MOLES AND MELANOMAS OF THE SKIN

J. HESELSON, B.A., M.B., CH.B. (CAPE TOWN), F.R.C.S. (ENG.), *Surgeon, Groote Schuur Hospital, Cape Town*

In spite of the fact that these pigmented lesions have afflicted (or beautified) the human race from time immemorial, there is still widespread uncertainty about, and fear of, their nature and behaviour among both the public and the medical profession.

CLASSIFICATION

In recent years there have been a considerable number of articles on the subject. Based on the classification of Allen and Spitz,[1] naevi or moles may be subdivided according to the levels in the skin where the collections of melanocytes or naevus cells occur, and on their appearance, as follows:

Benign

1. Junctional:
   (*a*) Early (quiescent, lentigo);
   (*b*) Late (premalignant or activated).
2. Intradermal.
3. Compound.
4. Juvenile or prepubertal melanoma.
5. Blue naevus (including Mongolian spot and naevus of Ota).
6. The extensive hairy mole (hamartoma).

Malignant

1. Malignant melanoma (melanoma):
   (*a*) Superficial;
   (*b*) Deep.

In the junctional naevus the naevus cells are found only in the basal portion of the epidermis, i.e. at the junction with the cutis vera or dermis. At the periphery, there may be superficial spread with cells isolated from the main lesion, and this scatter may account for recurrence after excision.

In the intradermal naevus the cells are in the superficial portion of the dermis, whereas compound naevi show cells in both situations. The blue naevus is located in the deeper parts of the dermis.

BENIGN NAEVI

Junctional Naevi

They are the most immature naevi. They may, unusually, be present at birth or may appear at any time thereafter until old age. Many junctional naevi, otherwise invisible, become obvious when examined by a Wood light in the dark.[2]

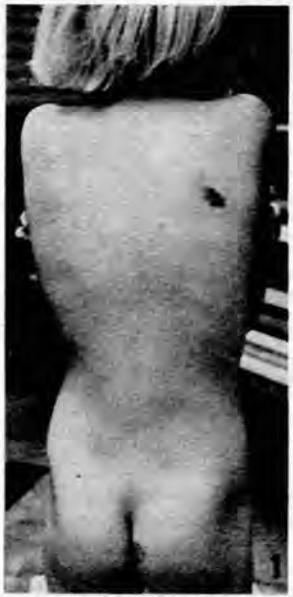


Fig. 1. Child, aged 8 years, showing junctional naevi.

They are far commoner in children, usually appearing for the first time about the third year of life. They increase in number up to puberty and may appear in crops. Spitz[3] found junctional change in 98% of moles in children, whereas, in adults, only 12 - 25% were of the junctional type.

Clinically, they are smooth or slightly raised and may be speckled and yellow or brown in colour, with a fuzzy border. They are found anywhere on the body; but on the palms, soles and genitalia they tend to persist as junctional naevi throughout life (Fig. 1).

Activated Junctional Naevi

When junctional naevi or the junctional elements in compound naevi become active, the cells show cytoplasmic and nuclear changes, including mitoses. Subepithelial infiltration with inflammatory cells, mainly lymphocytes, takes place.

There may be nothing in the clinical appearance to indicate this histological activity, or it may be revealed by the naevi becoming larger or darker, or both. In the adult this is a premalignant stage, although Becker *et al.*[4] regarded it as a form of established malignant melanoma *in situ*. Before puberty, however, these naevi possess little or no malignant potentiality. Allen[1] believed that almost all malignant melanomas, except the rare malignant blue naevi, arise in an area of junctional activity.

Intradermal Naevi

These are the 'common or garden' moles. Moles are seen commonly on the face and are rare before puberty. They are well elevated above the surface of the skin and vary in size. They vary in colour from pale to dark brown and they frequently sprout hairs. They are often pedunculated and their surface may be smooth or warty.

Pure intradermal naevi never become malignant.

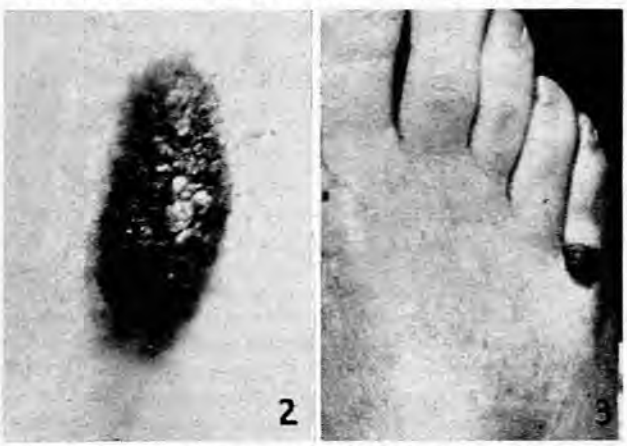


Fig. 2. Compound naevus with depigmentation of central warty area. This was regarded as suspicious of malignant change. Histological examination of the excised specimen, however, showed no evidence of malignant melanoma. *Fig. 3.* Compound naevus, showing faint rim of peripheral junctional change.

Compound Naevi (Figs. 2 and 3)

These are combinations of the intradermal and junctional types. Clinically, they resemble intradermal naevi and often the area of junctional change (a narrow flat band of pigmentation about the periphery) is difficult to see. Compound naevi are usually seen in the pubertal and older age periods. The ring of junctional change may provide the starting point of a malignant melanoma.

Juvenile (Prepubertal) Melanomas

These are rare specific histological entities found in about 6 - 8% of naevi removed from children, and very rarely in adults. The histological appearances are like those of malignant melanomas, but these lesions are benign. In later life 6% become converted to melanocarcinomas. Spitz[3] had collected only 13 cases by 1948; McWhorter and Woolner[5] found only 11 among all the pigmented skin lesions removed from children at the Mayo Clinic from 1907 to 1949. In Groote Schuur Hospital, Cape Town, from 1952 to 1959 there were 2 such cases:

(*i*) A European female, aged 14 years, with the lesion on the ear;

(*ii*) A Coloured female, aged 6 years, with the lesion on the conjunctiva. This was excised in 1954, but recurred the following year and was again excised. There was no sign of recurrence in July 1959.

These melanomas usually occur before puberty. They are commonly lightly coloured, reddish or brown, and most frequently occur on the face. They are usually smooth and elevated, but may be warty. They grow rapidly and are larger and more elevated than the usual childhood naevi. Although they look ugly clinically, they do not metastasize.

Blue Naevi (Fig. 4)

More than half of these occur on the dorsal surfaces of the hands and feet; they are rarely found on the palms and soles.[6] They are also common on the face. Their

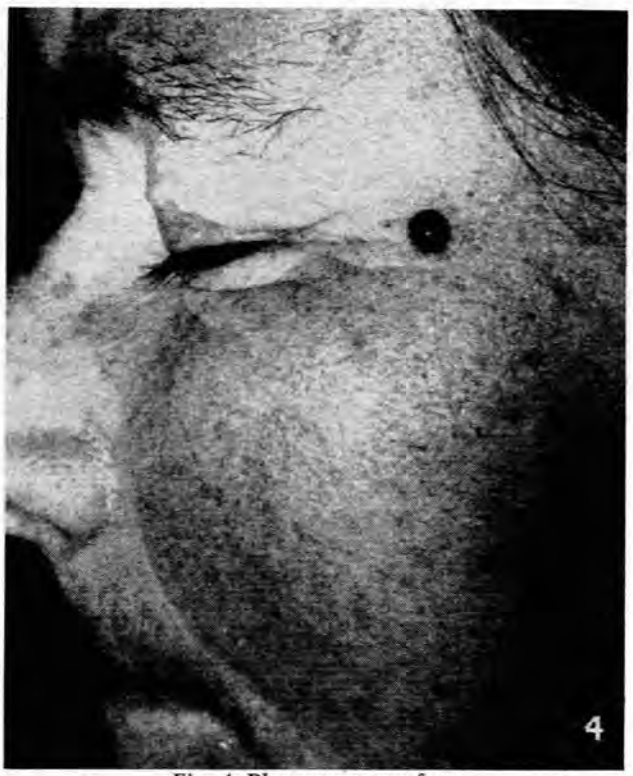


Fig. 4. Blue naevus on face.

colour can vary from yellow to blue-black. They are usually smooth and hairless, but may be papillary. Malignancy is extremely rare.

Mongolian spots. These are, pathologically, blue naevi which are said to be present in the lumbo-sacral region in all Mongolian children at birth, and later fade away. They may occur elsewhere, and may be very large. Gelfand[7] stated that they occur in about 50% of African infants. They affect 75% of Cape Coloured infants between 3 and 10 days of age.[8] The Kalahari Bushmen refer to them as 'jug spots' because the mothers' water containers are carried in close proximity to the sites of these naevi.[9]

Naevi of Ota. These were described by Ota in 1939 and are believed[7] to be blue naevi involving the face and eye. They affect the sclera, retro-orbital fat, and even the periosteum.

Giant Hairy Moles (Fig. 5)

These are present at or shortly after birth and may be up to a foot square in size. The amount of hair and pigmentation in them varies. They do not increase in size except with body growth, but tend to become darker, more hairy and keratotic. There are often associated vascular and other malformations, hence they may be regarded as hamartomata. Pathologically, they are compound naevi.

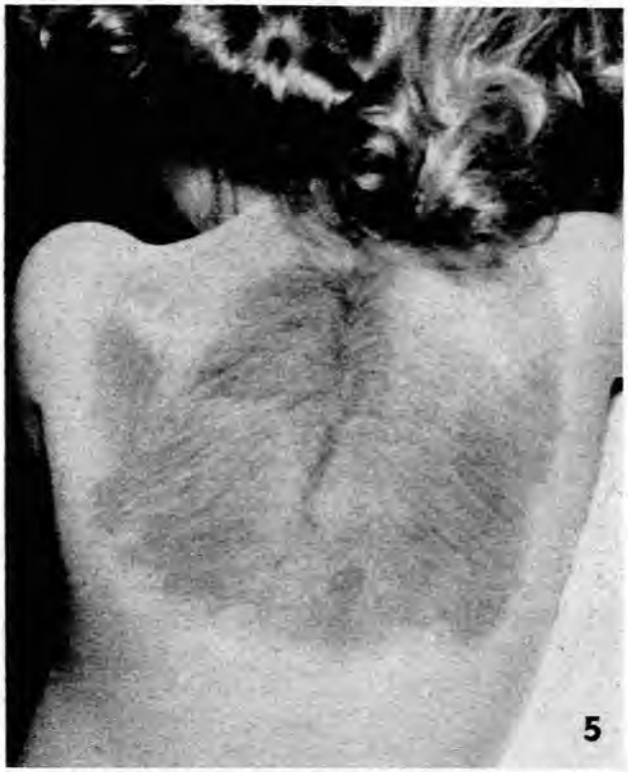


Fig. 5. Giant hairy mole in a very fair-complexioned child aged 2 years.

Discussion

There appears to be a natural history in the development of naevi in which the naevus cells, starting in the basal layer of the epidermis, sink gradually to the depths of the dermis. The different types of naevi represent different ages of the lesion. While there is some correlation between the type of lesion and the age of the patient, this is not absolute, and fully mature intradermal naevi may be found in young babies. At Groote Schuur Hospital, 232 Coloured and 130 African patients had scattergrams of their moles carried out by Mr. T. Schrire and his assistants. From an analysis of these figures (Table I) it can be said that there does not appear to be much difference in the percentage distribution of moles in the Cape Coloured, African and European,[2] although the total number of naevi in the Coloured and African seems to be less than in the European.

Diagnosis

There is a 20% or greater error in the diagnosis of naevi, since the clinical appearances are not completely reliable.[10] Common lesions to be mistaken for naevi are seborrhoeic keratosis, haemangiomas, sclerosing angiomas (dermatocytomas), common warts, freckles, granuloma pyogenicum, squamous papillomas, basal-cell epitheliomas especially if pigmented, and even cutaneous haemorrhages.

Treatment

The 2 main reasons for removing a naevus are (*i*) aesthetic and (*ii*) fear of malignancy. There is nothing to support the thesis that adequate excision of a mole should not be carried out until it bothers the patient. It may then be too late. A benign mole, completely excised, will never recur as a melanoma. We should remember that (*a*) junctional naevi in children almost never become malignant, and (*b*) the average number of pigmented naevi in the European adult is about 10 - 20, and only 12 - 25% of these show junctional change. If the incidence of malignant melanoma is taken as 1 per 50,000 population, then this would mean excising up to half a million naevi in order to prevent 2 melanomas in a population of 100,000. This would obviously be impracticable.

In order to limit the number excised it is advisable to remove any mole that shows *any* type of change, e.g. increase in size or pigmentation, loss of hair or pigment, oozing of serum or bleeding, itching, ulceration, or the presence of pain. In addition any naevus that is black or blue or is situated on the palms, soles, mouth, genitals,

TABLE I. RACIAL DISTRIBUTION OF MOLES IN CAPE TOWN

Race and sex	*Upper limb*	%	*Head and neck*	%	*Trunk*	%	*Genitalia*	%	*Lower limb*	%	*Average no. of moles per person*
C. Female (102) ..	63		123		259		0		118		5·5
C. Male (114) ..	67	12·4	99	20·4	200	43·5	4	0·5	126	23·2	4·2
M. Female (6) ..	6		3		13		0		9		3·5
M. Male (10) ..	2		3		14		1		6		2·6
A. Female (45) ..	24	17·2	17	9·8	75	47·3	0	0·0	42	25·5	3·5
A. Male (85) ..	41		20		103		0		54		2·4

A. = African, M. = Malay, C. = Cape Coloured. Numbers in brackets represent number of persons in each group.

or beneath the nails, should be excised. Chapman and Klopp,[11] who followed such a policy in 1,400 well patients at a Cancer Detection Clinic, excised 65 moles, i.e. 5%, and found unsuspected melanomas in 2 patients. On this basis, in a population of 100,000 persons, 5,000 moles should be excised annually. Pack,[2] in addition, recommended excision of suspected junctional moles early in pregnancy, since malignant melanoma is 3 times commoner in pregnant than in non-pregnant women.

Though the theory that long-continued trauma is a factor in the conversion of moles to malignant melanomas has not been proved, it is probably wise to remove any junctional naevus in a site where it is subjected to recurrent injury or irritation, e.g. on the face, where it is constantly irritated by shaving .

It is only necessary to excise the lesion completely, with a few millimetres of skin around it clear of the most peripheral pigmentation. The excision need not be carried deeply. The juvenile melanoma, also, should be treated conservatively. The giant hairy mole can safely be excised segmentally in stages, though melanomatous change has occurred very rarely.[5]

It is, of course, absolutely essential that histological examination be done, preferably of several areas, and all forms of therapy which do not permit this are to be condemned. We still see too many patients in whom a so-called mole has been treated by diathermy or excised in the surgery, without histological examination being done, and who return after varying intervals with local recurrence or metastases from a melanoma.

MALIGNANT MELANOMAS

In this section, the words 'melanoma' and 'malignant melanoma' are used as interchangeable terms.

Incidence

Workers in various centres have given the following figures: MacDonald (Connecticut) — 0·93% of all cancers; Clark and MacDonald (Texas) — 1·5% of all cancers; Ackerman (USA) — 2·9% of all cancers; Sylven[12] (Stockholm) — 7·0% of all epitheliomas of the skin; present series (Groote Schuur Hospital, all races) — 3·7% of all epitheliomas of the skin; Grieve (Cape Town, all races)[13] — 3·4% of all skin cancers, 0·85% of all cancers; Grieve (Cape Town, Whites)[13] — 2·7% of skin cancers, 0·9% of all cancers.

At Groote Schuur Hospital, from 1952 to August 1959, 65 patients were seen with malignant melanomas out of a total of 1,766 patients with cancers of the skin, of which 585 were squamous, 1,015 basal-cell, and 101 epidermoid carcinomas, apart from the 65 malignant melanomas. The diagnosis was based on the pathological report in every instance. Of the melanomas, 56 were primary and 9 recurrent (Table II). This is a high incidence compared with some other centres, e.g. 132 cases in 27 years at Westminster Hospital.[14]

During the same period, 268 benign moles were removed at Groote Schuur Hospital, of which 22 were of the junctional

TABLE II. MALIGNANT MELANOMAS AT GROOTE SCHUUR HOSPITAL, 1952 - 1959

Group	*Diagnosis*	
Group I	Definite melanomas	52
Group II	Doubtful melanomas	6
Group III	Definitely malignant, probably melanomas	7
	Total	65*

* Of these, 56 were primary, and 9 (14%) recurrent. There were 2 juvenile melanomas.

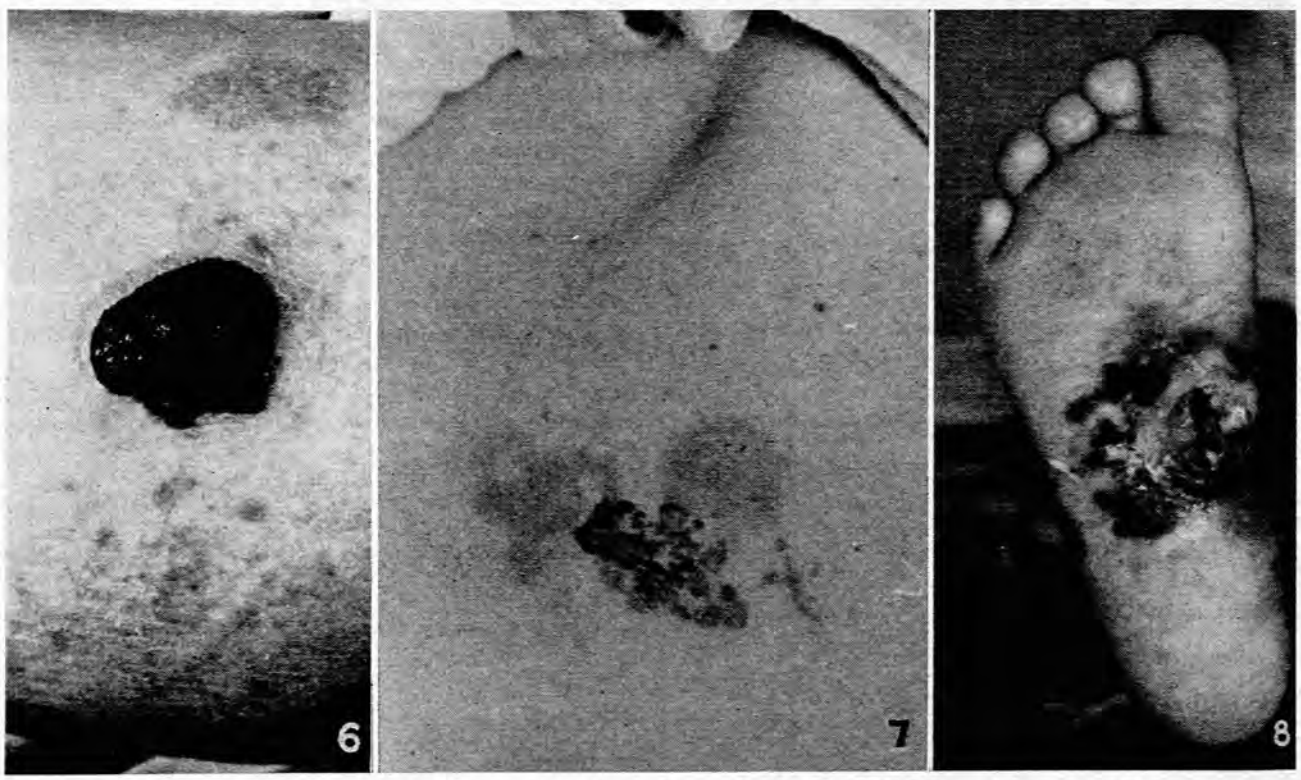


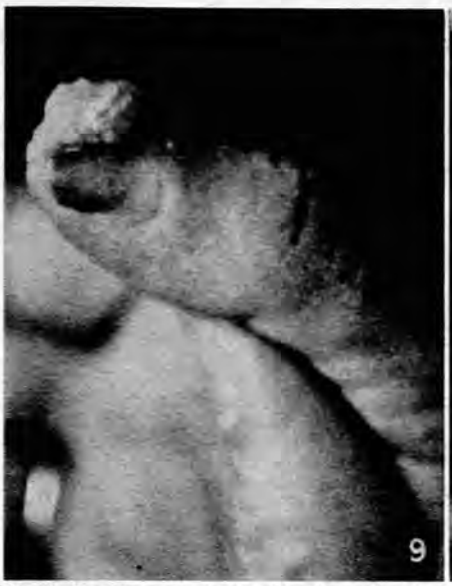
Fig. 6. Malignant melanoma on the arm. *Fig. 7.* Melanoma of nipple area in a male, showing peripheral film of pigmentation. *Fig. 8.* Malignant melanoma on foot, showing satellite nodules and peripheral pigmentation.

variety (8·2%), suggesting that most were removed for cosmetic reasons and that not enough junctional naevi were removed prophylactically. Of the 65 cases included as malignant melanoma, 6 were regarded by our pathologists as doubtful, but probable, malignant melanomas, and 7 were definitely malignant, probably melanomas (Table II).

Clinical Appearance

Malignant melanomas usually present as nodular warty lesions, occasionally in the centre of a mole (Fig. 6). They may be non-pigmented, especially when situated on the sole, toe or finger. Minute satellite nodules or a fine film of spreading pigmentation may appear around the periphery of the main lesion (Figs. 7 and 8). On the other hand they may look like an insignificant mole.

The clinical diagnosis is correct in fewer than half the cases. The differential diagnosis is the same as for benign naevi. In addition, infection and ulceration of mature intradermal naevi, or the changes in naevi which occur in puberty and pregnancy, may suggest melanomatous change (Figs. 9 and 10).



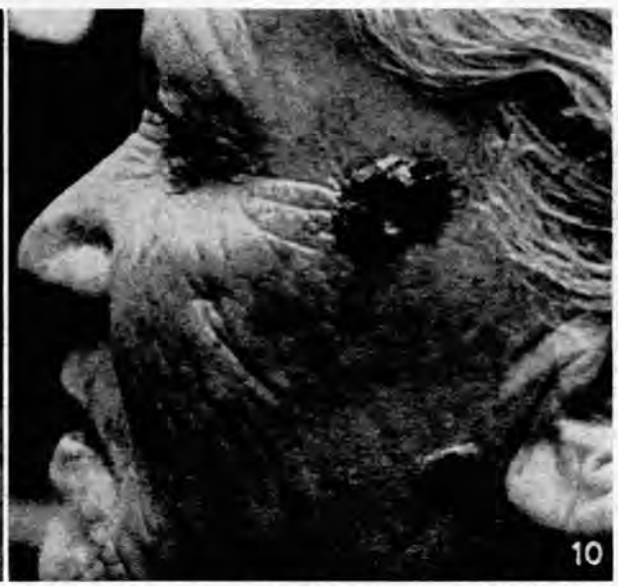


Fig. 9. Granuloma on thumb with subungual ecchymosis. This was diagnosed as a non-pigmented melanoma. *Fig. 10.* Upper lesion: pigmented rodent ulcer which was thought, before biopsy, to be a malignant melanoma. The patient was unwilling to have surgery performed, and was referred for radiotherapy. Lower lesion: an example of a senile wart (keratosis).

TABLE III. MALIGNANT MELANOMAS AT GROOTE SCHUUR HOSPITAL—AGE INCIDENCE

Decade	*No. of cases*
2nd	1
3rd	2
4th	19
5th	11
6th	7
7th	9
8th	11
9th	3
Total	63*

* In 2 cases the age was uncertain.

Age

More than 60% are said to occur between the ages of 30 and 60 years. The Groote Schuur Hospital figures show a similar age distribution (Table III). Malignant melanomas in childhood are extremely rare. In 1954, McWhorter and Woolner,[5] surveying the world literature, would only accept 18 out of 102 cases of reported melanomas in children as true malignant melanomas. This, of course, excluded juvenile melanomas and the very rare transplacental transmission which has been recorded.[15]

There has been no example of a malignant melanoma in a child in our series.

Sex

There is said to be no difference in the sex incidence,[1,15] though some have found a preponderance in females.

In this series (Table IV) there were 41 females and 23 males (1 uncertain). In European and Coloured patients there was a preponderance of females. Among the Africans there were 4 males and 2 females, but this may be due to the predominance of males in our migrant Bantu population.

TABLE IV. MALIGNANT MELANOMAS AT GROOTE SCHUUR HOSPITAL—RACE AND SEX (64 CASES*)

	White	*Coloured*	*African*	*Total*
Females	33	6	2	41
Males	17	2	4	23
Total	50	8	6	64

* One case was uncertain.

Race

It has often been stated that malignant melanoma is a common form of cancer in the African, but this appears to be relative, owing to the infrequency of basal-cell carcinoma in the coloured races. In the African, malignant naevi are commoner on the leg, where the long-continued and recurring injuries from walking barefoot are the usual explanation given. This is likely, since Oettle[16] found that the rural Bantu (as opposed to the urban, who are more likely to wear shoes) were more susceptible to melanomas of the foot.

At Groote Schuur Hospital (Table IV), there were 50 cases of melanoma among White, 8 among Coloured and 6 among African patients. In the same period the ratio of admissions for European, Coloured and African patients to Groote Schuur Hospital, was 6:5:1. Taking this into consideration, there is still a preponderance of melanomas in the White group.

Table V has been compiled from figures obtained from various authors writing on malignant diseases, including malignant melanoma, in the African. It can be seen that the incidence of melanoma expressed as a percentage of total cancers is generally not considerably higher than that found by other writers who quote the incidence in the White races (*cf.* section on incidence). Where the incidence is high there may well be some explanation.

In the albino, malignant melanoma is extremely rare,[17] but in animals malignant melanoma occurs almost exclusively in the depigmented varieties, e.g. in the so-called white, or grey horse. In South Africa there is also an especially high incidence in the Angora goat, totalling 40% of all caprine neoplasms. It occurs in other varieties of goats as well, such as the Swiss milch type, but has not been encountered in other than white goats.[18] It has been suggested that skin colour and malignant melanoma may be controlled by closely related genes.[19]

TABLE V. INCIDENCE OF MALIGNANT MELANOMAS IN AFRICANS

Author		Year	Number of malignant growths	No. of skin malignancies	No. of skin melanomas	Melanomas as % of skin cancers	Melanomas as % of total cancers
Pirie[32]	(SA)	1921	139 (S)	15	6	40	4·3
des Ligneris[33]	(SA)	1927	81 (P)	22	17	77	20·9
Smith and Elmes[34]	(Nigeria)	1934	500 (S)		40		8·0
Burman[35]	(SA)	1935	268 (P)	20	4	20	1·5
Vint[36]	(E. Africa)	1935	546 (S)	195	59	30	10·8
Elmes and Baldwin[37]	(Nigeria)	1947	1,000 (S)		62		6·2
Davies[38]	(Uganda)	1948	225	3	1	33	0·4
Gelfand[39]	(Rhodesia)	1949	334 (S)	81	29	36	8·7
Gelfand[39]	(Rhodesia)	1949	74 (A)	2	1	50	1·3
Cohen *et al.*	(SA, Tvl.)	1952	456 (P)	37	9	26	1·5
Oettle	(SA, Tvl.)	1955	7,485 (S)	458	117	24	1·9
Wainwright and Roach[40]	(SA, Natal)	1957	1,921 (S)	271	90	33	4·7
Grieve[13]	(SA, Cape Town) ..	1959	200 (P)	5	4	80	2·0

P = patients, S = histological sections, A = autopsies, SA = South Africa, and Tvl. = Transvaal.

Site

Proportionate to the skin area involved there is an undue preponderance of malignant melanomas in the head and neck, the sole of the foot and the genitals. The ratio of malignant melanomas to moles in these areas being roughly 2:1, 50:1 and 30:1 respectively.[3]

No one has yet satisfactorily explained the discrepancy in distribution of moles and melanomas. Our Groote Schuur Hospital series showed the highest incidence in the lower limb and the head and neck. This accords with the findings of others.

Spread of Malignant Melanoma

Apart from increase in size, this is *via* the lymphatics or the blood stream. Lymph spread may occur by way of the cutaneous lymphatics, producing skin nodules, or *via* the deeper lymph trunks to the lymph nodes without involving the intervening tissue. This spread *via* the deep perivascular lymphatics is especially likely in lesions below the elbow or knee.[15]

Blood spread to any distant organ may occur without earlier involvement of the lymph glands.

Prognosis of Malignant Melanoma (Skin)

Ever-increasing 5-year survivals are being reported. The New York Memorial Hospital figures[2] show progressive improvement in the 5-year cure rate from 12% in the series reported by Adair in 1936 to 21·4% in 1952 and 37·7% in 1959. At the same time the operability rate improved from 26% in 1936 to 73·3% in 1952. Other authors have recorded even better figures, e.g. Daland[20] in 1959 reported 52% 5-year cures — 71% of these without glands and 26% with glands.

This improvement is ascribed largely to: (*i*) education of the public and of the medical profession, and (*ii*) more radical surgery.

These are better figures than those for cancers of the lung and stomach, and the gloom engendered by such terms as black cancer and black death appears somewhat unjustified. It should, however, be emphasized that these figures are from special centres where the lesions are probably smaller than those we frequently see.

Factors influencing prognosis include:

1. *Size.* The larger the primary lesion the worse the prognosis, the 5-year cure rate being about 60% for the melanomas smaller than 2 cm. and dropping to 16% and less for larger lesions.[21,22] In the Groote Schuur Hospital series more than half were over 2 cm.

2. *Site.* Melanomas on the trunk have a worse prognosis, especially if near the midline where they may spread to several lymph-node groups.[2]

3. *Sex.* The prognosis appears to be definitely better for females than for males.[23-25]

4. *Pregnancy.* Not only do moles increase in number and size and become darker during pregnancy, but also the incidence of malignant melanoma is 3 times greater than in non-pregnant women. There is a greater rapidity of growth and a low cure rate.[2]* The prognosis is not affected by the termination of pregnancy.[11] It is therefore necessary to perform the same urgent essential treatment for pregnant women as for other patients.

5. *Lymph-gland metastases.* The 5-year cure rate is considerably lower when lymph glands are involved microscopically and still lower when the glands are clinically involved.[2,21] Of the group with clinically uninvolved glands, 20 - 40%[21,26] show microscopic invasion. Very occasionally, no obvious primary focus can be found, and the first sign is enlargement of the glands with pain and tenderness.

6. *Degree of pigmentation.* This bears no relation to malignancy. Either the primary or metastases may or may not be pigmented. Excess pigment may diffuse into the blood stream, producing a darkening of the skin and may be excreted into the urine — melanosis and melanuria.

7. *The histological appearance* of the cells and the degree of anaplasia and reticulum content appear to be unreliable guides to prognosis. The superficial malignant melanoma which looks clinically like a benign mole, carries a very much better prognosis.[1]

8. *Local excision.* Where the treatment is inadequate, it has been estimated that the patient has a 2% chance of survival.[27]

9. *Delay in treatment.* It has been shown that a delay of more than one month in definitive treatment meant a fall in survival rate of more than half, from 39·9% to 17%.[2]

TREATMENT OF MALIGNANT MELANOMAS

Doubtful Melanoma

Since there is a 50% error in the diagnosis of malignant melanoma, the first step in the doubtful lesion should be a conservative local excision. My personal conviction is that local anaesthesia should not be used for the surgery of this highly malignant tumour. The injection of the anaesthetic solution opens up tissue planes and may force melanoma cells into the lymph or blood capillaries. One should scrupulously avoid touching or handling the lesion itself. If histology shows malignancy, a more radical excision, which would nearly always necessitate skin grafting, should be performed. On the face and trunk, skin grafting can be avoided by sliding flaps.

Frank Melanoma

In the apparently frank malignant melanoma the lesion may be excised more widely and should include the underlying subcutaneous tissue and deep fascia.

* Recently, contrary to the usual belief, George *et al.*[41] have shown, by a careful study of a large group of pregnant women with melanomas, that their behaviour is essentially the same as that in a control group of non-pregnant women with melanomas.

Biopsy

This should be by complete excision except in very large lesions, the adequate removal of which would entail a mutilating procedure.

Extent of Local Excision

This depends on the site, e.g. on the face near the eye excision will perforce be less radical than elsewhere on the body surface. Some suggest an amount of skin beyond the edge of the lesion equal in extent at least to the diameter of the lesion. An attempt should be made to obtain a margin of at least 2 inches from the edge of the lesion, with about 4 inches or more of the underlying superficial and deep fascia and fascia propria of the muscle, going deeper if necessary. A general rule is that the larger the lesion the more tissue surrounding it should be removed.

There is no doubt that the ruthless excision of the primary growth is the most important part of the treatment of malignant melanoma. The patient's life may depend on those few inches of skin and underlying tissue. All series show a high survival rate with local excision alone, but these are probably small lesions. The skin graft should be cut from a different limb and the donor area dressed before the excision of the primary growth.

For subungual melanomas, wide excision is by amputation through the shaft of the metacarpal or metatarsal. If the lesions extend onto the foot, a Symes' amputation is probably wiser, whereas, when the heel is affected, a below-knee amputation may be necessary.

Lymph Glands

These may be dealt with either by: (*a*) a monobloc operation, or (*b*) a dissociated operation.

Once the lesion has been shown pathologically to be a malignant melanoma, lying close to the regional lymph glands, these should be removed with an intervening area of skin, and a wider area of subcutaneous tissue and deep fascia, irrespective of whether they are clinically involved or not, i.e. the monobloc operation. Pack stressed that the skin over the glands should be sacrificed as well. The only exceptions are in the very old or frail or where (e.g. in the centre of the trunk) all 4 lymphatic fields may equally well drain the primary lesion. Here it is justifiable to wait to see which, if any, gland fields become involved.

In the dissociated operation, where lymph-gland metastases are clinically evident, many advise waiting 7 - 14 days after the primary excision before the gland dissection is done. Pack[29] allowed 6 weeks to elapse after excision of the primary growth. This delay appears undesirable in view of the high incidence of occult metastases in the glands and the possibility of further spread from these; in any case it has not proved effective.[2]

Hip-joint Disarticulation or Forequarter Amputation

Except in aged subjects, if metastases in the axilla or groin have been demonstrated from a distant focus, e.g. hand or foot, hip-joint disarticulation plus dissection of the iliac and obturator glands is advised by Pack[2,29] for the lower limb, and a forequarter amputation with dissection of the lower cervical glands for the upper limb. Where the iliac glands are fixed, he advises hindquarter amputation. He admits, however, that major exarticulations are still under trial.

Local Recurrence

This is a difficult problem and has been dealt with by energetic local excision,[25] degloving of the limb,[24] hip or forequarter amputation, and more recently by limb perfusion with phenylalanine mustard[30,31] or similar substances. We have 2 patients who have lived for 10 and 11 years respectively since the first operation for melanoma, with repeated operations for excision of recurrences. In one the inguinal and iliac glands were uninvolved. In the other the glands of both groins were invaded and the patient has had no recurrence for 7 years. Recently, at laparotomy, when an endometrioma was excised, no evidence of recurrence could be detected.

Radiation

This has yielded no cures. High dosage of high-voltage irradiation may afford useful palliation.[2,14]

Nitrogen Mustard

This has been disappointing when given parenterally, but perhaps has shown more promise when given by regional perfusion *via* the heart-lung machine.[30,31] Castration, adrenalectomy and pituitary irradiation have all been useless, as has been the administration of hormones.

SUMMARY AND CONCLUSIONS

1. A review of moles and melanomas is presented with some local experiences. Both moles and melanomas appear to be definitely more common in the European than in the African.

2. We should be excising more junctional and compound naevi prophylactically.

3. The doubtful melanoma should be excised under general anaesthesia.

4. Ruthless local excision of the primary malignant lesion is absolutely mandatory.

5. Improvement in prognosis would seem to depend largely on more radical surgery.

It is a pleasure to express my thanks to Dr. J. G. Burger, Medical Superintendent of Groote Schuur Hospital, for permission to publish; to Prof. J. H. Louw for his encouragement; to Prof. J. G. Thomson and Dr. C. J. Uys of the Pathology Department, University of Cape Town, for their help and for making the pathology records available; to Prof. J. M. Grieve for data; and to Mr. T. Schrire, with whom I was associated in the Pigmented Lesion Clinic.

Mr. G. McManus, of the Department of Surgery, has been most helpful with the preparation of charts and photography.

REFERENCES

1. Allen, A. C. and Spitz, S. (1953): Cancer, **6**, 1.
2. Pack, G. T. (1959): Surgery, **46**, 447.
3. Spitz, S. (1948): Amer. J. Path., **24**, 591.
4. Becker, S. W., Fitzpatrick, T. B. and Montgomery, H. (1952): A.M.A. Arch. Derm. Syph., **65**, 511.
5. McWhorter, H. E. and Woolner, L. B. (1954): Cancer, **7**, 564.
6. Dorsey, C. S. and Montgomery, H. (1954): J. Invest. Derm., **22**, 225.
7. Gelfand, M. (1957): *The Sick African*, 3rd ed. Cape Town: Juta & Co.
8. Matas, S. (1941): S. Afr. Med. J., **15**, 121.
9. Thomas, E. M. (1959): *The Harmless People*. London: Secker and Warburg.
10. Becker, S. W. (1949): A.M.A. Arch. Derm. Syph., **60**, 44.
11. Chapman, D. and Klopp, C. T. (1954): Med. Ann. Distr. Columbia, **23**, 308.
12. Sylven, B. (1949): Acta radiol. (Stockh.), **32**, 33.
13. Grieve, J: Personal communication (part of a demographic survey of cancer in Cape Town to be published).
14. Cade, S. (1957): Brit. Med. J., **1**, 119.
15. Lewis, C. W. D. (1956): Ann. Roy. Coll. Surg. Engl., **19**, 156.
16. Oettle, A. G.: Personal communication.
17. Shapiro, M. P., Keen, P., Cohen, L. and Murray, J. F. (1953): Brit. J. Cancer, **7**, 45.
18. Jackson, C. (1936): Onderstepoort J. Vet. Res., **6**, 328.
19. Pillsbury, D. M., Shelley, W. B. and Kligman, A. M. (1956): *Dermatology*, p. 15. Philadelphia: W. B. Saunders.

20. Daland, E. M. (1959): New Engl. J. Med., **260,** 453.
21. Lane, N., Latter, R. and Malar, J. (1958): Cancer, **11,** 1025.
22. Lund, R. H. and Ihnen, M. (1955): Surgery, **38,** 652.
23. Wright, R. B., Clark, D. H. and Milne, J. A. (1953): Brit. J. Surg., **40,** 360.
24. Pack, G. T., Gerber, D. M. and Scharnagel, I. M. (1952): Ann. Surg., **136,** 905.
25. Vogler, W. R., Perdue, G. D. and Wilkins, S. A. (1958): Surg. Gynec. Obstet., **106,** 586.
26. Royster, H. P. and Baker, L. M. (1957): Ann. Surg., **145,** 888.
27. Preston, F. W., Powers, R. C., Clark, T. H. and Walsh, W. S. (1954): A.M.A. Arch. Surg., **69,** 385. Quoted in *op. cit.*[14]
28. Pack, G. T., Scharnagel, I. and Morfit, M. (1945): Surgery, **17,** 849.
29. Pack, G. T. (1956): J. Bone Jt Surg., **38A,** 249.
30. Stehlin, J. S., Lee Clark, R., Smith, J. L. and White, E. C. (1960): Cancer, **13,** 55.
31. Creech, O., Ryan, R. F. and Krementz, E. T. (1959): J. Amer. Med. Assoc., **169,** 339.
32. Pirie, J. H. H. (1921): Med. J. S. Afr., **17,** 87.
33. des Ligneris, M. J. A. (1927): S. Afr. Med. J., **1,** 102.
34. Smith, E. C. and Elmes, B. G. T. (1934): Ann. Trop. Med. Parasit., **28,** 461.
35. Burman, C. (1935 - 1936): S. Afr. J. Med. Sci., **1,** 12.
36. Vint, F. W. (1927): Kenya E. Afr. Med. J., **5,** 383.
37. Elmes, B. G. T. and Baldwin, R. B. T. (1947): Ann. Trop. Med. Parasit., **41,** 321.
38. Davies, J. N. P. (1948): E. Afr. Med. J., **25,** 117.
39. Gelfand, M. (1949): S. Afr. Med. J., **23,** 1010.
40. Wainwright, J. and Roach, G. G. (1957): S. Afr. Cancer Bull., **1,** 167.
41. George, P. A., Fortner, J. G. and Pack, G. T. (1960): Cancer, **13,** 854.

BIBLIOGRAPHY

Fitzpatrick, T. B., Becker, S., Lerner, A. B. and Montgomery, H. (1950): Science, **112,** 223.
Masson, P. (1951): Cancer, **4,** 9.
Szabo, G. (1954): Brit. Med. J., **1,** 1016.
Beerman, H., Lane, R. A. G. and Shaffer, B. (1955): Amer. J. Med. Sci., **229,** 444 and 583.
Cawley, E. P. (1952): A.M.A. Arch. Derm. Syph., **65,** 440.
Wright, C. J. E. (1949): J. Path. Bact., **61,** 507.
Wainwright, J. and Roach, G. G. (1957): S. Afr. Cancer Bull., **1,** 162.
Ellis, F. (1939): Brit. J. Radiol., **12,** 327.
Todd, M. C. (1944): Lancet, **2,** 532.