears, nose, dorsum of hands and forearms.

Progression to squamous cell carcinoma follows a prolonged latent period of a decade or more, and the resultant SCC’s themselves are generally low grade with low metastatic potential with the exception of those on the ear and lip which tend to behave more aggressively. Signs of change to frank SCC can be subtle – a more rapid growth phase may be noted especially if there are multiple solar keratoses and one appears to be behaving differently. There may be an increased erythema, pain, induration or breakdown in the integrity of the skin surface evidenced by oozing, crusting or bleeding. Even to the practised eye it can be extremely difficult to differentiate reliably between solar keratosis, Bowen’s disease, early SCC and superficial or morphoeic basal cell carcinoma (BCC). Keratoacanthomas can usually be differentiated by a classical history of an ‘explosive’ growth phase over a number of weeks but current practice is to regard all these ‘sheep in wolf’s clothing’ as SCCs and treat appropriately. The presence of multiple keratoses can be associated with arsenical exposure. Examples of this would be ingestion of Parrish’s food, which was previously used as a children’s tonic, or exposure to Fowler’s solution historically used for the treatment of psoriasis. Such keratotic lesions classically appear on the palms and soles which are themselves unusual sites for the development of actinic keratoses.



## TREATMENT OPTIONS

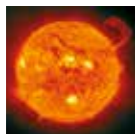
Indeed, arsenic exposure predisposes to a variety of skin cancer types. Particular mention also should be made of actinic cheilitis presenting as whitish or erythematous patches on the lips which can progress to erosion, ulceration and frank SCC. This is especially true of the lower lip. Dermoscopy can be helpful although signs are often subtle. The typical appearances are of an erythematous ‘strawberry-like’ pseudo-network. It is also a useful tool to exclude other pathologies.

There are various accepted treatments for solar keratoses and each will have its enthusiastic advocates. It is worth noting, however, that 25% of solar keratoses may remit spontaneously over a 12 month period<sup>3</sup> and as the spectrum of disease changes and resources potentially contract, an observational approach may become more commonplace. BAD Guidelines advocate an observational policy is an option for mild disease.<sup>4</sup> Established treatments include:

- a) Topical application of 5-fluorouracil (Efudix®) cream – this is a relatively cheap and effective treatment but care must be taken with fair-skinned individuals as skin reactions can be significant and a well established protocol should be followed. My preferred regime is:
  - i) Apply the cream thinly twice a day except thinner facial lesions where once daily is often sufficient. Be particularly careful not to get the cream near to the eyes. Wash your hands thoroughly after use;
  - ii) After a few days (usually three to five), the keratoses will go red and may, in some cases, even blister and weep. There may be some soreness;
  - iii) Once the reaction has started, and provided it is not too sore or severe, continue the application of the cream for a further three weeks. If at any time the reaction is really unpleasant and sore, stop treatment and apply a steroid cream or ointment (usually hydrocortisone 1% will be prescribed). If a severe reaction has occurred it is likely that the keratoses will have been effectively treated and no further application will be required. If in doubt, consult your doctor and he/she will advise as to whether you should continue treatment;
  - iv) At the end of treatment (usually after three weeks in all) any reaction still present may be treated with hydrocortisone 1%. It may take four to six weeks after the end of treatment before all the lesions have healed.
- b) Cryotherapy using very short bursts of liquid nitrogen spray of 5-15 seconds. You must be confident of the diagnosis as in such techniques the “evidence” is obviously destroyed.
- c) Curettage and cautery.
- d) Other topical treatment options include imiquimod 3.5% (Zyclara®) or 5% imiquimod cream (Aldara®), 3% diclofenac gel (Solaraze®) and Actikerall® which combines a less potent concentration of 5-fluorouracil (0.05%) with salicylic acid as a keratolytic agent. Aldara® has a licence to treat superficial BCC and actinic keratoses. Treatment regimes for Solaraze® are lengthy (up to 90 days) and require careful selection of appropriate patients. It is likely only to influence quite superficial disease. More data is needed on long term outcomes, both clinically, histologically and in terms of cost effectiveness. Actikerall® is a more individual lesion targeted treatment used for between 6 and 12 weeks.

- e) Photodynamic therapy (PDT) – this is a treatment modality that is not, in fact, new but is undergoing somewhat of a renaissance. It remains available, however, only in certain centres. It combines the application of a topical photosensitiser with exposure to particular wavelengths of light. Capital equipment costs are high and the main drawback can be the pain induced during treatment and long term outcomes remain uncertain. The option of topical day-time PDT requiring no hospital based treatment is more recently becoming available in certain areas.
- f) Excision if the lesion is indurated or clinically suspicious of having undergone transition to SCC.
- g) Consideration of starting nicotinamide 500mg twice daily prophylactically should be given to high risk patients.
- h) Debate continues regarding the pros and cons of treatment of more extensive areas of actinic damage, so called ‘field change’.

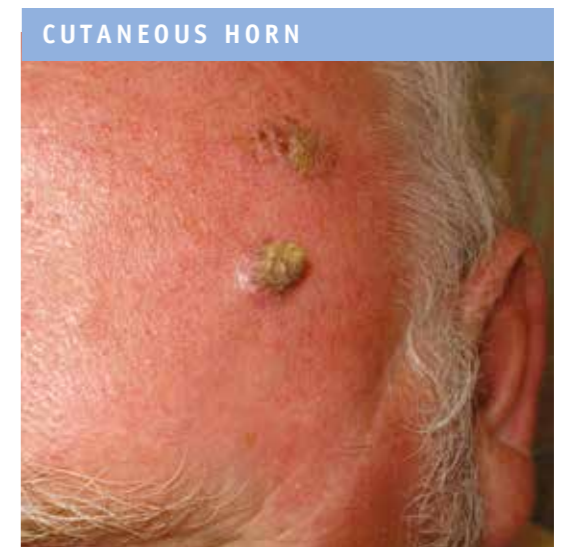
**BOWEN’S DISEASE** (Intraepithelial carcinoma) – This common condition is ‘*in situ*’ SCC. The potential for malignant changes is considerably higher than solar keratoses, estimated at 3-5%<sup>5</sup> of untreated cases with a consequent metastatic potential of 13%. Clinically, it presents as an asymptomatic well defined erythematous, scaly plaque with a pattern of centrifugal spread. It can mimic an isolated patch of eczema or psoriasis. Sites of predilection include the face, lower limbs and fingers. A degree of definition helps to distinguish it from a solar keratosis while the absence of a finely elevated ‘whip cord’ margin help to distinguish it from a superficial BCC (see page 5). However, such differentiation can be very challenging. Dermoscopy can be useful but the microscope is often the final arbiter. There is occasionally an association with a past history of arsenical exposure and treatment modalities mirror those of actinic keratoses, as discussed previously.



There are a number of other conditions considered to be potentially pre-malignant stages of SCC; these include Erythroplasia of Queryat (penile intraepithelial neoplasia) which equates to an *in situ* SCC of the penis and presents as a sharply demarcated erythematous plaque. There is a 10% risk of progression to invasive carcinoma.

Squamous cell carcinomas are also well recognised to arise in areas of prior skin damage, either through radiation or chemical exposure, or in sites of chronic sepsis and inflammation such as scars, sinuses and leg ulcers (so called Marjolin’s ulcer).

In addition, up to 15% of cutaneous horns have histological changes of SCC in the base and all such lesions should be submitted for histological examination.



**KERATOACANTHOMAS** – These are considered by some authorities a ‘form fruste’ of frank squamous cell carcinoma. These tumours characteristically arise *de novo* and erupt rather like a small volcano over a period of 6-12 weeks. They can grow at both an alarming rate and to quite a large size but on average plateau at between 10-20mm diameter. The history then is of a spontaneous regression over the next 3 months. Present advice, however, is to treat these tumours by excision if practicable. This is both to avoid the risk of missing frank squamous cell carcinoma, but also if keratoacanthomas are left to resolve naturally they may leave a more unsightly scar than would have been obtained by early surgical intervention. In certain circumstances, in experienced hands, curettage or careful observation and follow up may be justified. The situation is complicated, however, by the fact that histologically the pathologist can on occasion have problems differentiating between keratoacanthomas and well differentiated squamous cell carcinomas.



**BASAL CELL CARCINOMA (BCC)** aka basal cell epitheliomas or, more colloquially, rodent ulcers – these are extremely common tumours, representing 80% of all skin cancers, presenting most frequently on the face and unlike squamous cell carcinoma there is no commonly recognised pre-malignant stage. Men are affected twice as often. Worthy of mention, however, is the congenital naevus sebaceous which presents as an indolent, indurated hairless yellow plaque, most commonly on the scalp. This is estimated to have 10% risk of progression to basal cell carcinoma in adulthood, and there is need for consideration of the pros and cons of pro-active excision post puberty.

MALIGNANT CHANGES IN SEBACEOUS NAEVUS



The incidence of BCC has increased 75% in the last decade alone. The risk of development appears to relate to intermittent cumulative ultraviolet exposure. This is not the whole story, however, as the sites most common for BCC do not correspond exactly with sites of maximal sun exposure. There is a common perception that this is an ‘old person’s’ disease but it is really not unusual to diagnose BCC in people in their 30’s and 40’s. Most doctors equate the appearance of a BCC with that of a classical nodular cystic lesion with a ‘rolled’ edge, a central crusted or ulcerative core and the presence of telangiectasia. There are, however, a variety of other distinct presentations:

- **Superficial BCC** – These are common on the trunk and lower limbs presenting as very well circumscribed patches of erythema with a subtle ‘whip cord’ edge. They are frequently dismissed or overlooked and can reach very significant proportions before the diagnosis is reached, thereby narrowing the treatment options.
- **Pigmented BCC** – These are often classically nodulocystic but the presence of pigmentation can cause confusion and concern regarding the possibility of a malignant melanoma.

SUPERFICIAL BCC



PIGMENTED BCC



- **Cystic BCC** – These can present as well defined smooth domed lesions.
- **Morphoeic/pleomorphoeic** – As the name suggests, these can be varied in appearance, often presenting as non-descript, ill-defined lesions with chronic scabbing, scarring, infiltration and induration. The degree of invasiveness can be very advanced before the diagnosis is reached and often a procedure such as Mohs micrographic surgery is needed for disease clearance. Such advanced dermatological surgery is standard practice in the US but is only available in a relatively small but increasing number of specialised units here in the UK.

CYSTIC BCC



MORPHOEIC BCC



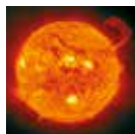
TREATMENT OPTIONS

There are numerous ways to treat BCC’s, all with enthusiastic proponents; these include many of the modalities already mentioned for actinic keratoses, namely cryotherapy, curettage and cautery, electrosurgery, excision and PDT (for thin lesions only). Also employed is radiotherapy for larger lesions, especially superficial BCC’s, and they can also be treated with intralesional 5-fluorouracil. There is no one right way to ‘skin this particular cat’! The operative must be confident and experienced in whatever modality they choose, and be prepared to be flexible in approach according to the age and fitness of the patient and the anatomical site.

Excision is the only method giving histological confirmation of clearance, but even this is an inexact science. Standard histological preparation of a specimen may miss tumour margins. Not infrequently tumours deemed histologically clear, clinically recur. Conversely, tumours that are reported to reach or involve the incision margin are often followed up rigorously with no clinical recurrence over a prolonged period. It may be by debulking, residual tumour is destroyed by the inflammatory immune response invoked. However, every effort to clear disease at the first time of asking should be made. Surgical clearance is recommended to be ideally with a macroscopic perilesional margin of 4mm where possible/practicable; between 1 in 2 and 1 in 3 tumours that histologically are reported to be at the deep margin do recur. The figure reduces to as little as 1 in 4-6 for those tumours reported to be at or near the lateral margin. The operative must be particularly careful to achieve tumour clearance at the deep margins if grafts or flaps are employed for, if not, the tumour can be deeply buried and clinical recurrence only evident when the tumour is well advanced.

There are a number of ‘high risk’ sites where particular skill is needed to obtain adequate clearance and cosmetically acceptable results; these include tumour in the post-auricular or nasolabial folds, tumour near a free margin such as an eyelid or lip, or tumours closely approximate to vital structures such as the eye itself. My general preference is to excise surgically tumours on the head and neck, and also on the body of younger patients when possible, and to use other techniques, especially double curettage and cautery for truncal and more peripheral tumours. Excision gives a 95-99% ‘cure’ rate in competent hands. Curettage and cautery is reported as giving a ‘cure’ rate of 80-90%. In the latter, the option to excise recurrences usually remains. We increasingly see very elderly, frail patients with high tumour loads. Careful consideration on a case to case basis of such patients needs to be given to consider how ‘aggressively’ they should be managed and a ‘watchful waiting’ approach may well be in the patient’s best interest in some instances.

**SQUAMOUS CELL CARCINOMA (SCC)** – This tumour is regarded as potentially more serious as it has the ability to metastasise. It is the second most common of the non melanoma skin cancers, occurring twice as commonly in men than women, and most commonly in patients over the age 70. Incidence has increased by 30% in the last decade in the UK. These tumours arise from the keratinising cells of the epidermis and its appendages, and also from mucous membranes. As previously discussed there are a number of pre-malignant conditions at risk of squamous metaplasia. Predetermined risk factors are chronic cumulative ultraviolet exposure, arsenicals, ionising radiation or impaired immune factors, for example secondary to transplantation and/or immunosuppressive medication. Indeed, transplantation poses particular risks with hundredfold increase in the incidence of SCC, and such tumours often behave more aggressively.



Clinically, the presentation is of an indurated, inflammatory or ulcerated nodule most commonly in the dorsum of the hands or the face. High risk sites for primary lesions most prone to metastases include the ear, lips, scalp, eyelids and nose.<sup>6</sup>

In keeping with other non melanoma skin cancers, there are to date no reliable prospective randomised studies to determine the relative efficacies of the various treatment modalities used. Excision is the treatment of choice. Squamous cell carcinoma is also sensitive to radiotherapy. Excision for larger tumours is considered best practice with an optimum margin of at least 5mm. Recommendations regarding margins depend on histological subtypes whether the tumour is well, moderately or poorly differentiated. This can often be following initial histology and the advice of the local MDT. Cure rates are reported of 95%. Micrographic surgery (Mohs surgery) where tumours are excised under perioperative histological control is provided by specialist units, can be considered for tumours arising on high risk sites, for larger tumours over 2cm, and also for patients with coexisting immunosuppression, for recurrent tumours and for those that appear clinically deeper and more infiltrative.

**TREATMENT OPTIONS**

**MALIGNANT MELANOMA** – Never truer is the old adage ‘prevention better than cure’ than when applied to malignant melanoma. A close second best is early diagnosis and treatment as this most aggressive and life threatening of tumours is infinitely amenable to often simple surgical intervention in its early stage, but once metastasised, treatment options remain more limited although genetic mapping and a raft of new drug therapies offer hope for the future. It is a tumour of major and increasing importance, commoner now in the UK than either cervical cancer or Hodgkin’s disease. There are almost 12,000 new diagnoses and 2,000 deaths annually (2008).<sup>7</sup> The peak age of incidence is in the fourth decade. There has been a threefold increase in the last two decades which represent the greatest proportional rise of any malignancy, except carcinoma of the lung in females. This applies both to the UK and Western Europe. This increasing trend is predicted likely to be at least sustained for the next two decades.

A number of risk factors are well established:

- The relationship of melanomas and ultraviolet light exposure remains unclear with significant intermittent exposures considered a risk factor for most melanomas but chronic life time exposure is more likely to be a factor in lentigo maligna melanoma
- History of malignant melanoma or dysplastic naevus syndrome; there is a reported 5% incidence in 1st degree relatives
- Presence of large numbers of benign melanocytic naevi (the average person has approximately 40)
- Dysplastic naevus syndrome defined as the presence of over 100 naevi, many greater than 1cm diameter and of atypical appearance

The evidence regarding the risk of sunbeds is no longer equivocal. Injudicious use increases risk. It is now illegal in England, Scotland and Wales to use sunbeds under the age of 18.

Clinically, the diagnosis depends on a number of factors; these have been defined by Professor Rona Mackie of Glasgow University.<sup>8</sup> The original seven point check list has now been refined to three major and four minor factors. Indeed the importance of the diameter of the lesion should no longer be relied upon as nowadays dermoscopy allows for much greater diagnostic accuracy of smaller lesions.

**Table 1. Year 2000 Glasgow seven point check list (Mackie 2000 p8 Primary Cutaneous Malignant Melanoma)**

**Major features**

1. Recent *growth* in new or pre-existing pigmented lesion.
2. Recent *change* in *shape* of new or pre-existing pigmented lesion.
3. Recent *change* in *colour* pattern of new or pre-existing pigmented lesion.

**Minor features**

4. Symptoms – mild itch is the commonest.
5. Oozing or crusting – frank bleeding or ulceration.
6. Inflammation in and around the lesion.
7. Largest diameter 5mm or greater. Although some melanomas are smaller than this, the majority are 5mm in largest diameter by the time they are recognised.

Over 90% of patients presenting with malignant melanoma give a history of a changing lesion over the previous few months. The lesion itself will often have three separate colours or shades within it; these can vary from black to brown to red or even occasionally white, which is due to partial regression. The majority of melanomas arise *de novo* rather than superimposed on existing melanocytic lesions. The back is the highest risk area for men, accounting for a third of all melanomas, in contrast to women where 50% arise on the lower limbs. These sites clearly support the theory of sun exposure as a risk factor.

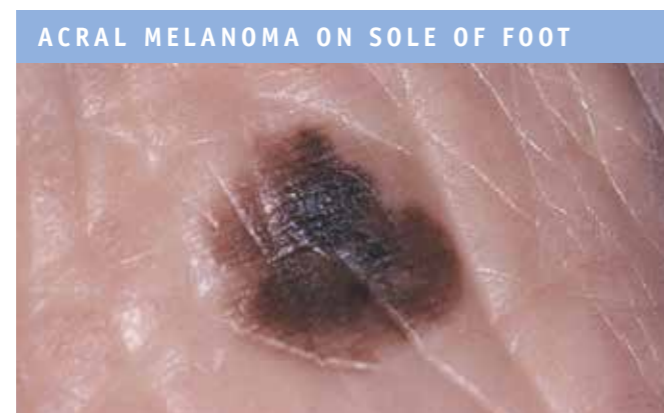
On a positive note, however, despite the worrying increase in the incidence of malignant melanoma, the number of deaths is **not** rising proportionately, neither unfortunately is it falling! This is due to a trend towards tumour presenting for diagnosis at an early stage as a result of greater public and physician awareness.

There are a number of distinct patterns of malignant melanoma:

- **Superficial spreading melanoma** – These constitute 80% of malignant melanomas in the UK;



- **Nodular melanoma** – Accounting for 10%;
- **Acral melanoma** (acral = Greek for extremity) – These occur on the soles and palms, the former more commonly. Diagnosis can be delayed, particularly on the soles, and these lesions can frequently proceed to ulceration. Subungual melanomas can be considered as a variation of acral melanoma. Pigmentation involving the nail fold (Hutchinson’s sign) may demonstrate an advanced lesion with a poor prognosis;



- **Amelanotic melanoma** – Somewhat of a misnomer as there is very often some pigment present on close examination;

**DIFFERENTIAL DIAGNOSIS**

- **Lentigo malignant melanoma** (aka Hutchinson’s Freckle) – These present as flat, brown stains most commonly in old age. The cheek is the site of predilection. They are histologically melanoma *in situ*. The history is usually of a very gradual extension over a number of years. The tumour can have reached quite a large size by the time of diagnosis, often in excess of 20mm. Old photographs can be helpful with a diagnosis here. These *in situ* tumours do have the potential for undergoing vertical growth phase to frank invasive nodular malignant melanoma.



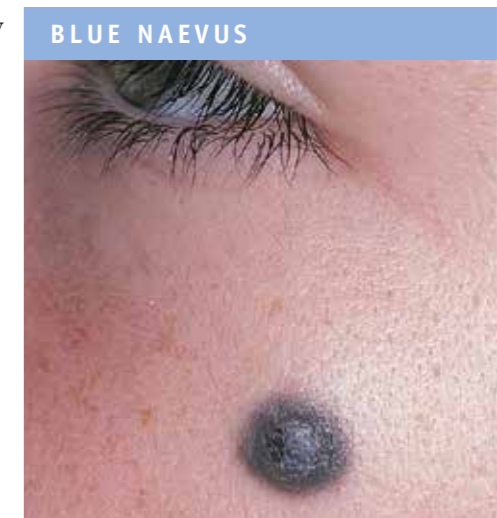
There are a number of benign conditions that can cause both confusion and concern:

- **Pigmented dermatofibroma/histiocytoma** – These lesions should, however, remain static and unchanging with a textural feel described as feeling ‘like a lentil underneath the skin’. An additional helpful clinical sign is that they exhibit the “dimpling sign” when pinched between two fingers;

- **Halo naevus** (Sutton’s) – These lesions commonly occur in adolescents and cause undue concern because of their changing nature. However, the well circumscribed perilesional depigmentation gives the diagnosis. The process is of a benign melanocytic naevus undergoing autoimmune destruction. The development of halo naevi on older patients should be treated with more suspicion;

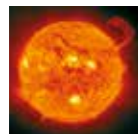


- **Blue naevus** – Their monomorphic blue/grey pigmentation due to their positioning deep in the skin, along with a regular outline and an established history of arising in puberty and being unchanging, differentiate these from malignant melanoma;



- **Pyogenic granuloma** – These sometimes need differentiation from amelanotic melanomas, but a history of trauma, a characteristic exposed site especially in a digit and a complete lack of pigment, support a benign diagnosis. Histology should however **always** be sent;

- **Junctional naevi** – These commonly occur on the palms and soles and often cause anxiety. The outline is often irregular but the colour is characteristically uniform;

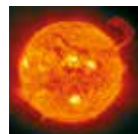


- **Seborrhoeic keratoses** aka seborrhoeic warts or basal cell papillomas (unkindly known in some older textbooks as ‘senile warts’ or even more unkindly ‘medalles de la cimetiére’ in France meaning ‘medals of the cemetery’!) – These benign skin growths are almost ubiquitous on elderly skin, either occurring singularly or multiply, with a predilection for the trunk and face. They have a ‘stuck on’ rough, cerebriform appearance usually with a clear definition from surrounding skin and the presence of plugs or pits on the surface. However, particularly when arising in an area prone to friction such as a bra strap or waistband, they can become irritated and inflamed and take on a ‘worrying’ appearance;



- **Angiomas** – These particularly can often be reliably distinguished with the use of a dermatoscope, discussed later in the article;
- **Pigmented BCC’s** – As previously discussed, these have a characteristic raised ‘rolled’ edge associated with such tumours.

Studies demonstrate that experienced clinicians can differentiate malignant melanoma with approximately 85% accuracy from other pigmented skin lesions. The results for primary care physicians were 50%. The ‘gold standard’ for diagnosis of malignant melanoma has remained the histological assessment. There are two well known classifications, the Breslow thickness and the Clarke’s levels. The former relates to a simple measurement of depth of tumour in millimetres; the latter to the anatomical depth reached by the tumour in the dermal/epidermal architecture.



**TREATMENT OPTIONS**

Treatment of established malignant melanoma is surgical. Frequently, by definition, this requires a narrow margin excision for diagnosis and a secondary procedure according to the staging of the tumour. The present thinking is of an excision margin of 1cm per 1mm of depth to a maximum of 3cm. Narrower margin excisions are considered adequate for *in situ* tumours. Incisional biopsy, with the exception of lentigo malignant melanoma, is frowned upon due to a theoretical but unquantified risk of vascular dissemination. A topical issue presently is the concept of sentinel node biopsy which requires the use of lymphoscintigraphy to identify lymphatic drainage pathways and subsequent biopsy of the ‘so called’ sentinel node. This is an expensive, invasive and labour intensive process which is useful for prognosis and staging, but until effective secondary treatments are available could arguably be considered of academic or prognostic value only.

Metastatic spread is classically to the lymph nodes, liver, lung, brain, bone and skin. Long dormant intervals of up to 20 years can occur before secondary tumour appears. Clearly, patients with malignant melanoma need access to ongoing advice, support and follow up now provided by cancer nurse specialists. Even if the malignant melanoma was to develop *de novo* during pregnancy, further pregnancy, hormone replacement and the oral contraceptive pill are not risk factors for malignant melanoma. Secondary treatment of metastatic malignant melanoma remains challenging. The disease can be radio, chemo and immunotherapy resistant, although radiotherapy can be a useful palliative measure. Recent breakthroughs in gene mapping of the disease and new pharmacological agents bring fresh hope of new and effective treatments.

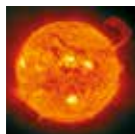
**Table 2. Prognostic indicators in malignant melanoma**  
(Hunter *et al.* Clinical Dermatology Vol 3 p204)

Indicator	Significance
Depth of primary tumour	Breslow <1.5mm good >3.5mm bad
Sex	Females do better than males.
Age	Prognosis worsens after 50 years, especially in males.
Site	Poor prognosis with tumours on trunk, upper arms, neck and scalp.
Ulceration	Signifies poor prognosis.

Problems do not end, however, with histological examination! A National Institute of Health review of histological diagnosis of early malignant melanoma employing eight pathologists with a special interest in the disease demonstrated major disagreement on examining a range of histological specimens. The summary of the study commented: “The conclusions of the article should be chilling, not only to physicians, but to patients and sobering to lawyers for plaintiffs”.



**PREVENTION OF SKIN CANCER** – Of the 100,000 new cases of skin cancer per year it is thought that 80-90% are related to sun exposure. Sunscreens have been pushed very energetically as the solution to this ‘epidemic’. The benefits of sunscreens are established for reducing sunburn, photoageing and photocarcinogenesis in animal models.<sup>9</sup> The benefits in tumour prevention in the population are unclear. One significant episode of sunburn in childhood is an established risk factor for the subsequent development of malignant melanoma. A WHO working group concluded, however, that although appropriate use of potent sunscreens (SPF = greater than 15) probably reduce the risk of SCC, paradoxically, users of sunscreens had an increased risk of malignant melanoma.<sup>10</sup> Sunscreens, theoretically, allow people to have longer exposures to potentially carcinogenic wavelengths of ultraviolet light while negating nature’s reminder of too much sun exposure – **sunburn!** This problem was historically perpetuated by the fact that until recently the emphasis in sunscreens was UVB protection, which is what the SPF refers to. This imbalance has been corrected somewhat by rating systems for UVA protection as well to produce more ‘balanced’ sunscreens. The use of sunscreens cannot be relied upon as a single strategy but has to be an integrated skin protection package along with other photo-protective measures. This has been energetically pursued in Australia with enormous public health campaigns to raise awareness, such as ‘Slip, Slap, Slop’ which reminded people to slip on a shirt, slap on a hat and slop on the sun cream. Consideration of the vitamin D status of patients that are rigorously practicing sun avoidance needs to be given.



**Dermatoscope** – This instrument previously referred to in this article is a simple and affordable skin magnification system that resembles a glorified auriscope. It has an increasing number of enthusiastic practitioners but should only be considered as a diagnostic aid and not a replacement for careful history taking, traditional clinical assessment and experience. At the end of the day, as in most things in life, there is **no** substitute for the latter. You just have to see an awful lot of skin lesions before you can reliably separate the pathological ‘wheat from the chaff!’ The dermatoscope, however, is particularly good at differentiating angiomatous lesions and seborrhoeic keratosis from more worrying pathologies. It also is very useful in the diagnosis of BCCs. It is important to get professional training in the use of this instrument. The availability of such courses continues to expand.



## SKIN SURGERY

In this article it is not appropriate to provide a comprehensive treatise on all aspects of skin surgery. Personally, I prefer this term or the term ‘local anaesthetic surgery’ to ‘minor surgery’ as somehow this trivialises this type of surgery. Indeed, in many ways surgery carried out under local anaesthetic, with a patient who is awake and aware of what is going on, can be much more challenging and often requires excellent communicational and interpersonal skills from the doctor and his support staff. Ultimately, it is rather a medical truism to state that every physician must assess his experience and technical expertise when providing any service. Historically, dermatologists were often confident diagnosticians concerning skin lesions, but didn’t necessarily have the technical surgical abilities to treat such problems. On the other hand, surgeons were not confident of diagnosis but did have the necessary skills. This is increasingly a thing of the past as SPR rotations produce good and often excellent dermatological surgeons, in addition to the traditional diagnostic skills.

The national scene regarding the provision of skin surgery has altered fundamentally through the process of NICE since I first wrote this chapter and has caused huge controversy, frustration and anger to many. After much cross-party discussion over a number of years involving the RCGP, BAD and PCDS, a clear standardised pathway has emerged to allow so called GPwERs (GPs with extended roles) to develop sub-specialisation in skin cancer diagnosis and management. It remains my firm opinion that the majority of ‘lumps and bumps’ surgery, including uncomplicated skin cancer, could safely, economically and conveniently be provided in community settings if the necessary skills are properly taught, the audit and supervision robust and the financial rewards were proportionate. Further details as to how to become a GPwER are available through the RCGP website.

Finally I would like to pass on one personal observation regarding the practice of biopsying skin rashes when there is little attempt to reach at least a workable differential diagnosis. This has been described by one of my colleagues as ‘the last bastion of the therapeutically destitute!’ If the clinician fails to make a diagnosis or an accurate differential diagnosis when presented with the complete clinical picture, how can he expect his histopathological colleague to make such a diagnosis on a small piece of tissue marinated in haematoxylin and eosin! However, I digress!

For those who like acronyms, the one that I constantly regurgitate is that if you are sending histology, the pathologist will be SAD unless he has at least three pieces of information on the specimen form, in addition to patient details.

S = Site

A = Age

D = Duration (history)

This information is often pivotal in order to narrow a differential diagnosis.





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