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MAKING ROUTINE WORK INTERESTING

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Most of us remember one, perhaps two teachers who have helped us to learn and to want to learn more, because we have been inspired by their enthusiasm for their subject. They were able to make study exciting, make the ordinary seem extraordinary and exalt the commonplace as if it were the crown of the subject. This may be one of the most precious gifts in a teacher – to crown the commonplace; to exalt the everyday; to rejoice over the routine; and to overcome the ordinary.

In dermatology, as in many branches of medicine, we are fascinated by the unusual. Meetings are held to demonstrate patients with rare and exotic diseases. In our work, while we may hope to meet the rare and unusual, whether we are a medical assistant or a specialist dermatologist, we deal every day with common conditions.

How can we remain fresh so that our patients can be helped and can be confident about the care they receive? How can we be a valued member of our health care team? I have three suggestions, but they are not listed in order of importance because a priority may differ from person to person.



Depigmentation on the face of a woman with extensive vitiligo. The depigmentation is prominent around the eyes and mouth.

Photo: JJ Nordlund

1. To Read Journals and Books

First, we can read. I welcome this Journal, *Community Dermatology*, because it is designed to help those in the front line of health care, to provide them with a free and accessible Journal which has articles which are relevant to their work. Through reading this Journal, it will be easy to learn current best practice in old diseases and new ideas about the mechanisms which account for the signs and symptoms which they produce. It is only as these mechanisms are broken down, piece by piece, that new drugs can be designed, whether to block mechanisms that are harmful or to augment those that are helpful. Good reading is:

1. **Instructive** - as it will guide towards better practice.
2. **Informative** - as it will give a wider base of knowledge and understanding.
3. **Infectious** - (ideally) when those who have read are so enthusiastic about their new practice and knowledge that they want to pass it on to their colleagues.

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Making Routine Work Interesting

I hope that some of those who read *Community Dermatology* will be encouraged to develop the habit of reading more widely. For those in Africa, Ben Naafs, from his rich experience of tropical dermatology, has written an excellent chapter with many fine pictures on tropical skin disease in the third edition of *Principles of Medicine in Africa*, published by the Cambridge University Press. This book will be a wonderful reference book too as it covers the whole range of the medicine of Africa.

2. To Share Learning and Experience

My second method to keep fresh and enthusiastic follows from the infectious value of reading: it is to share what one is doing and learning with colleagues, senior or junior. As health care has become more complex, and as it is now being forced to adapt to social, environmental and occupational factors which may profoundly affect the management of a patient, much care is now given through the health team. How appropriate, therefore, that any member of such a team should share practice, ideas and enthusiasm with the other members. Team meetings can be made more enjoyable as one enthusiast tells colleagues about what he or she has read. This will not only be good for clinical practice, and so for the patient, but it will also be very good for the morale and spirit of the team.

3. To Keep Accurate Records

So to a third method, one which is particularly relevant for those who struggle with large numbers of patients and the burden of work with little support. Even when time is very full, when demand is heavy, when resources or drugs are limited, it is still possible to keep good

records of what one is doing. Why not get into the habit of recording the problem cases in a notebook or on a computer? As you do this, begin to ask yourself questions about your patients. This is certain to lead to more disciplined clinical practice and so to better care and a brighter outcome for patients. In many countries, and particularly in their more remote regions, little has been recorded about the local pattern of disease so that the authorities lack the accurate data necessary for planning the local health service. Let me give an example. When I was working in an African medical school, we saw a child with widespread oedema, haematuria and a raised blood pressure, the classical signs of acute glomerulonephritis – but why? He was dressed in very ragged clothes and carried a bowl to beg for his food. When we examined him we found that he had scabies; as he had obvious pustules, widespread with the scabies lesions, we concluded that the responsible organism was *Streptococcus pyogenes* and that the glomerulonephritis was due to the immune response to this organism. Little did we realise that at his school many boys had the same infected scabies, some also with evidence of glomerulonephritis. Clinically, we could have gone no further than to deal with his immediate problem of scabies, but this would have been totally inadequate. His management had to be complemented by actively doing something about the other schoolboys. Any intervention would be so much better done by a community nurse; thus, our team could work together to address the significant health care needs of those schoolboys.

Every health care professional, medical assistant, clinical officer, health officer, nurse, medical officer, who reads, who discusses problems with colleagues,

who records data and who asks questions is certain to enjoy their work. Of course, it will be busy and there will always seem to be more to do than can be done, but a lively, interested approach to clinical work can truly overcome the ordinary and will even enable us to rejoice over the routine; inevitably, others in the health team will also be helped.

Continuing Medical Education

Much is written today about continuing medical education (CME) and some less developed countries have already established national programmes of CME, which is probably better called 'continuing professional development' because some members of the health team have little 'medical' work. Programmes may involve outreach visits by specialists from a central hospital and small meetings may be held, so that hours of CME may be added up to meet a required minimum. All such activity can be empty unless it makes a difference to clinical or community practice. It is often very difficult, for example, for isolated health officers or medical officers to keep clinically fresh and enthusiastic. In a health service which wants its health workers to provide better care, such isolated people must be visited by those who can instruct and encourage them, and they must have books and journals. Ideally, they will also make their own personal learning plan with its goals and will have someone to supervise them; they should then be able more easily to reach the goals they have set themselves.

Those of us who have the privilege of being engaged in health care know how very interesting our practice can be. We fail our colleagues if we do not do all we can to help them to read, to consult and meet, and to enquire and record. As these excellent habits become more established among health care professionals in less developed countries, so clinical practice will be sharpened and patients, perhaps long forgotten on account of a chronic disease, will be given renewed care and hope – what better goal?

Note: I am the senior editor of *Principles of Medicine in Africa* but neither I nor any editor or contributor has received or will receive any royalties from sales of the book.



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VITILIGO

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Vitiligo is a skin disorder in which white spots appear on the skin (see photo on front page). These can be localised to one area of skin, one side of the body, or most of the body. Vitiligo afflicts about one of every 200 people throughout the world. People of all ethnic backgrounds and skin colours are equally susceptible to getting it, although the depigmentation is more visible in those with darker skins. It most commonly affects children and young adults but it can begin at any time in life.

To understand what vitiligo is, it is helpful to review the morphology of the skin. The outer layer of skin is called the epidermis. This is composed of three cell types (Figure 1):

- Keratinocytes (approx 93% of cells) which make keratin
- Langerhans cells which are immune macrophages
- Melanocytes (or pigment cells) - about 5% of the cells.

The function of the melanocytes is to synthesise melanin, a pigment that is most commonly brown or black (called eumelanin). The melanocytes synthesise melanin which they transfer into the surrounding keratinocytes (Figure 1). This melanin remains in the keratinocytes as they mature and move up through the epidermis.

What is Vitiligo?

Vitiligo is an acquired disorder (cause unknown) that destroys the melanocytes in the epidermis causing the skin to become white. The depigmented skin on patients with vitiligo is perfectly normal except for loss of pigment.

Types of Vitiligo

There are three types of vitiligo:

- Focal
- Segmental (unilateral)
- Generalised (bilateral).

Focal vitiligo is defined as a few patches of depigmentation scattered haphazardly

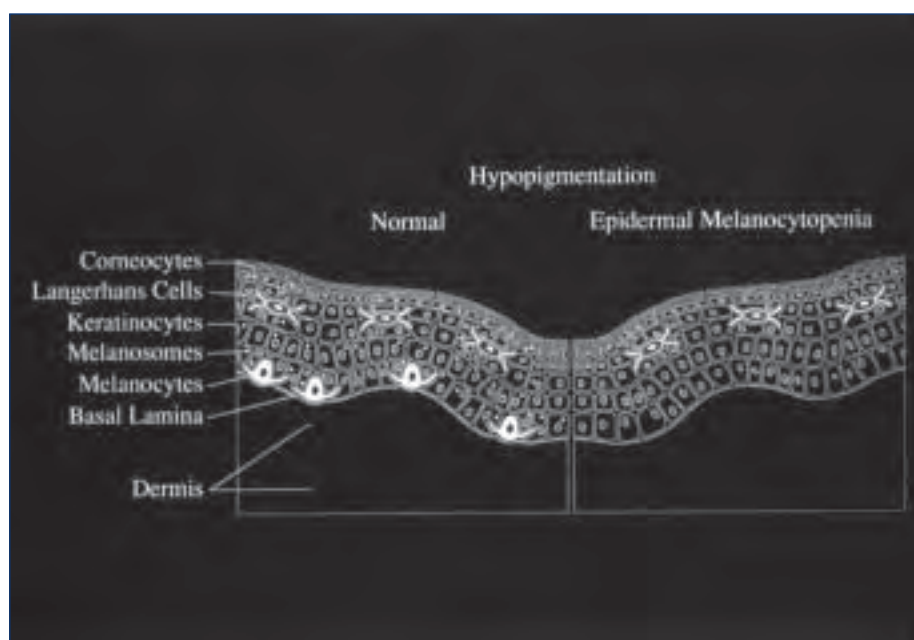


Fig. 1: A schematic drawing of the epidermis. On the left side of the figure, the three main cells of the epidermis are illustrated, keratinocytes, melanocytes and Langerhans cells. Granules of melanin are present on this side. On the right side of the slide, there is a drawing of the skin affected by vitiligo. The melanocytes along the base of the epidermis are missing. There are no melanocytes and no melanin in this skin. Otherwise the epidermis is normal.

Drawing: J J Nordlund

on the skin. Once they appear, they tend to remain for the rest of the patient's life.

Segmental (unilateral) vitiligo affects an area of skin on one side of the body (Figure 2). It might affect one part of the face, one side of the neck, one side of the chest or one leg. The depigmentation stops about at the midline but it does not conform to the cutaneous dermatomes.

Segmental vitiligo can appear at any age but it is more common in children. After its onset, it tends to spread for about 1 to 2 years, after which it does not spread. It has a good prognosis in that it does not spread to other areas of the skin. Segmental vitiligo often affects melanocytes in the hair follicles as well as in the epidermis, so any hairs in the depigmented skin will be white. Patients with pigmented hairs within the vitiligo have an excellent chance of getting their pigment back with treatment.

Those with white hairs in the patch of vitiligo cannot respond to medical therapies and require some form of surgical grafting to regain their colour.

Generalised (bilateral) vitiligo is the most common type of vitiligo. In contrast to segmental (unilateral) vitiligo, generalised vitiligo affects both sides of the body (front page and Figure 3) in a



Fig. 2: Segmental or one sided vitiligo. The upper part of the chest and the shoulder have areas of depigmentation and hypopigmentation. The right side of the chest is spared and the depigmentation stops near the midline. There is normal skin, depigmented skin and light skin. This is called trichrome vitiligo. In a few months, the entire area will be white.

Photo: J J Nordlund



Fig. 3: The hands of a patient with vitiligo. Involvement of the hands, feet and face are typical of acrofacial vitiligo.

Photo: JJ Nordlund

symmetrical pattern. It typically begins bilaterally on the fingers, toes, feet, ventral surface of the wrists and around the eyes and mouth. At this stage it is called acrofacial vitiligo. Very commonly, acrofacial vitiligo progresses so that the depigmentation spreads to the arms, axillae, chest, genitalia and front of the shins. The hairs usually retain their pigment so that generalised vitiligo can respond to medical treatment. The vitiligo process however tends to continue so loss of pigmentation continues for years. Because it continues to be active, patients who respond well to medical treatment often lose the pigment again after the treatment is discontinued.



Fig. 4: Pityriasis (tinea) versicolor. There are patches of partial pigment loss (hypopigmentation) on the back, an area typically spared by vitiligo. Note that the smaller lesions are all less than one centimetre in diameter, have a slightly scaly surface and coalesce to form larger patches. A potassium hydroxide scraping would be strongly positive for hyphae and spores.

Photo: Barbara Leppard

Histology of Vitiligo

A routine biopsy taken from the border between the vitiligo and normal skin and stained with haematoxylin and eosin (H&E) will often look normal. There may be a few mononuclear cells in the epidermis at the junction of the pigmented and white skin. But, if the skin is stained with melanocyte specific stains, it will be apparent that the melanocytes have been destroyed (Figure 1). Normally a biopsy is not needed to make the diagnosis of

vitiligo. It may be necessary to distinguish vitiligo from other skin diseases causing loss of pigment such as hypopigmented mycosis fungoides or discoid lupus erythematosus.

Clinical Appearance of Vitiligo

All types of vitiligo are characterised by totally white or depigmented skin. The skin surface is normal except for the loss of colour. The surrounding skin has normal pigmentation. At times the border area will have partial loss of colour – this is called trichrome (three colour) vitiligo (Figure 2). This partial loss represents vitiligo in progress; the white skin completely devoid of melanocytes, the hypopigmented border having a partial loss, and the normal skin a full complement of melanocytes.

Differential Diagnosis

Vitiligo is almost unique in that it only affects melanocytes so that the affected epidermis is normal in all other respects. Other skin diseases which can be confused with vitiligo are:-

• Pityriasis (tinea) versicolor

This is a minor infection caused by the yeast, *Malassezia furfur*. It causes hypopigmentation rather than depigmentation, i.e., partial loss of pigment rather than complete pigment loss. It is characterised by small

(5-10mm) macules with fine surface scale on the chest, upper back (Figure 4) and neck in teenagers and young adults. A scraping for potassium hydroxide examination will confirm the presence of spores and hyphae. The surface scale makes it easy to distinguish from vitiligo.

• Leprosy

Leprosy is an infection caused by *Mycobacterium leprae*. In the tuberculoid form, there may be one or more hypopigmented, anaesthetic patches of skin (Figure 5); they are almost never depigmented. Some induration might be detected on palpation. A biopsy will confirm granulomatous inflammation in the lesion.

• Piebaldism

Piebaldism is a genetic disorder of depigmentation, inherited as an autosomal dominant trait. It looks like vitiligo but is present from birth and remains present throughout life. Vitiligo is never present at birth, but usually begins after the age of 2-3 years. Like vitiligo, piebaldism also only affects the melanocytes causing the skin to be white but otherwise normal. The lesions are found on the frontal hairline, the face, chest, abdomen and the knees. The hair is always white within the lesions.

• Discoid lupus erythematosus

This is an autoimmune disease in which the basal cells of the epidermis die and the melanocytes empty their



Fig. 5: Tuberculoid leprosy: a single hypopigmented anaesthetic patch on the forearm.

Photo: Barbara Leppard



Fig. 6: A man with disoid lupus erythematosus on the face and nose. The skin is erythematous and atrophic. The margins of the lesions are hyperpigmented.

Photo: JJ Nordlund

pigment into the dermis. It begins with well defined, red, scaly plaques on the face (Figure 6), ears and scalp. As the lesions progress there is atrophy of the epidermis, plugging of the hair follicles and change of pigmentation – both hypo and hyperpigmentation. If the loss of pigment is prominent, it can be confused with vitiligo, but the history of the preceding rash will usually make the diagnosis obvious. If in doubt, a biopsy will easily distinguish between them.

• Pityriasis alba

Pityriasis alba is a mild form of eczema which is common in children. The cheeks, upper parts of the arms and thighs are the most commonly affected areas. The skin is hypopigmented and has a slightly scaly surface (Figure 7).

• Hypopigmented mycosis fungoides

Mycosis fungoides is a malignant lymphoma of the skin. Typically, it presents

as red scaly plaques. However, at times, especially in children and teenagers, it can present as hypopigmented patches on the skin which can be difficult to distinguish from early vitiligo. It does not cause total depigmentation. Usually, a biopsy is required to make this diagnosis and to distinguish it from other causes of hypopigmentation.

• Halo naevus

This is a pigmented mole with a white halo around it (Figure 8). With time, the naevus disappears leaving a round white patch. Eventually, the colour returns to normal. It is most commonly found on the back or upper chest. About one third of young people in their teenage years will have one or more of these benign lesions.

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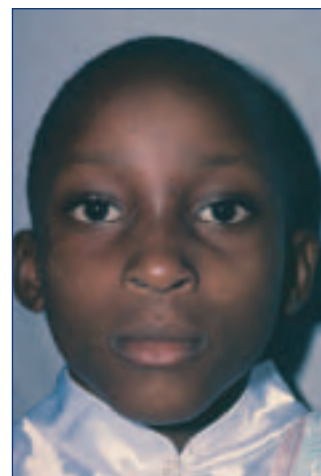


Fig. 7: Pityriasis alba on the face of a child. The patches are hypopigmented rather than depigmented.

Photo: Barbara Leppard



Fig. 8: Halo naevus. This is a normal way for the body to get rid of moles (melanocytic naevi). There is a normal mole in the centre of a white circle.

Photo: Barbara Leppard

TREATMENT OF VITILIGO

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The depigmentation of vitiligo is caused by the loss of melanocytes. To regain the colour, it is necessary for the absent melanocytes to be replaced. The melanocytes must come from the reservoir within the hair follicle (in the outer root sheath and the bulge area). These cells can be stimulated by treatment to divide and migrate out of the follicle into

the surrounding skin. They then appear as perifollicular freckles (Figure 9). The freckles coalesce and the skin repigments.

It follows, therefore, that for treatment of vitiligo to be successful there have to be pigmented hairs within the patch of vitiligo. Some parts of the skin, called glabrous (smooth) skin, have no hair. The dorsum of the fingers is mostly without hair. The skin over the knuckles, the ventral surface of the wrist, the lips, the ankles and feet are all hairless. These areas never respond to medical treatments. Acrofacial vitiligo (see front page and Figure 3 of the article on vitiligo) typically affects the

hands and feet, so successful treatment for such patients will leave them with white hands and feet and depigmented lips.

Generalised vitiligo usually spares the melanocytes in the hair follicles. Occasionally, patients will have small areas of depigmentation with white hairs. Segmental vitiligo commonly affects follicular as well as epidermal melanocytes. Although segmental vitiligo has a limited duration of activity, and, thus, is less likely to spread widely, its ability to destroy follicular melanocytes means it is not treatable with medical therapies.

Possible Methods of Treatment

Topical steroids

Topical steroids are one of the best treatments for vitiligo. They are easy to use, relatively inexpensive, widely available and have few side effects if used properly. It is thought that topical steroids work by suppressing the vitiligo process and stopping the destruction of melanocytes.

There are large numbers of topical steroids - those that are very potent (clobetasol propionate 0.05%) to those that are very weak (hydrocortisone 2.5%). All steroids are applied once daily. For the eyelids, face or body folds, weak or moderately potent steroids (hydrocortisone 2.5% or clobetasone butyrate 0.05% [Eumovate]) can be applied safely for short periods of time. For other parts of the skin, potent or very potent steroids (such as betamethasone valerate 0.1% [Betnovate] or clobetasol propionate 0.05% [Dermovate, Temovate]) are applied. One easy way to use steroids is to have the patient apply them once daily each night before bed for the first two weeks of each month. Applications of steroids will produce repigmentation in at least half of those treated.

Prolonged daily use of potent or very potent steroids should be avoided because the treatment for vitiligo requires use for at least 3-4 months and often for 6 to 12 months. Applications of high potency steroids carry significant risks if applied for these prolonged periods of time, especially when applied around the eyes, on the face or body folds. Complications include glaucoma, acne, irritation of the face, skin atrophy and / or striae.

Systemic steroids

Systemic steroids are best avoided in the treatment of vitiligo because of the potentially serious side effects such as osteoporosis,

diabetes mellitus, hypertension, glaucoma and others.

Topical tacrolimus and pimecrolimus

Both of these medications are calcineurin inhibitors. In recent times, there have been cautions about both these drugs causing lymphomas in mice. The risk is small if they are used carefully. One easy way to treat vitiligo is to alternate applications of steroids with tacrolimus or pimecrolimus. The steroids are applied once nightly for the first two weeks of each month and either tacrolimus or pimecrolimus once nightly for the last two weeks of each month. These cycles are repeated for 3 to 6 months. If a calcineurin inhibitor is not available, then a potent steroid can be alternated with two-week applications of a weaker steroid.

Ultraviolet light

Ultraviolet light is very helpful in treating vitiligo. It is thought to stimulate the proliferation of melanocytes in the follicular reservoir, in contrast to steroids and calcineurin inhibitors which halt the vitiliginous destruction of melanocytes. Using ultraviolet light and topical agents together works very well.

The easiest light to use is natural sunlight. Patients can get about 30 minutes of direct exposure to sunlight 3 to 5 times each week. The exposure to light is combined with the topical treatments described above for optimal success. The

topical medications are applied before bedtime, not before exposure to the sun. The combination of steroids, tacrolimus and ultraviolet light gives the best results - about 75% of those treated getting much repigmentation. It is rare for patients to get 100% of the colour back. The face, legs and dorsum of the arms respond best. The hands, fingers, feet, toes, ankles, wrists, chest and lips rarely respond.

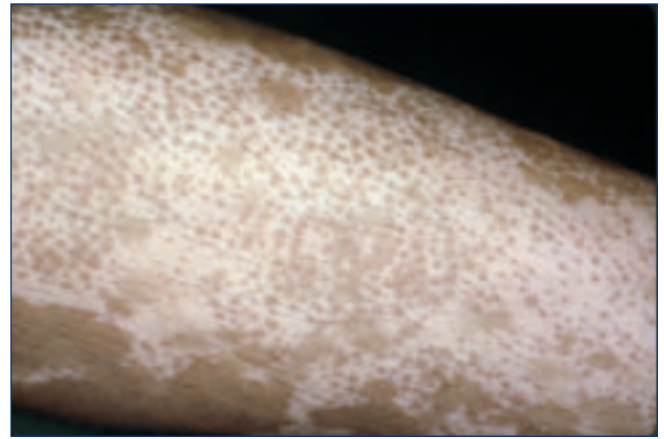


Fig. 9: Vitiligo responding to therapy. Note the freckles which represent melanocytes migrating from hair follicles and producing melanin for the depigmented skin. The presence of pigmented hair follicles is critical for successful treatment of vitiligo.

Photo: JJ Nordlund

It is important to remember that injury of any type can spread vitiligo and depigmentation. Surgical cuts, scratches, inadvertent injuries all can spread the depigmentation. In the same way, sunburn and excessive sun exposure can spread vitiligo. Although the person ideally needs a small amount of light to optimise his chance of repigmentation, excessive light can be harmful. Especially for those with light skin colour, we recommend the use of sunscreens and protective clothing, like hats, when in the sunlight at times other than during treatment periods.

PUVA

In the past PUVA (Psoralen + UltraViolet A light) was the treatment of choice for vitiligo. 8-methoxypsoralen 20-40 mg, and exposure to ultraviolet light or sunlight has been used successfully to repigment such patients. The patient takes the pills and one hour later exposes himself to natural sunlight for progressive periods of time. The first exposure is for 5 minutes usually at noon although other times of the day can be used. Exposures must be done at the same time each day. Subsequent exposures are done every 2-3 days and are increased by 5 minutes until the white skin becomes a mild pink colour the day after treatment. The dose of pills and light are kept constant after that time.

PUVA is difficult for many reasons. The pills are not always available and are expensive. They cause nausea and vomiting in many patients. It is also now known that PUVA can lead to skin cancers 15 or 20 years later (basal cell carcinomas, squamous cell carcinomas and malignant melanoma). In addition, PUVA requires the wearing of UVA blocking glasses for



Fig. 10: Pinch grafts for treating vitiligo. 3-4mm punches of normal skin are removed from an area of normal skin and inserted into small cuts within the patches of vitiligo.

Photo: Barbara Leppard

24 hours after taking the pills. For all of these reasons, topical steroids and calcineurin inhibitors with sunlight are thought to be a better option than PUVA.

Surgical treatment

Surgical transplants (pinch grafts) can be used successfully for carefully selected patients (Figure 10). The techniques are generally expensive and not available except in specialist centres.

Removing residual pigment

Some patients with vitiligo lose nearly all of their skin pigment (Figure 11). Trying to get the colour back in such patients is usually unsuccessful. For these patients, removing the remaining pigment is a better option. It is accomplished by the application of 20% monobenzyl ether of

hydroquinone cream twice daily. This will cause permanent depigmentation where it is applied. It should not be used for normal patterns of vitiligo. It is not readily available and requires some training to use safely and successfully.

Conclusions

All treatments need to be given for at least 3-6 months, sometimes as long as 1-2 years. The patient will need reassurance during the process. Cosmetics can be helpful to hide the disfigurement. Sunscreens are important to avoid burning. Success is never 100% but can be significant for a large number of those affected.

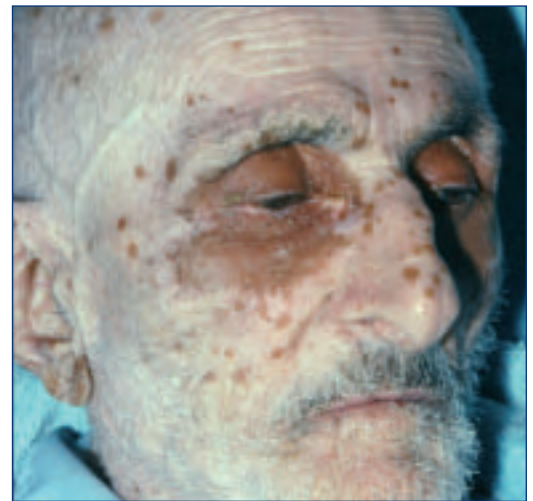


Fig. 11: Almost universal vitiligo. The brown colour around this man's eyes is all that is left of his original skin colour. This is the kind of patient suitable for treatment with monobenzyl ether of hydroquinone.

Photo: Barbara Leppard

Living with Vitiligo

LIVING WITH VITILIGO: A PERSONAL EXPERIENCE

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Vitiligo has been my constant companion for more than 50 years and is an integral part of who I am. Although at times it has almost overwhelmed me and there have been many low points of despair, especially when I was younger, it has contributed in great measure to my achievements and my ability to empathise with people with skin disease and disfigurements.

The Beginning and Course of the Disease

As is commonly the case, the course of my vitiligo has been erratic but with a general tendency to spread. Most people develop vitiligo before 20 years of age. The first white spots appeared on my knees and hands when I was around 12 years old. At that time, I was living in Jamaica where I was born but I do not remember feeling bad about it. My parents and aunt were upset when it was diagnosed but I was much loved and never made to feel different in any way. I had a happy childhood in an extended family of grandparents, aunts, uncles and cousins. I did have some spontaneous repigmentation and for many years no new spots appeared.

Ethnic Concerns

Some of my experiences have been similar to those of others, whatever their ethnic backgrounds. As I am black, most people imagine that I must suffer more than so-called white people. However, it has been shown that skin colour and extent of disease do not always indicate the degree of distress felt by the person with vitiligo.¹ Other factors such as self esteem and love and support from family are very important. Vitiligo can cause great stigma in some cultures where sufferers can be virtual outcasts from their communities. Some women with vitiligo, for example from India or Pakistan, where the disease can be confused with leprosy, have no marriage prospects. Vitiligo is more noticeable on dark skin and the loss of colour can bring a fear of loss of identity. I found it difficult to imagine an all white body and prayed that an effective treatment would be found before this happened.

Progression of the Disease and Psychological Implications

Adolescence can be a very difficult time to develop vitiligo. I was devastated when it spread to my face, particularly my lips, when I was 17 years old. I avoided kissing, and did not feel attractive, believing that no one would want to marry me. I was lucky! I was not teased or bullied at school, which is quite common. Although there is no cure, psoralen used with sunlight or

ultraviolet light (PUVASol or PUVA) can improve the condition, at least for a time. One of my uncles was a general medical practitioner (GP) and he managed to get topical psoralen from the United States which I applied to the patches (except my lips). I wore a lint mask with the eye areas cut out and exposed my face to sunlight in the morning, before going to school. The treatment did help, and for a couple of years the improvement was maintained. But I became very self conscious. This was a particularly cruel blow at a time when I was discovering my own sexuality and the talk among my friends was all about boy-friends.

Covering up with Cosmetic Camouflage Creams

One of the ways of coping with this disease is to disguise the white patches and many practitioners feel that this is the solution to the psychological distress felt by patients. Cosmetic camouflage creams can be prescribed in the UK and a voluntary service to show how to use them is provided by the Red Cross. I came to London in 1959 to further my education and was pleased to find an enlightened GP who prescribed the creams and so I covered up and faced the world. However, it is only a temporary solution, as it is only too easy to hide behind the cosmetic camouflage make-up. It is not possible to cover up your entire body, so if it does spread, as in my case, you have to face up to it.

Is Vitiligo Hereditary?

Many people with vitiligo find it difficult to form relationships and are concerned about the possibility of passing the disease on to their children. I was no exception. I met the man who became my husband during my post graduate teacher training course. It was very difficult for me to tell him about my vitiligo as I was convinced he would walk away. To my amazement, he asked me to marry him. We now have two children and two grandsons, none of whom have so far shown any signs vitiligo. Two thirds of people with the disease have close relatives who have it and recent research shows a genetic predisposition and a link to autoimmune diseases.²

How Vitiligo can Affect your Everyday life

During my pregnancies my vitiligo improved but, after my second child, the white patches began to spread more rapidly. My long, slim legs were affected, and my arms too. I was devastated. I stopped wearing short sleeves and no longer wore shorts in the summer. Holidays became a nightmare and I no longer enjoyed going to the beach with the children. I loved the sea but felt unattractive, exposed and vulnerable in a swimsuit. For more than 20 years I did not have a single item of clothing with short sleeves in my wardrobe.

The Importance of Support Networks: The Vitiligo Society

By the time I was in my mid-40s, I was beginning to get depressed as the condition worsened. I felt panic stricken at the thought of turning completely white. I had by this time given up teaching and was working as an academic librarian in a university. One day, a psychology PhD student who had become a friend, told me about a programme on the TV which was to change my life. It was about a Sri Lankan woman with vitiligo who was setting up a patient support group for people with the disease. I went to the first meeting at St Thomas' Hospital where I saw nearly 150 people with vitiligo. This had a huge impact. I joined the committee of what was then called the Vitiligo Group and later became Chairman of the Medical Research Committee.

In 1993, I took early retirement and threw myself wholeheartedly into the Vitiligo Society, which had become a national charity with a growing membership. I was elected Chairman and then began a whole new life, learning new skills, becoming the face of vitiligo on the media, raising the

profile of the disease among health professionals and giving talks in the UK and abroad. This was a period in which my confidence and self esteem were raised - helping others also helped me. An important part of my coping is knowing as much as possible about the disease. I also came to believe passionately in patient involvement in health care and research which led me to join the Cochrane Collaboration Skin Group (<http://www.nottingham.ac.uk/csg/index.htm>). The lack of good trials for vitiligo was of great concern. With the aim of highlighting this and stimulating research, I instigated and became lead author of the Cochrane systematic review of interventions for vitiligo, published in January, 2006.³

The Cochrane Systematic Review of Interventions for Vitiligo

Nineteen trials with a total of 1350 participants were included in the review. The randomised controlled trials (RCTs) generally had low numbers of participants and only RCTs of repigmentation and not other methods of managing vitiligo were able to be included. There is some evidence, from individual trials, to support short term benefit from topical steroids, various forms of ultraviolet light, including sunlight used with topical preparations (e.g., PUVAso), and other therapies including skin grafting and *Ginkgo biloba*. However, the long term benefit and safety of these treatments have been poorly reported.

The Possible Value of Psychotherapy

During my time as Chairman, my vitiligo continued to spread until it affected 65% of my body. This coincided with the menopause and I was not coping, getting more and more depressed and emotionally drained, while still giving support to others. I decided to have counselling which lasted for 9 months. This was of enormous benefit, raising my self esteem and putting the disease, which had filled my life and threatened to engulf me, into perspective. There is growing evidence to suggest that psychotherapy can be of benefit in the management of psoriasis.⁴ Although the main impact of vitiligo is psychological, we found only one randomised controlled study comparing cognitive behavioural therapy with person centred approach in our search for the Cochrane review.⁵ It will be included in the updated version due to be published in 2008. The study was not conclusive and more studies are needed to estab-

lish the value of this approach for vitiligo patients.

The Challenge of Relapse

As is the case with some other skin diseases, vitiligo cannot be cured at present. Improvement is often short-lived and this should be fully explained to patients. In the early course of my disease, PUVAso and PUVA were both effective for a time. I also became a research patient for 5 years and tried a new experimental treatment, pseudocatalase, in combination with narrowband UVB, which was very successful. I stopped the treatment three and a half years ago and there are now signs that repigmented patches are losing colour. I have now started narrowband UVB monotherapy at my local hospital. Clinicians and researchers should give some thought to ways of maintaining regained pigment and patients should be followed up as a normal part of disease management so that early signs of recurrence can be detected and treated.

Life with vitiligo has been interesting, allowing me to meet people from all walks of life and to embark on many fascinating journeys of discovery. Far from ruining my life, vitiligo has enriched it.

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DIAGNOSIS OF SEXUALLY TRANSMITTED INFECTIONS IN RURAL HEALTH CENTRES

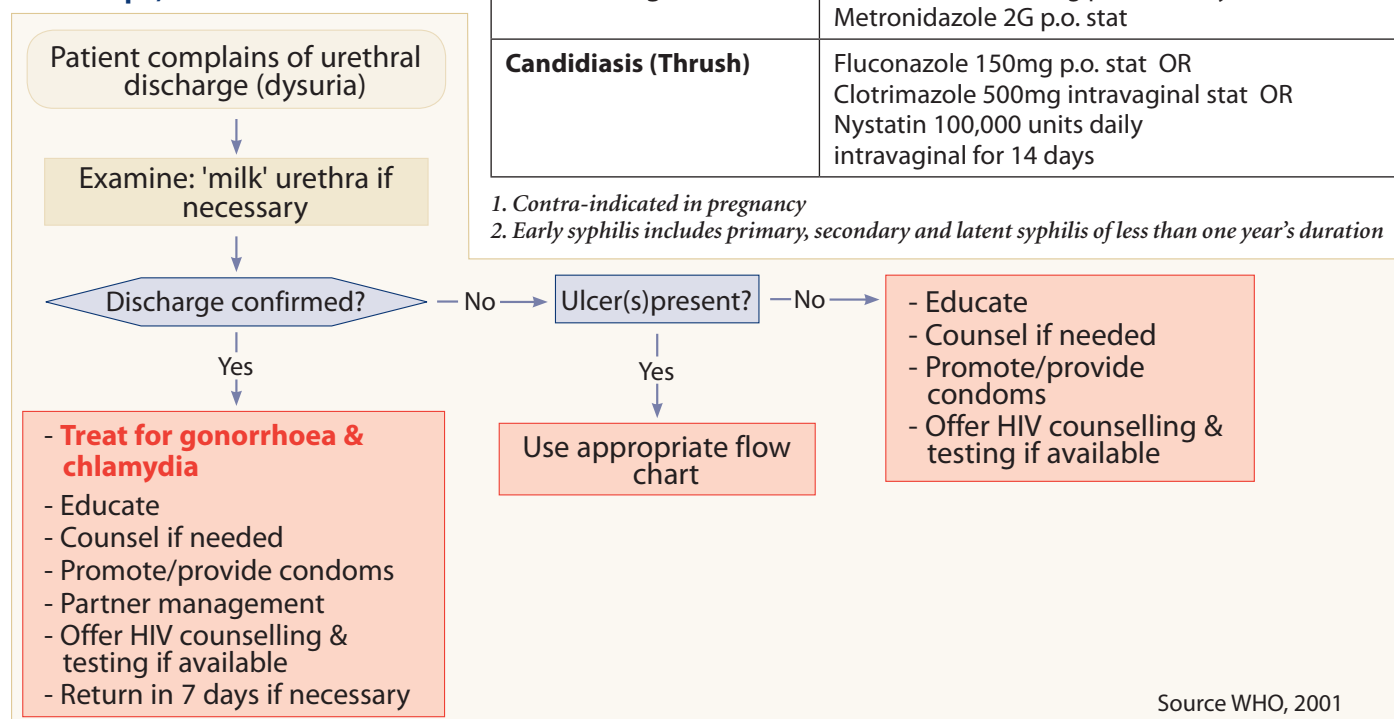
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The World Health Organization (WHO) recommends that, in clinics where laboratory tests are not available, sexually transmitted infections (STIs) should be managed syndromically. That is to say, patients presenting with symptoms and clinical findings suggesting an STI should be treated for all the infections which commonly cause that clinical picture.

WHO flowcharts are available for the syndromic management of urethral discharge and painful, swollen scrotum in males,

Fig. 1: Flowchart for Urethral Discharge in Men (without microscope)



vaginal discharge and lower abdominal pain in females, and genital ulcers and painful swelling of the groin for both sexes.

Urethral Discharge in Men (Figures 1 & 3)

Men with urethral discharge are treated for both the common causes of that syn-

drome – *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. Table 1 shows the treatment regimens recommended by WHO for selected STIs.

If a microscope is available, a Gram stain can be done on a sample of the discharge to diagnose gonorrhoea. *N. gonorrhoeae* are Gram negative cocci, arranged in pairs

Table 1: Recommended Treatment for STIs (WHO)

Infection	Treatment regimen
Gonorrhoea	Ciprofloxacin ¹ 500mg p.o. stat OR Cefixime 400mg p.o. stat OR Ceftriaxone 125mg i.m. stat OR Spectinomycin 2G i.m. stat
Chlamydia	Doxycycline ¹ 100mg p.o. bd for 7 days OR Azithromycin 1G p.o. stat
Syphilis (early)²	Benzathine benzylpenicillin 2.4 million units i.m. stat OR Procaine benzylpenicillin 1.2 million units daily for 10 days
Syphilis (late)²	Benzathine benzylpenicillin 2.4 million units weekly for 3 weeks OR Procaine benzylpenicillin 1.2 million units daily for 20 days
Chancroid	Erythromycin 500mg p.o. qds 7 days OR Azithromycin 1G p.o. stat OR Ciprofloxacin 500mg p.o. bd 3 days
Herpes	Aciclovir 400mg p.o. tds 7 days
Trichomonas vaginalis	Metronidazole 2G p.o. stat
Bacterial vaginosis	Metronidazole 500mg p.o. bd 7 days OR Metronidazole 2G p.o. stat
Candidiasis (Thrush)	Fluconazole 150mg p.o. stat OR Clotrimazole 500mg intravaginal stat OR Nystatin 100,000 units daily intravaginal for 14 days

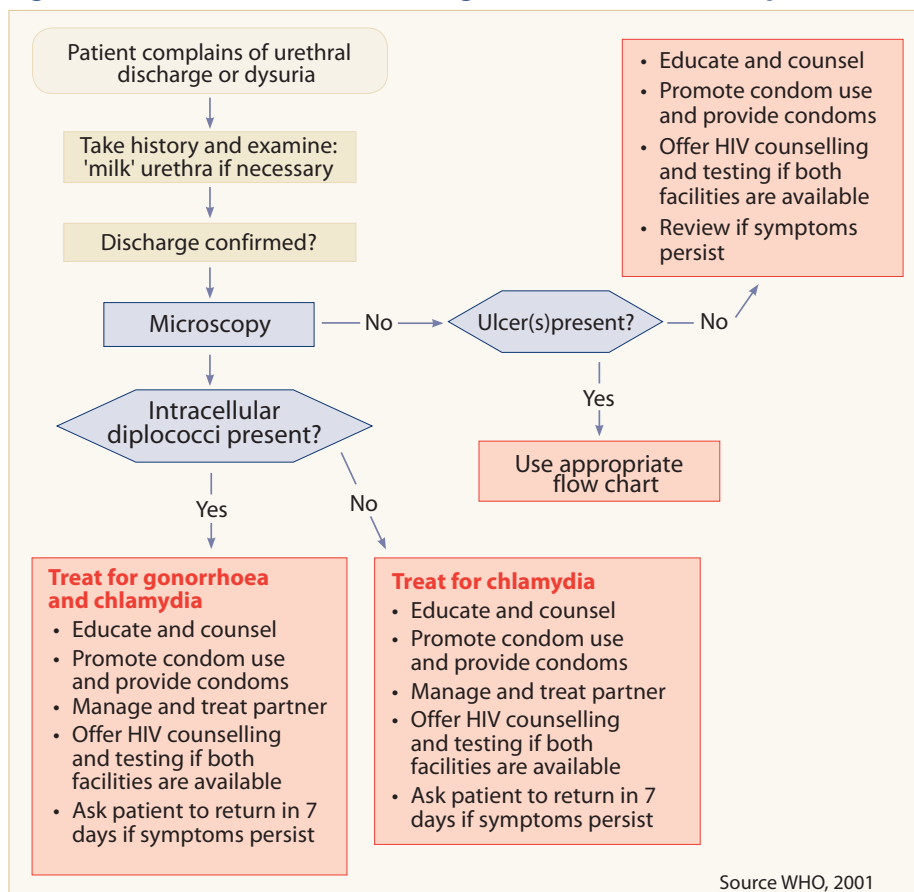
1. Contra-indicated in pregnancy

2. Early syphilis includes primary, secondary and latent syphilis of less than one year's duration

Source WHO, 2001

Diagnosis of Sexually Transmitted Infections

Fig. 3: Flowchart for Urethral Discharge in Men (with microscope)



inside pus cells (Figure 2). If they are seen, the patient is treated for both gonorrhoea and chlamydial infection, since *Chlamydia* cannot be diagnosed reliably by microscopy, and co-infections are common. If *N. gonorrhoeae* is not seen, the patient is treated for chlamydial infection only.

Genital Ulcers (Figure 4)

The three common causes of genital ulcers in the developing world are *Herpes simplex*, *Treponema pallidum* (which causes syphilis) and *Haemophilus ducreyi* (which causes chancroid). Chancroid, a disease particularly associated with core groups such as sex workers and their clients, has become less common. However, genital ulcers due to *Herpes simplex* virus have become more common in Africa as the HIV epidemic has progressed. Ulcers due to herpes usually heal within a few days and do not require treatment, but may be severe and persistent in patients with advanced HIV disease, in whom they may need to be treated with antiviral drugs such as aciclovir.

Vaginal Discharge (Figure 8)

Syndromic management works less well for women with the common complaint of vaginal discharge, which has five common causes (Table 2).

Most cases of vaginal discharge are caused by candidiasis (thrush) or bacterial vaginosis (BV), which are caused by changes in the bacteria normally present in the vagina, and are not sexually transmitted diseases. Thrush can be diagnosed clinically if a speculum examination is possible (Figure 5). BV and *Trichomonas vaginalis* (TV) infection can be diagnosed microscopically: BV on a Gram stain of a vaginal swab (Figure 6), and TV by

looking at a wet preparation showing the characteristic moving parasites (Figure 7). The syndromic management flowchart for vaginal discharge is shown in Figure 8.

If all women presenting to clinics with vaginal discharge were treated for gonorrhoea and chlamydial infection, around 90% of them would be treated unnecessarily. This amount of unnecessary treatment is a waste of precious resources, and exposes many women to side effects of treatment they do not need. Informing women with vaginal discharge that they have an STI requiring treatment of their sexual contacts may put them at risk of stigma, divorce or violence from their spouse or regular partner, and should not be undertaken lightly. The WHO flowchart for vaginal discharge therefore contains a risk assessment step which tries to distinguish between women who need treatment for gonorrhoea and chlamydial infection and those who do not. This risk assessment has been shown to be an imprecise tool in numerous studies.

There is a great need for simple, point-of-care tests for the detection of *N. gonorrhoeae* and *C. trachomatis* that could be performed in primary care settings but, unfortunately, such tests are not yet available. The advantages and disadvantages of syndromic management for STIs are shown in Table 3. Syndromic management works well for male patients whatever the syndrome, and for patients of either sex with genital ulcers. However, it works less well for women with vaginal discharge or lower abdominal pain, which have more possible causes.

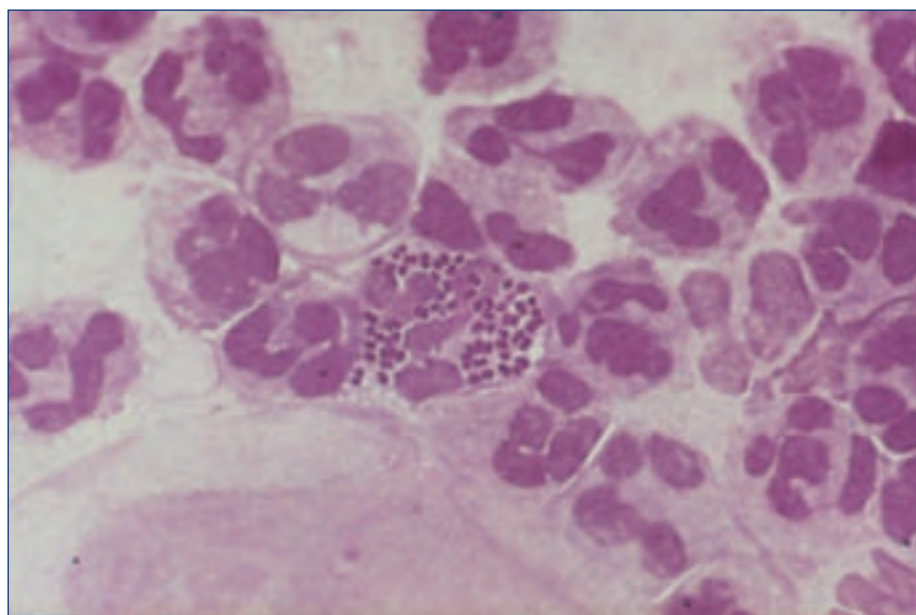


Fig. 2: Gram negative intracellular diplococci

Diagnosis of Sexually Transmitted Infections

Table 2: Causes of Vaginal Discharge

Cause	Sexually transmitted	May cause serious complications	Can be reliably diagnosed by microscopy
<i>Candida albicans</i>	No	No	Yes: Gram stain showing typical fungal morphology
Bacterial vaginosis	No	Probably not	Yes: Gram stain showing reduced proportion of lactobacilli
<i>Trichomonas vaginalis</i>	Yes	No	Yes: Wet mount showing motile, flagellated organisms
<i>Neisseria gonorrhoeae</i>	Yes	Yes	Low sensitivity in women
<i>Chlamydia trachomatis</i>	Yes	Yes	No

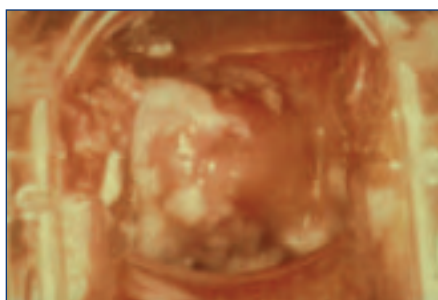


Fig. 5: Vaginal thrush (candidiasis)

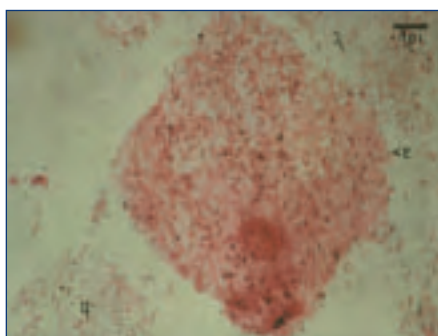


Fig. 6: Gram stain for bacterial vaginosis

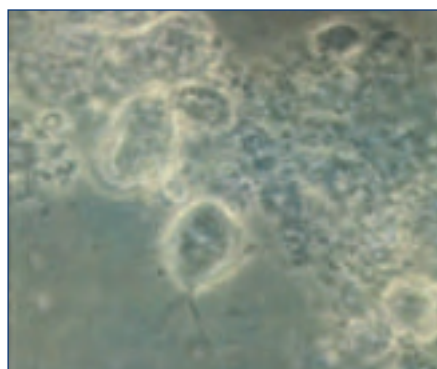


Fig. 7: *T. vaginalis* in a wet prep

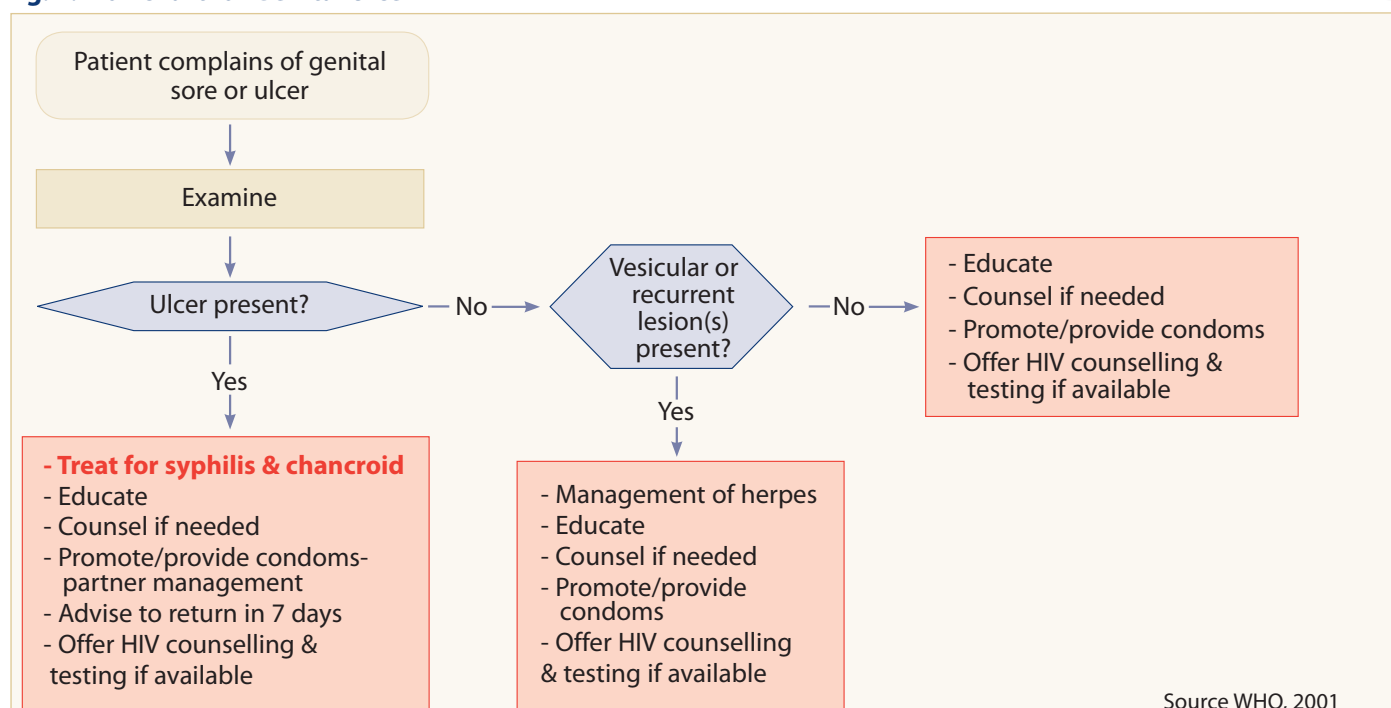
The starting point for syndromic management is a patient with a symptom. Yet many, perhaps most, STIs are asymptomatic. If STIs are to be controlled, it is essential that infected individuals without symptoms should be identified and treated. Until recently, this has not been possible in primary health care settings without access to a laboratory. Fortunately, we now have rapid, dipstick-type point-of-care tests for syphilis that do not require electricity or equipment, and can be performed and interpreted reliably by

health care staff without formal laboratory training.

Multi-site, prospective evaluations conducted by the WHO STD Diagnostics Initiative (SDI) have shown that these tests are comparable to laboratory based reference tests for anti-treponemal antibodies, even when performed on whole blood. These tests will make it possible, for the first time, to screen patients for syphilis in primary health care settings without electricity or laboratory equipment. Highest priority should be given to those at high risk, such as patients presenting with symptoms of other STIs, and – given the serious adverse effects of syphilis on the unborn child – to pregnant women.

The SDI will continue to promote the development, evaluation and adoption of rapid, point-of-care tests for STIs, and to make tests with acceptable performance characteristics available at affordable prices through the WHO bulk procurement scheme. Further information on SDI activities is available at: www.who.int/std_diagnostics/

Fig. 4: Flowchart for Genital Ulcer



Source WHO, 2001

Diagnosis of Sexually Transmitted Infections

Fig. 8: Flowchart for Vaginal Discharge (with speculum/bimanual)

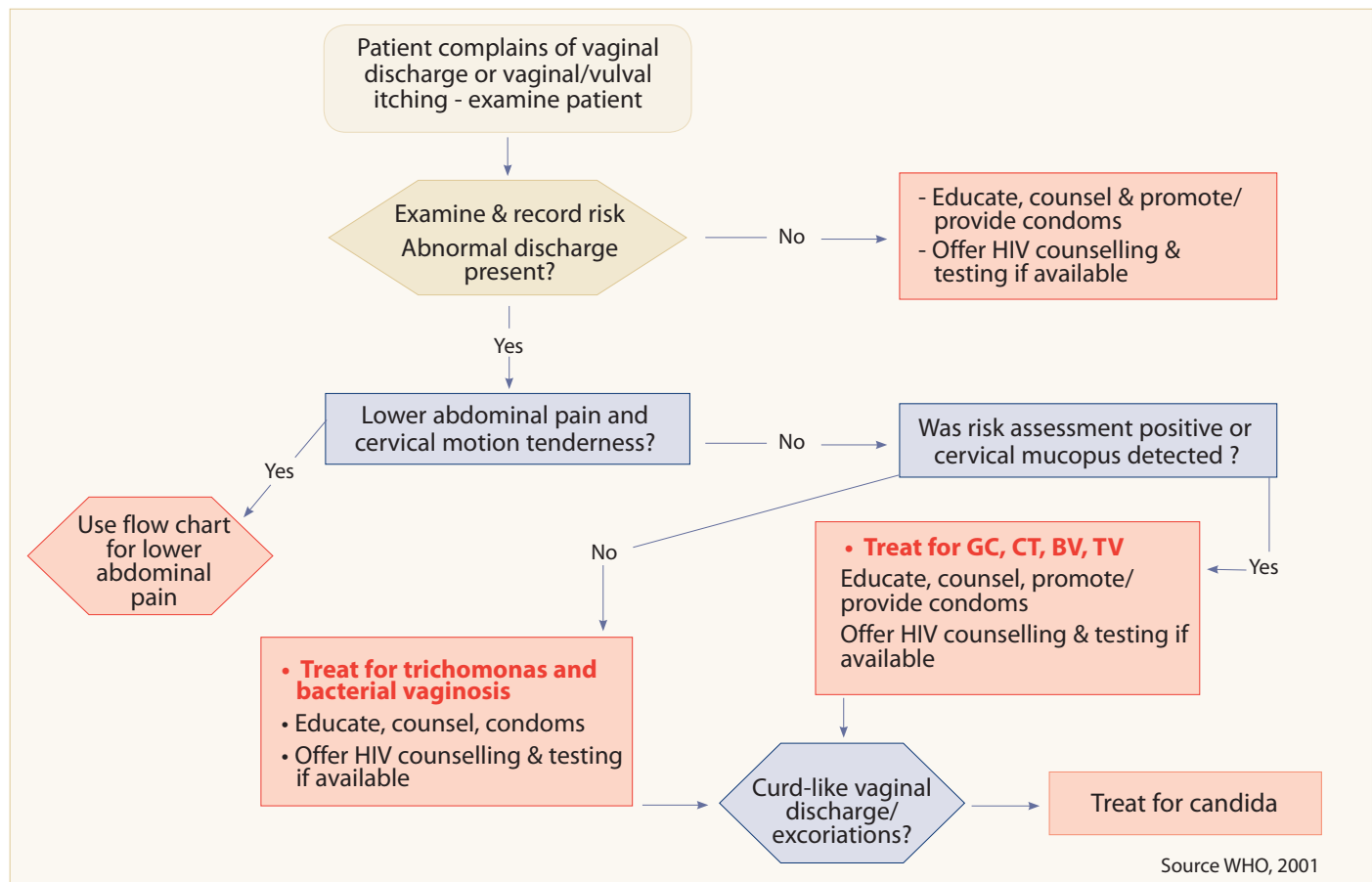
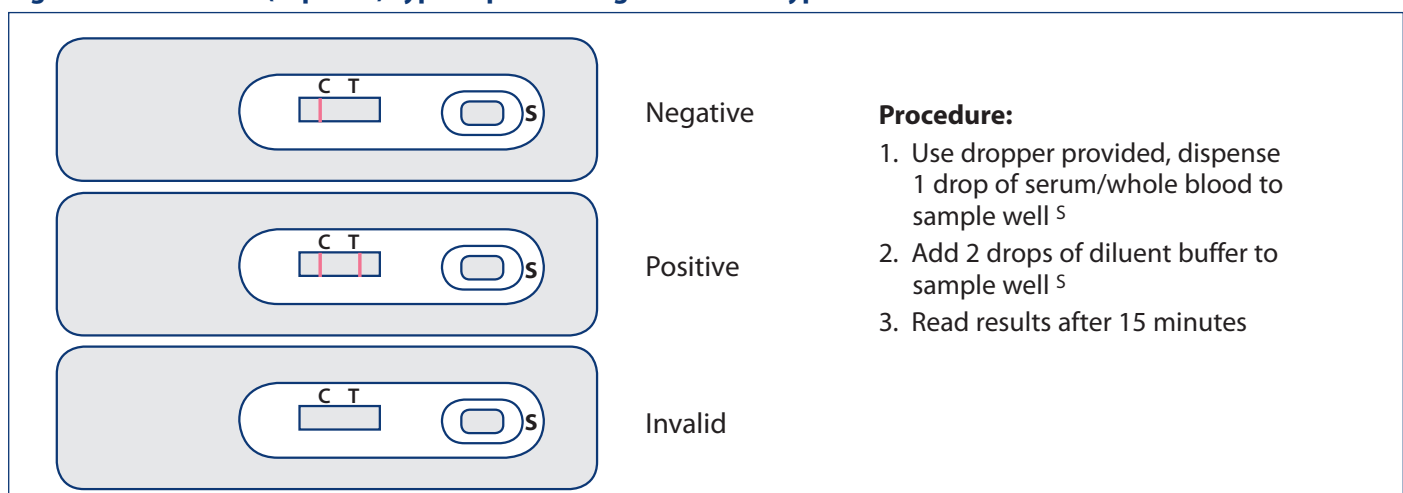


Table 3: Advantages and Disadvantages of Syndromic Management

Advantages	Disadvantages
<ul style="list-style-type: none"> • Problem-orientated (responds to patient's symptoms) • Highly sensitive and does not miss mixed infections • Treatment given at first visit • Provides opportunity and time for education and counselling • Avoids expensive laboratory tests • Avoids unnecessary return visit for laboratory results • Reduces referral to specialist centres • Can be implemented at PHC level 	<ul style="list-style-type: none"> • Overdiagnosis and overtreatment with the following consequences: <ul style="list-style-type: none"> - Increased drug costs - Possible side effects of multiple drugs - Changes in vaginal flora - Potential for increased drug resistance - Difficulties with partner notification • Requires (re)training of staff

Fig. 9: A Lateral Flow (Dipstick) Type Rapid Serological Test for Syphilis



THERE IS A ROLE FOR THE DERMATOLOGY NURSE IN ASIA

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India

Representing the International Skin Care Nursing Group (ISNG) in India

India has a population of one billion, most of whom live where there are no dermatologists. Skin problems are very common but skilled advice is out of reach. When an expert is visited, the consultation is hurried as the queue for advice is very long. Doctors are seen as 'demi-gods', with whom communication is limited, and of whom few questions are asked and almost none clarified.

Experience of working in the UK, and studying the developing qualification of the dermatology nurse, has encouraged me to tell both dermatologists and nurses in Asia what I have learned about this highly evolved, special role, and nurse-led clinics or day care. Perhaps, in India, taking up skin care nursing is perceived as a non-emergency speciality, and for nurses who are unwell or pregnant it is a common 'rest posting'. It is unpopular because it deals with stigmatising conditions that make patients with skin problems fearful to touch and unwelcome even to nurses. There is little incentive in being rotated briefly through 'skins'.

In the rural and suburban locations, the nurse's role is mostly to provide maternal and child health and there is no advocacy for basic dermatological services. Until recently, the management of the most stigmatising skin disease, leprosy, was run by an eradication programme with its own dedicated staff. This was the closest to a skin care programme in India and yet it was unskilled in the care of the more common skin diseases. The skin as an organ, deserving a public health orientated, education programme has not yet been understood.

The potential role of a nurse, with skills as a carer and an educator of individuals and populations, can be identified with placements in outpatients and on the ward, as well as freely moving from both into the community.

In the Outpatient Clinic

To offset the hurried consultation with the specialist, the nurse discusses with the

patient basic knowledge about their problem. Confusing treatments are explained and warnings about side effects are given. Enough time is given to education about caring for the skin and to prevention. Some diseases need much explanation. For example, in the case of vitiligo, it is important to explain it is not contagious and that it is necessary to take care when using the sun as a tool for phototherapy, to avoid its harmful effects. Some therapies, such as steroids, also need much explanation.

On the Ward

The nurse specialising in skin care has the skills and motivation to manage:

1. Acute erythroderma.
2. Drug reactions such as toxic epidermal necrolysis.
3. Reactions in leprosy.
4. Bullous diseases.
5. Psoriasis.

This is because, unlike even the most dedicated general nurse moving between wards, he or she has worked with such problems consistently over a long period. Such conditions require skills in washing, use of emollients and other topical therapy, hydration and prevention of secondary infection. Patient comfort and the application of dressings are enhanced by frequent practice and long experience. Special areas affected by psoriasis, such as the scalp or genitalia, cannot be treated in the same way as the elbows and knees. For patients who have travelled with their relatives very long distances, the additional training that the nurse may give to the accompanying persons can reduce the length of stay and frequency of re-admission to the ward.

Specialist Clinics

My experience in the UK taught me the value of long contact time and lack of hurry in special clinics, such as for the leg ulcers, PUVA, camouflage, and many other nurse-led clinics, where discussion of patient concerns and the learning of self-help skills need to be encouraged.

Community Liaison

A relatively new approach to patient management has been the development of liaison between the hospital and the home. The expert nurse working on the wards or in outpatients is encouraged to visit the home and transfer skills to commu-

nity nurses, auxiliary midwives or family members. This reduces treatment failures on discharge and re-admission is less likely. Importantly, it empowers patients and carers with skills that are adapted to home facilities.

Lack of knowledge about skin hygiene and about cross infection for common viral, bacterial, fungal and parasitic disorders can be remedied by relevant on the spot demonstrations. Explaining myths, but judging the value of integrating traditional practices, can also be very valuable. The practitioners of Indian or Chinese systems of medicine are often very willing to integrate with biomedicine - and nurses are often more sympathetic than doctors to such practice and more likely to be told by the patient about their use.

Conclusion

No human interaction can be successful unless it is culturally sensitive. The nurse-patient relationship is no exception. In Asia, we should not blindly copy the UK's dermatology nurse, but there are common needs and solutions. These can be identified and adapted to Indian needs and practice.

The aim of this short article will have been achieved if the reader thinks about whether there is a need for the skin care nurse model to be recognised in Asia. There are many dermatologists, in the urban areas, and many practitioners of Indian systems of medicine, in rural areas, whose practice would be enhanced if they included nurses as partners and worked with patients, their family members and the nurse, as a team. In this way, they could improve management, especially of the common problems, and they could make sure the time consuming, low technology skin care procedures are well done and the specialist's instructions are understood and carried out. The specialist could then focus on diagnosis and prescribing for 'special' problems as well as the higher technologies, increasingly required for diagnosis or skin ablation and repair.

Where the nurse's role in dermatology is strongly supported by the dermatologist, it is no threat to their private practice, as the contact with a more knowledgeable nurse results in increased awareness of the value of a specialist opinion. □

Research Reports from the Regional Dermatology Training Centre, Moshi, Tanzania

Provided by Barbara Leppard DM FRCP

Assessment of over-the-counter availability of topical steroids in Kampala, Uganda

by the late Jimmy Uketh-Dhogu

Side effects of topical steroids were not seen in Tanzania and Uganda until the mid 1990s, because before that they were either not available or too expensive for most people to buy. In 1997, Jimmy noticed how many patients were attending the RDTC with side effects of topical steroids which had been bought 'over-the-counter' in order to lighten or beautify the skin! He wondered whether they were also widely available in Kampala, thinking that if they were, there would be a problem, since at that time, there was only one dermatology clinic in the country, at Mulago Hospital in Kampala.

He went to 148 dispensing units in Kampala, 37 of each of the following:

- Pharmacies
- Clinics
- Drug shops
- Open markets.

At these dispensing units he looked at which topical steroids were available, how much they cost and whether information leaflets were provided with them, and in what languages. At the same time, he issued a questionnaire to the dispensers to find out whether they:

1. Sold topical steroids on demand.
2. Required a doctor's prescription.
3. Knew anything about the side effects of topical steroids.
4. Gave any advice to individuals purchasing these drugs.

What he found was that 1% hydrocortisone was available in all dispensing units and the stronger topical steroids (*Eumovate*, *Betnovate*, *Mediven*, *Betasone*, *Betalan*, *Betasol*, *Maxobeta*, *Stiedex*, *Synalar*, *Metosyn*, *Dermovate*, *Movate*) were widely available. Worryingly, the stron-

gest steroid (*Dermovate*) was the cheapest everywhere.

All the pharmacists were aware of the side effects of topical steroids, as were 77% of the doctors. Only 30% of paramedical staff and none of the businessmen had any such knowledge. All the pharmacists, 1/3 of the doctors and paramedical staff, but none of the businessmen gave instructions to the people buying topical steroids from them. Instruction leaflets in the tubes were common but only in English and Gujarati; none of them had instructions in the local languages (Lugando and Kiswahili).

He recommended that topical steroids, especially the very potent ones, should be licensed and no longer available to be sold by whoever wants to sell them. That would put an end to 'over-the-counter' sales and would ensure that they were only available with a doctor's prescription. Unfortunately, in the 10 years since this recommendation it still has not happened. □

Assessment of the use of cosmetics and skin lightening agents among teenagers and adults in Nairobi, Kenya

Nicholas O Ochieng

Nicholas did this project because so many patients were attending clinics in Nairobi with side effects of cosmetics and skin lightening agents. He visited 831 individuals (aged 14-52) at home in 5 different areas of Nairobi (so that individuals with low, medium and high income were included). For those who could read and write, he had them fill in a questionnaire about their use of cosmetics and skin lightening agents in his presence; for those who could not, he filled the forms in for them. He looked at the products they had at home and noted their contents where stated.

He found a very large number of products being used with some interesting names! Some contained bleaching agents normally used for clothes, e.g.,

Jik, a powdered detergent, *Omo* - and a popular brand of toothpaste, *Colgate*. Others contained hydroquinone in very high concentrations, topical steroids and mercury.

More than 75% of females and 43% of males said that they used cosmetics. Sixty-three percent (63%: equal numbers of males and females) admitted to using skin lightening agents. Most had not been issued with any information, or warning about these products and about 20% had noticed side effects on their skin.

He found it very worrying that potentially harmful drugs were

freely available and being sold as 'cosmetics' to the unsuspecting public. □

Product name	Chemical	Content
Envi	Hydroquinone	12.8%
Cleartone	Hydroquinone	14.3%
Princes Patra	Hydroquinone	9.4%
Ambi	Hydroquinone	1.1%
Dorot	Mercury	1.9%
Drulla	Mercury	3.0%
Laevate	Betamethasone	0.1%
Betnovate	Betamethasone	0.1%

Commercially available skin lightening agents and what they contain

Hydroquinone	Corticosteroids/steroids	Mercury	Mixture of two or more chemicals	Those of unknown chemical compounds
<i>Ambi</i> <i>Cleartone</i> <i>Miki</i> <i>Tura</i> <i>Mekako</i> <i>Amira</i> <i>Dear Hear</i> <i>Envi</i>	<i>Movate</i> <i>Desonide</i> <i>Top Society</i> <i>Synalar</i> <i>Dermovate</i> <i>Laevate</i> <i>Hydrocortisone</i> <i>Betaderm</i>	<i>Rico</i> <i>Drulla</i> <i>Dorot</i>	<i>Cocoa butter</i> <i>Palmer's skin success</i> <i>Top Gel</i>	<i>Venus de Milo</i> <i>Fair and Lovely</i> <i>Shirley</i>

The chemical content in a random sample of some skin lightening agents by the Kenya Government Chemist analysis

A SHORT GUIDE TO HELPING YOURSELF AND THUS HELPING OTHERS IN COMMUNITY DERMATOLOGY

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Dr Michael Waugh was a consultant in sexually transmitted infections in a busy teaching hospital in the North of England. His early professional career was in dermatology and he has always been interested in the concept of dermato-venereology. Dr Waugh has travelled in Russia and Eastern Europe many times since the end of communism and has regularly visited and taught throughout Asia. He has also taught frequently at the Regional Dermatology Training Centre at Moshi, Tanzania, in the last 10 years.

Aims

This is a short and, hopefully, helpful guide - to help not only you but those around you; your family and friends, and those you are asked to help, your patients.

The Guide

'Manners maketh man'. A wise saying - to help oneself but also to enable one to help others, because it requires some discipline.

Oneself

A healer's day will be long with potentially infinite demands. Thus, you need to adopt a balanced lifestyle which allows time for

self, family and friends, and a professional life. It won't help patients if you are not well.

On getting up - reflect, be quiet and praise your God or gods, never asking for yourself, but for what you can do for others. Try to organise a schedule for the day which allows for other requests on your time to be adopted as well. Eat a good breakfast; you need to get through a morning, in most cases seeing lots of patients. Try to get some short exercise before the day starts. Incorporate your own health maintenance as part of your professional life.

Throughout the day, take short breaks of say 10 minutes from work once an hour. It's too easy to become too tired and tense. Stop for a midday meal for at least half an hour. You don't work any more efficiently by not stopping...

Establish or participate in local professional support networks, such as mentoring programmes, professional supervision or formal professional groups. Recognise that both professional and personal life will be affected by work-related stress and learn about the physical and emotional characteristics of excessive stress and burn-out.

You will always have more to do than you think you can do, but remember an excessive work load tends to cause the sort of depression that often affects high achievers. For two hours before going to bed do 'quiet things' and remember to keep them simple; not easy for a busy mind. Never go to bed hating anybody or in a bad temper.

Your Profession and Patients

Try to find time to keep up-to-date with your practice. Let's say one hour a day throughout your life.

Remember, it's a privilege to be able to help the sick. Remember you are the servant of others at that time. Your whole being must listen, observe, smell and examine - as well as you have been taught - those patients who have come to be helped by you. If you, yourself, are peaceful and observant you will find it so much easier to help your patient and to teach your students and the families of your patients. Try not to hurry, try not to raise your voice, and learn to remember small details. These will come with practice. Your brain is much better at remembering details, if trained, than any computer.

Remember the culture in which you practise. There are great differences in how humans interact throughout the world. Remember and learn the everyday customs of social interaction in whichever area you practise.

Remember the emotional problems you may have dealing with very ill children, and the differences between men's and women's ways of dealing with health and personal problems. Remember the impact of death on your patients, and yourself.

Remember that professional practice should be a joy always. If you don't find it so, try to find a mentor with whom to discuss the problem. But for most of us, every day brings its rewards - the immense joy of our work.



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