PITYRIASIS ROSEA

A REVIEW OF ITS CLINICAL ASPECTS AND A DISCUSSION OF ITS RELATIONSHIP TO PITYRIASIS LICHENOIDES ET VARIOLIFORMIS ACUTA AND PARAPSORIASIS GUTTATA*

JAMES MARSHALL, M.D.

Department of Dermatology, University of Pretoria

Pityriasis rosea (P.R.) is a relatively common skin disease of world-wide distribution. The common macular type was first described by Gibert in 1860, and since this time many modifications and varieties have been added. Historical details can be found in an article by Percival[1] and in the *Nouvelle Pratique Dermatologique*.[2]

P.R. is a self-limiting disease of young people with certain characteristics which are clearly seen in many cases. It often begins with a solitary (large) lesion, the herald patch, which appears almost always on the body and is followed after a few days to a few weeks by a secondary disseminated eruption of smaller lesions (often in hundreds) over the body and limbs. The secondary rash appears in crops over a week or two after which the lesions gradually heal and disappear without scarring in 6-12 weeks. Symptoms are mild and usually unimportant; itch is often present in the early days but is rarely severe or persistent. The course of P.R. has been compared to that of early syphilis and the exanthemata. Most patients are children or young people. Second attacks are very rare. The disease is endemic and most cases are seen, the world over, during the colder months of the year. Apart from seasonal fluctuations there are also times when case incidence reaches proportions deserving the term epidemic. The number of occasions on which more than one case has occurred in a household or in other closely associated people is not large.

The course of a typical case of the macular variety is as follows. The herald patch appears on the body as a ringed or oval plaque, from 2-5 cm. in diameter, which may enlarge peripherally still further. The border is red, finely scaling and slightly elevated; the centre is flat and pink or yellowish-brown and healing begins there before it does in the edges. The herald patch, especially if it is on the back, may not be noticed by the patient.

A few days to a fortnight after the herald patch appears the secondary rash begins to erupt and lesions appear in crops over the body, neck, upper arms and thighs for about a fortnight. The secondary lesions are usually of two main types, (*a*) small pink finely-scaling macules, 2-10 mm., and (*b*) round or oval nummular lesions (medallions), 10-30 mm., with a pink, slightly elevated, finely-scaling edge and a flat or depressed, yellowish, atrophic-looking centre. The skin in the centre is like tissue paper and may also scale off. These lesions resemble the herald patch and may run together to form large circinate plaques. Some of the macular lesions may alter to the nummular type.

Healing begins after 2-4 weeks, first in the lesions that appeared earliest, and is usually complete by 6-12 weeks when all scales have fallen off leaving an unblemished skin. Rarely the sites of the lesions remain slightly depigmented or hyperpigmented for a short time after healing is complete.

The histological features, to be detailed later, are those of an eczematide.

Although most cases of P.R. (78% of 1,556 cases, according to Benedek[3]) conform more or less to the above description a great variety of untreated cases differing in behaviour and clinical features from the macular type have been described. A form of classification, modified from that of Klauder,[4] is shown in Table I.

The Herald Patch

The herald patch, usually solitary, is almost always found on the body; and there are no particular sites of election. It is rarely seen away from the covered areas of the body but cases are described where it has been found on the face, palms, soles or genitals (even the glans penis) and I have recently seen a case where it occurred on the scalp. It is usually 2-5 cm. in diameter (larger than the secondary lesions) but may be smaller or very much larger (giant herald patch). It may pass unnoticed by the patient and be identified only by its size, or it may apparently be absent. Rarely there may be 2 or even 3 herald patches. Atypical lesions, although less common than in the secondary eruption, are sometimes seen.

When the herald patch is seen before the secondary rash has appeared it is frequently diagnosed and treated as tinea corporis; and the stronger fungicides such as iodine and Whitfield's ointment may cause it to become eczematous. If the lesion is in the axilla or groin it may be very difficult to differentiate from tinea axillaris or cruris.

The herald patch remains solitary, in most cases, for 4-14 days, but the secondary rash may appear with it or only after a much longer delay of as much as 6-7 weeks. There are authentic cases where the herald patch has not been followed by a secondary rash.

The Secondary Rash

The lesions of the secondary rash are usually widely and symmetrically distributed over the body, neck and adjacent parts of the limbs; those on the back and in the axillae often lie along the lines of the ribs. The palms and soles are very rarely affected. Degos,[5] like many other observers, states that pityriasis rosea 'almost always spares the face and always spares the scalp'. This is not correct; Haxthausen[6] has drawn attention to the fact that the scalp is usually affected in children and the face

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

and scalp are quite often affected in the Bantu, children and adults, of South Africa (Figs. 1 and 2). Scalp lesions are, of course, more easily visible in the Bantu because of the short hair. The secondary rash may remain relatively localized around the herald patch on any part of the body. I have several times seen cases in men in which the lesions were largely confined to the untanned bathing-trunk area, only faint fleeting lesions being

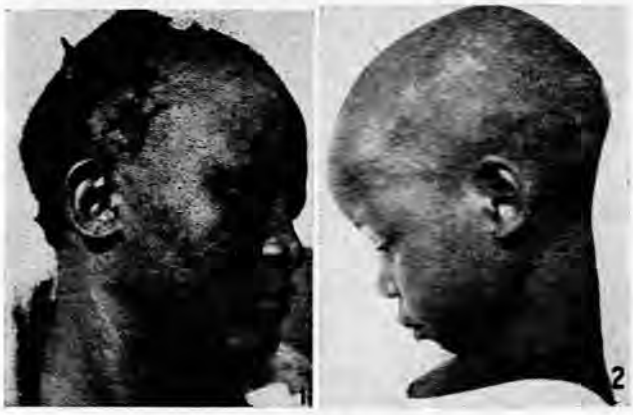

Fig. 1. Pityriasis rosea of the face in a Bantu.
Fig. 2. Pityriasis rosea of the scalp in a Bantu child.

present elsewhere; and this phenomenon has been noted by others. This beneficial effect of sunlight may well explain the relative rarity of lesions on the face and extremities.

Apart from the macular lesions those most commonly seen are vesicular, urticated and small papular. Vesicles, in untreated cases, are rarely larger than pinhead size, but occasionally the description varicelliform may be warranted. Pustules are also sometimes seen. When urtication occurs the oedema is usually confined to the edges of the lesions and very rarely is urticaria simulated. Among the papular forms the lichen planus-like variety is common (in South Africa at least), and tiny lichenoid papules are thickly set in the edges of the lesions and scattered in the immediate surroundings.

I have included in my classification the *Pityriasis circiné et marginé* of Vidal. Although Gougerot[2] and some other observers classify it separately the majority consider it simply a type of P.R. in which the lesions are fewer, larger and of longer duration. They may be distributed mainly in the axillary and crural regions, resembling closely the lesions of tinea cruris et axillaris.

The other varieties shown in the table need no further explanation. It is rarely that one sees a case in which all the lesions are of some unusual type; in most cases among the atypical lesions are some typical macular or nummular lesions, and perhaps the herald patch, to point to the diagnosis.

Healing, in an uncomplicated case, takes place without leaving any scar. Post-lesional depigmentation or hyperpigmentation of unimportant degree may remain. Hyperpigmentation is relatively common in the Bantu and may take some months to fade entirely.

Vesicular, bullous and erosive lesions of the buccal mucosa have been reported by a few observers and Wile[7] has described lesions on the vulva. I have seen only one case with mucosal (buccal) lesions; tiny haemorrhages and pinhead erosions were scattered over the cheeks in a European with severe diffuse vesicular P.R. affecting also the face.

The secondary rash appears in crops over a few weeks and all lesions have healed, in the average case, in from 6-12 weeks. There are, however, cases in which fresh crops of lesions continue to appear for very much longer. The case of longest duration that I have seen lasted just over a year; the clinical picture was of a typical macular P.R.

Differential Diagnosis

In a typical case of the macular variety with an obvious herald patch the diagnosis is rarely difficult. *Pityriasis versicolor* may be simulated, but in this condition the lesions are generally confined to the chest and shoulders, the course is extremely chronic and the causative fungus is very easily demonstrable in epidermal scales. *Pityriasis sicca* (streptococcal eczematide) may cause difficulty in children, but the lesions here are often confined to the face (which is seldom spared) and are rarely so diffuse as in typical P.R. Untreated, it lasts longer than P.R. and recurrences are common. *Psoriasis*, except in an acute guttate, punctate or small nummular attack bears little resemblance to P.R. Its lesions are papular and infiltrated and the diagnosis can, if necessary, be established by biopsy. The lesions of chronic *seborrhoeic dermatitis* (*eczématides figurées*) on the chest and back may resemble closely those of P.R. but their distribution, often in the centre of the back and chest, greasy scaling, chronicity and tendency to recurrence are distinguishing features. The seborrhoeic skin is no more susceptible to P.R. than is any other. *Lichen planus* may be simulated very closely by the small papular variety of P.R. but can easily be identified by histological examination. It is interesting to note that cases of lichen planus with a 'herald patch' have been described. There is no suggestion, however, that the two conditions are in any way related. *Tinea corporis* may be confused with vesicular P.R. but the lesions are rarely so numerous as those of P.R. and the fungus can be identified in scrapings from the lesions. Dermatophytids may also resemble the lesions of lichenoid P.R. but the primary fungal infection is usually obvious. *Secondary syphilis* may copy most of the varieties of P.R. and if no other sign of syphilis such as mucosal lesions, adenopathy or an active or healed chancre are present the result of a serum test for syphilis will be necessary to confirm the diagnosis. The *exanthemata*—measles, german measles or varicella may rarely be simulated by P.R. especially in cases with general symptoms.

Points of resemblance between P.R. and pityriasis lichenoides et varioliformis acuta and parapsoriasis guttata will be examined later.

General Symptoms

About 25% of cases complain of a little *itch* in the first week of the secondary rash; a small percentage of patients have severe itch that may last for several weeks and be most intractable. Other signs and symptoms are rare; they include *malaise*, *headache*, *adenopathy*

(glands in the posterior triangle of the neck especially), *low fever*, and fleeting *joint and muscle pains*. Such phenomena occur at the onset of the disease. *Tonsillitis*, *whitlows*, and other *infections* have been noted before the onset of P.R. I have seen one case with a mild *hepatitis* and *icterus* accompanying the secondary rash. When general symptoms occur they resemble those of the milder exanthemata. No characteristic changes in the blood or any other organ have been noted.

Incidence

The case incidence of P.R. varies, according to the American and European observers quoted by Percival,[1] between 3 and 10 per 1,000 patients with skin disease. One author cited, Nekam, gave a much higher incidence of 40 per 1,000. The incidence in the Transvaal among Europeans is about 18 per 1,000 cases with skin disease, and my impression is that the incidence among the Bantu is the same. The figure for Europeans in South Africa is significantly higher than the average in the USA and Europe.

No significant difference in incidence of P.R. in males or females has ever been noted. The disease has been reported from every part of the world and as far as South Africa is concerned it does not seem to have a predilection for any particular race. Niles and Klumpp,[8] however, report only 2 cases in Negroes among 219 American patients of all races. P.R. tends to be more severe and generalized in the Bantu than in Europeans in South Africa.

P.R. is most frequent in patients between 20 and 30 years of age, and occurs almost always between 5 and 50. It is very rare over 50; and the youngest patient I have seen was in his 2nd year.

Most writers state that P.R. occurs most frequently either in spring or autumn or in the colder months of the year. From my records the same seems to be true in South Africa, where the lowest numbers are seen at midsummer in January and February; and there are significantly higher figures in the cold months. Apart from any seasonal changes in incidence there are periods when the disease seems to be epidemic. This is shown in Percival's figures[1] from Edinburgh and has been referred to by many other observers. Such an epidemic in the Transvaal occurred in 1950-52. In spite of its apparently epidemic incidence it is exceptional to find even the most casual relationship between patients. The occurrence of two or more cases within a family or in some institution is rare enough to have warranted publication on a number of occasions.

Recurrences

Second attacks of P.R. are extremely uncommon. In such cases as have been reported the second attack has occurred after some years, e.g. after 4 years in Darier's patient.[9] In the one case I have observed a second attack followed more than 30 years after the first.

Cause

The cause of P.R. is still unknown, but many theories have been put forward. It has been suggested that P.R. is due to infection by a fungus, streptococci, staphylococci, a spirochaete or a virus; that it is a tuberculide, a toxic exanthem due to gastro-intestinal auto-intoxication, or a neurotrophic dermatosis.

The fungus theory had some supporters, notably the late Professor H. Gougerot,[2] who believed the cause of P.R. to be the *Cryptococcus du Boisii* (*C. anomoeon*). This organism can be found in the scales of most cases of P.R. as masses of spores in the follicular sheaths. The dry scales must be mounted direct in xylol and a careful search 'for an hour if necessary' will discover the organisms. This fungus has never been cultured. The majority of writers consider it a saprophyte. (Vidal considered the cause of his *pityriasis circiné et marginé* —now identified with P.R.—to be a similar or identical fungus, which he named *Microsporon anomoeon*). The fact that P.R. affects the scalp frequently in children but rarely in adults is of course reminiscent of the microsporon infections.

Streptococci have been suggested as the cause of P.R. by a number of authors, notably Périn (cited by Gougerot[2]) and Gourvitch,[10] on the basis of finding streptococci in the scales, positive intradermal reactions to streptococcal vaccine, and the appearance of P.R. in patients who were suffering or had recently suffered from manifest streptococcal infections.

The spirochaete theory is considered in an article by Hollström.[11] Following the work of Lennhoff, who was able to demonstrate spirochaetes in specimens of skin stained with mercury sulphide, Hollström cultured spirochaetes from 5 cases, and even managed to subculture them. He claimed that the course of P.R. was markedly shortened by treatment with bismuth or arsenicals. Objections to Hollström's theory are that his bismuth-treated cases recovered as quickly as those treated with the much more potent spirochaeticide arsenic; and that patients under treatment for syphilis may develop typical P.R. (apart entirely from P.R.-like drug reactions). It has recently been suggested by Schirren[12] that the spirochaetes discovered by Lennhoff in P.R. and other skin diseases are simply artefacts of various kinds.

The virus theory is the one most widely held at present. P.R. generally behaves in its course, age incidence and epidemiology, like one of the milder exanthemata and an attack almost always confers life-long immunity.

The portal of entry of the organism, whatever it may be, is also disputed. Contact or droplet infection can obviously be excluded. The wearing of new clothes before the attack, using an affected person's clothes or sharing a sufferer's bed, going to public baths, have all been suggested as factors in a few or many cases. The most plausible idea, to my mind, is that of Louis Brocq who suggested that P.R. might be spread by an insect vector, the flea, and that the herald patch is the site of inoculation. The secondary rash could well result from embolic distribution of the causative organism after its multiplication in the primary lesion; the cutaneous reaction might be one due to infection or to allergic response after the patient had become sensitized. The herald patch is nearly always on the trunk, the site of election for flea bites. The flea theory would fit in with the clothing and public-bath observations mentioned above. Flea-bites are very common in South Africa,

especially among children, for nearly every household has at least one domestic animal. Against the flea theory is the fact that P.R. is commonest in the cold months.

Hissard[13] reports a case where the herald patch appeared a few days after a wasp-sting on the cheek and was followed after 12 days by a typical secondary eruption (but this, of course, cannot be used in support of the flea theory).

Passage

Many experiments in passage of P.R. to animals and man have been made in the past, but no convincing successes have yet been reported. The materials used have been scales and blister fluid (produced by cantharides plasters or CO_2 snow) from herald patch and secondary lesions inoculated by scarification or intradermal injection. Wile,[7] after a long series of failures, produced an eruption in three volunteers by using blister fluid from the herald patch of a case occurring during an 'unusually severe epidemic'. After 3 or 4 days a sparse, itchy, papular eruption appeared, but there was no herald patch at the inoculation site and in no case did the rash last more than a week. Thomson and Cumings[14] had one possible successful transmission with filtered saline extract of scales; and they cite the case of Edelston who, after handling a case of P.R. accidently scratched himself and developed a herald patch at this site and later a typical secondary rash. Gourvitch,[10] and Joyeux, Burnier, and Duché,[15] report unsuccessful trials of passage.

We have made some experiments in passage using ground-up whole skin in a saline-penicillin-streptomycin solution. The fluid was injected intradermally. No reaction whatever was produced and this suggests that the eruptions described by previous experimenters in this field may well have been streptococcides.

If a flea is the vector it is possible that its mouth secretions may contain some substance that facilitates entry of the causative organism into the body; or it might be that the organism must pass a part of its life history in the flea before it can once more cause disease in man. Experiments in which we added ground-up fleas (from a dog) to the above suspension were also unsuccessful.

Attempts to culture a virus on human epithelium and on monkey kidney have so far been fruitless. The possibility of 'silent' growth was excluded by negative results from injection of protein-free washings from the media.

The failure of experiments in passage and in culture does not exclude the possibility of a virus as cause. The culture media used so far may not have been appropriate; and we have not yet tested the possibility that the causative organism may have to multiply or undergo some change in the flea before it is again transmissible to man.

Treatment

The average case requires no treatment beyond reassurance about the prognosis. Over-treatment leads to eczematization of the lesions and increase of symptoms. If itch is severe, one may prescribe baths in solution of potassium permanganate or sodium bicarbonate, calamine lotion with 2% phenol, and aspirin, antihistaminics or even barbiturates for sedation. Sunshine in reasonable doses definitely speeds resolution (the predilection for untanned areas has already been noted). I have used parenteral arsenic and penicillin without the slightest influence on the course of P.R.; and the more recently discovered antibiotics used for virus diseases were equally ineffective. Convalescent serum has been used without significant effect. Cortisone and ACTH had no effect in a few cases tested.

PITYRIASIS ROSEA, PITYRIASIS LICHENOIDES ET VARIOLIFORMIS ACUTA AND PARAPSORIASIS GUTTATA

Pityriasis rosea is mentioned by most authors in discussing the differential diagnosis of two relatively rare conditions parapsoriasis guttata (Para. G.) and pityriasis lichenoides et varioliformis acuta (P.L.V.A.) and it has been tentatively suggested in the past that P.R. and P.L.V.A. might have similar causes. At first sight there would seem little reason to suggest a relationship between P.R. and the other two conditions; but there are some interesting points of resemblance. It should be remembered that there was originally much controversy over the nature of P.L.V.A., which is now recognized as an acute variety of Para. G. for there is little resemblance between the course and appearance of typical examples of the two diseases. Cases representing transition forms between the two, however, present convincing evidence of their relationship.

Parapsoriasis Guttata

The classification of parapsoriasis has undergone many revisions since 1902 when Brocq made his first proposals. Most dermatologists now take the view of Civatte,[16, 17] the greatest authority on the subject, that Para. G. must be dissociated entirely from parapsoriasis lichenoides and *parapsoriasis en plaques*. The last two diseases are related and *formes de passage* link them; and their histological appearances are similar, the important changes being mainly dermal. Both may be forerunners of mycosis fungoides. Para. G. is always a benign disease and the important features in its histological picture are changes in the epidermis.

The rash of Para. G. may quite closely resemble that of P.R. It consists of many isolated lesions, 5-10 mm., slightly papular and pink at first but soon turning to reddish brown, which after a few days are covered by an adherent scale. The infiltrate disappears and there remains only a thick grey scale that can be detached *in toto*. The scale falls off in a week or two leaving a brown macule which fades in its turn. The evolution of a lesion takes 3 weeks but, as new ones are constantly appearing in crops the mixture of lesions at different stages of development gives a quite characteristic appearance. The body and limbs are usually affected, and the hands, feet, face and scalp and mucous membranes are almost always spared. Streamlining of lesions on the back, as in P.R. may be seen. In many cases the disease lasts for years without affecting the general health in the least. Sometimes the rash is recurrent with intervening free spells lasting weeks or months, and the recurrences may be seasonal. There are also typical cases which last only

for a few months and then disappear never to recur. Of 4 cases (adults) seen by me in 1951 2 are still active 4 years later and 2 cleared up within a year. All were Europeans; I have not yet seen Para. G. in the Bantu. Two cases have been seen in 1955, a boy of 10 and a woman of 40.

Apart from the common variety there are also described micropapular, hyperkeratotic, confluent and acute papulo-vesicular varieties. Post-lesional leukoderma of a transient nature is quite frequent and rarely leukoderma is a marked feature (*parapsoriasis en gouttes leucodermique*). Scar formation is not seen in typical Para. G.

The disease commonly begins in youth (Gross[18] quotes Riecke as giving the highest age incidence between 15 and 25 years) and children are often affected. In some cases, among the typical lesions, one sees also some papulo-vesicular and necrotic elements. Such cases represent transition forms between Para. G. and P.L.V.A.

Pityriasis Lichenoides et Varioliformis Acuta

This description is reserved for those cases of parapsoriasis guttata which run an acute course and in which all the lesions are papulo-vesicular or necrotic. The name was coined by Habermann of Bonn in 1925 but credit for discovery of the condition is generally given to Mucha of Vienna (1916). Even before 1916 cases of Para. G. with purpuric and necrotic lesions had been described (cited by Lapière[19]) and the first description of an atypical Para. G. of the P.L.V.A. type (dermatitis psoriasiformis nodularis) is probably that of Moller and Afzelius of Stockholm in 1903 (Gross).

The rash of P.L.V.A. has, according to European and American descriptions, the same distribution as that of Para. G. the body and limbs being affected and the hands, feet, face, scalp and mucous membranes spared. In the South African Bantu, however lesions on the face, hands and feet are frequent (Fig. 3). The lesions appear in crops so that various stages of development can be seen at any given time, and the disease runs its course in a few weeks to a few months or a year at the most. The commonest lesions are red papules and papulo-vesicles, 2-5 mm., which often proceed to central necrosis and crusting to give an appearance suggestive of papulonecrotic tuberculides. Hundreds of lesions are usually present. Purpuric papules, papulopustules, bullae and varioliform vesicles are also sometimes present. Typical lesions of Para. G. may also be seen and healing lesions can look identical to those of P.R. In some of my cases in the Bantu the healing stage has copied P.R. so exactly that no other diagnosis would have suggested itself to one who had not seen the primary lesions and examined them histologically. Malaise, fever, joint pains and glandular enlargement may be noted in the early days of the disease; and local infections may precede an attack. Aphthae and ulcers of the buccal mucosa and ulcers or herpetiform lesions of the genital mucosa have been noted, though rarely.

Fig. 3. Pityriasis lichenoides et varioliformis acuta in a Bantu.

Varioliform scarring results in some of many of the lesions and post-lesional leukoderma is common. It can readily be understood from this description that P.L.V.A. was not at first readily accepted as a form of Para. G. Mucha considered it to be so and later descriptions of transition forms showing typical lesions of both acute and chronic types seem to make it clear that we are dealing with two manifestations of the same disease. Cases are described in which the original rash was that of typical chronic Para. G. but where later necrotic lesions appeared and the picture turned to that of P.L.V.A. and the disease ran an acute course.[19] In other cases beginning as P.L.V.A. the picture has changed to that of Para. G. and the course has been chronic.[20, 21]

Recurrences of P.L.V.A. have been described, but the recurrences have nearly always followed closely one upon the other over a space of a few months.[22] P.L.V.A. is a disease of young people with an age distribution like that of P.R. and Para. G. The youngest patient I have seen was between 1 and 2, the oldest about 45. All but one of my South African cases have occurred in the Bantu. It is interesting to note that the incidence of P.L.V.A. is sporadic. The first group (of 13 cases) was seen in Vienna; later outbreaks were reported in England, Poland, Russia, Japan, France and the USA (see table in Gross).[18] Most cases in the literature occurred in spring or autumn (Basset cited by Joulia and le Coulant).[23]

The question of etiology is well reviewed in this recent article by Joulia and le Coulant. It seems most probable that P.L.V.A. is a microbic disease but no micro-organism has yet been demonstrated. These authors refer to unsuccessful experiments by Sirota with a complement fixation reaction and inoculation of guinea pigs and my Mazzaro with culture on allantoic membrane. They note the resemblance of the lesions of P.L.V.A. to those of papulo-necrotic tuberculosis, and refer to successful treatment of the disease with remedies used in tuberculosis. Gougerot's view that benign parapsoriasis is a nodular cutaneous reaction of defence in persons sensitized against a micro-organism seems reasonable. The recurring crops of lesions would correspond with embolism of the causative organisms from a chronic focus to be destroyed in the skin. An intense necrotic reaction (miniature Koch's phenomenon) would be of good prognostic significance. Varying degrees of sensitization in different individuals would explain variations in the clinical picture of parapsoriasis and it could be classified, like Gougerot's trisymptomatic

disease, with the microbic allergides. Gougerot's theory leaves open the question as to whether such a reaction is caused by a specific micro-organism or whether it might be non-specific and result from one of a variety of infective agents. The fact that anti-tuberculous remedies have sometimes been followed by cure is surely no reason to postulate a tuberculous origin for a spontaneously healing disease.

My attention was first drawn to the resemblance between P.R. and P.L.V.A. during the period 1950-52 when, with an 'epidemic' of P.R. in Europeans and Bantu I saw a number of cases of P.L.V.A. in the Bantu. As I have already noted P.L.V.A. in the healing stage in the Bantu can bear a very close clinical resemblance to P.R. In the literature we find many references to cases in which differential diagnosis of the two conditions presented the greatest difficulty. I found that I was not the first to note a close resemblance between the two conditions. Drake,[24] in 1930, discussing cases of P.L.V.A. shown at the Royal Society of Medicine, London, remarked that most of them exhibited the peculiar streamline distribution along the lines of the ribs and in the axillary folds, and the greasy mica-like scale was also like that seen in P.R. Hence it was suggested that they had a similar cause. Macleod[25] also noted that the distribution of lesions resembled that of P.R. and suggested the possibility that it might be due to a specific infective virus.

I was distracted from pursuing the subject at the time by Dr. A. Civatte whom I consulted over the histological relationships. He found difficulties in admitting a relationship as the epidermal lesions in P.R. (eczematide) are not comparable to those seen in P.L.V.A. or Para. G. However, I returned to the inquiry recently when, with a rising incidence of P.R. I saw a case of P.L.V.A. and a case of Para. G. within one week. (I have since seen 3 more cases of P.L.V.A. and a second Para. G.) On checking my records I discovered a fact that had not previously struck me; the 4 cases of Para. G. which I had previously seen in South Africa all first consulted me during the period 1950-52. It is interesting to note that Drake,[26] in 1932, referred to the sporadic incidence of cases of Para. G.

There are many points of similarity in course and clinical appearances between P.R. and P.L.V.A.; the similarity is less marked in comparing P.R. and Para. G. I have summarized these points in Table II.

Histology

The important changes in Para. G.[17] are those in the epidermis. There is a dermal infiltrate, almost exclusively lymphocytes, which may be a prominent feature in the early stages. This infiltrate is diffusely distributed in the papillary layer and even a little deeper. Whatever the quantity of infiltrate in the dermis it is always abundant in the epidermis which it invades from the start in all its thickness. It penetrates immediately to the superficial layers seemingly without any resistance. This exocytosis is not preceeded by exoserosis as is the case in eczema where spongiosis makes a bed for the infiltrate. The attack on the epidermis is at first in multiple tiny foci. Wedges of large monocytes make the initial penetration and are quickly followed by lymphocytes (Fig. 4). The Malpighian cells lose their connecting filaments, swell and separate to give place to the invading cells. The epidermis soon repairs itself and looks again relatively intact but powdered with lymphocytes. It remains, however, for a time incapable of forming keratohyaline and dries off in parakeratosis to form a scale at the expense of its upper layers.

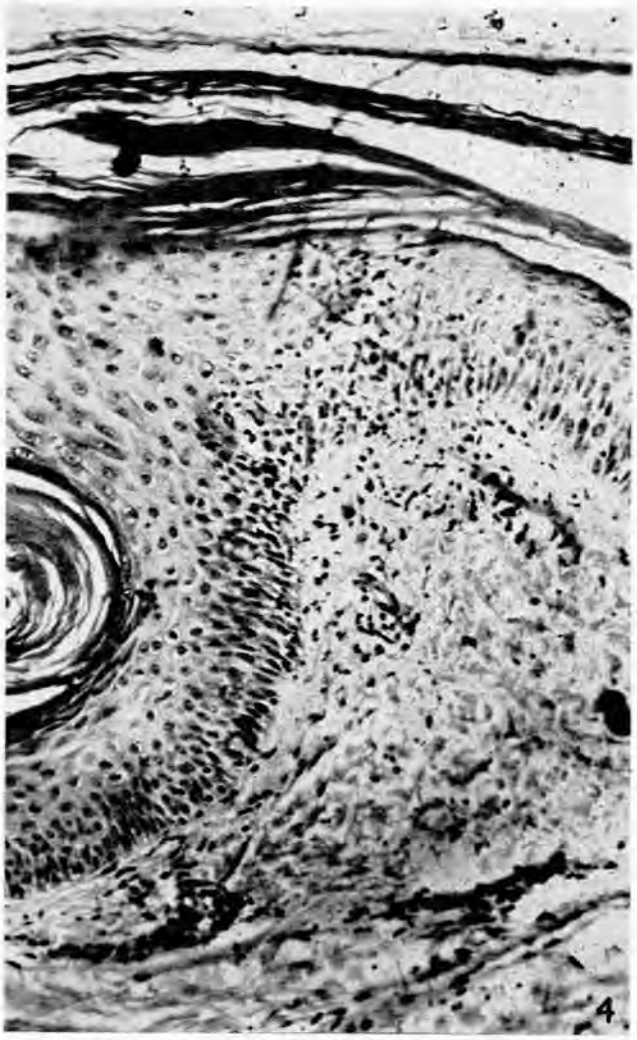

Fig. 4. Parapsoriasis guttata. A wedge-shaped focus of monocytes and lymphocytes penetrates the epidermis; parakeratosis and dermal infiltrate.

Sometimes the epidermal alterations are still more marked. As well as the dislocation from exocytosis there may also be degeneration. The corpus mucosum is sown with eosinophilic hyaline bodies, the remains of Malpighian or invading cells. This touches on the picture seen in P.L.V.A.

In P.L.V.A.[17, 27] the epidermal alterations are even more marked. There is an abundant exoserosis and even vesicular cavities between the Malpighian cells. These cells enlarge, their cytoplasm becomes transparent, their nuclei alter and their outlines are ill-defined. The whole corpus mucosum may become homogenized and undergo hyaline degeneration so that the central areas are

sporadic appearance of cases of P.L.V.A. is striking and it has been noted by Drake that cases of Para. G. also seem to come in crops. In my own experience cases of P.L.V.A. and Para. G. have been seen on two occasions when P.R. was epidemic. This suggested a relationship between P.R. and the two other clinically dissimilar but actually identical diseases. I have asked colleagues in Britain and Europe to refer to their records to see whether any such relationship in incidence could be traced, but in the (short) periods they could survey, nothing significant was discovered. It is interesting to note that 8 cases of P.L.V.A. were seen in London during the period 1926-29 (Gross); and Percival writes: 'During the period 1924-1930 the case incidence of P.R. increased considerably, reaching its highest point in 1930 and the increase appears to be maintained during January-June of 1931. It would seem, therefore, that since 1925 Edinburgh has had one "epidemic" of P.R. and is at present experiencing a second'.

Further study shows that there are many points of resemblance in the clinical appearances, age incidence, seasonal incidence, course and immunological behaviour, between P.R. and P.L.V.A. and to a less marked degree between P.R. and Para. G. It seems not entirely unreasonable to suggest that these conditions may have a common, probably viral, cause or be due to closely related infective agents. There are naturally objections to the theory. Why is P.R. common and always with us and the others rare and, in the case of P.L.V.A. certainly, sporadic in incidence? How can we postulate a common cause for two acute conditions and one chronic condition? The reason could lie in variation in virulence of the causative organism or in variation in immunological response to infection in the host. It is possible that the consistent failure of attempts at culture and passage is due to the fact that the lesions are allergides and that the microbic allergen is, therefore, being destroyed in the skin.

There are definite differences between the histological changes seen in the 3 conditions and it is generally easy to differentiate P.R. from the others. This, too, might be explicable by variations in reaction to a single cause. There is precedent for the suggestion and one can offer the examples of syphilis, tuberculosis and leprosy where the histological pictures produced at different stages can differ vastly.

SUMMARY

The clinical picture of pityriasis rosea in its typical and atypical forms is fully reviewed. Some variations in the behaviour of the disease in the Transvaal, particularly in the Bantu, are described. Pityriasis rosea seems to be commoner in the Transvaal than it is in Europe and America. Theories of origin are discussed and experiments in culture and passage are described.

It is noted that sporadic outbreaks of pityriasis lichenoides et varioliformis acuta and parapsoriasis guttata have coincided with epidemics of pityriasis rosea in the Transvaal. The clinical appearance, course, epidemiology etc., of the three diseases are discussed and compared and it is suggested that they may have a common cause or closely related causal agents.

TABLE I. THE VARIETIES OF PITYRIASIS ROSEA

Macular

Punctate, Guttate, Nummular, Circinate	Bilateral	Solitary plaque
	Unilateral	Cervico-cephalic
	Localized	Scalp
	Generalized	Bathing-trunk distribution
	Confluent	P.R. *gigantea*
	Diffuse	*P. circiné et marginé.*

Urticarial

P.R. ortié (Hallopeau), P.R. urticata (Vorner)

Papular

Maculo-papular
Follicular
Miliary
Lichen-planus-like

Vesicular

Maculo-vesicular
Papulo-vesicular
Varicelliform

Pustular

Pityriasis Rosea et Pustulo-crustosa

Other Atyrical or Complicated Types

Psoriasisform
Eczematous
Lichenified
Erythematous
Non-squamous
Haemorrhagic
Chronic (P.R. perstans)
With post-lesional hyperpigmentation or depigmentation

TABLE II

	Pityriasis Rosea	*Pityriasis Lichenoides et Varioliformis Acuta*	*Parapsoriasis Guttata*
Herald Patch	A feature of the disease, occurring in 12-90% of cases according to various authors.	Solitary lesion preceding main eruption has been described.	Not noted.
Rash	Commonly maculo— or papulo-squamous lesions, but many variants described. Lesions appear in crops.	Papules or papulo-vesicles going on to necrosis. P.R.— like lesions also common. Lesions appear in crops.	Papulo-squamous lesions. Lesions appear in crops.
Distribution	Body and limbs. Face, scalp, hands, feet and mucous membranes usually spared in adult Europeans. Face and scalp often affected in Bantu at all ages. Streamline distribution over ribs on back.	As for P.R.	Body and limbs. Face scalp extremities and mucous membranes usually spared. Streamline distribution on back.
General Symptoms	Malaise, headache, low fever, adenopathy, fleeting pains, may occur in early stages	As for P.R.	Nil.
Incidence	Relatively common.	Rare.	Rare.
Duration	Average case 6–12 weeks. Limit about 1 year.	6 weeks to 6 months. Rarely as long as 1 year.	Often very chronic, persisting with or without remissions for many years. Cases with typical lesions but running short non-recurrent course are described.

TABLE II (CONTD.)

	Pityriasis Rosea	*Pityriasis Lichenoides et Varioliformis Acuta*	*Parapsoriasis Guttata*
Recurrences	An attack usually confers lifelong immunity. Very few cases of second attacks described; intervals between attacks in such cases usually several years at least.	Recurrences or second attacks after long intervals very rare. Recurrences over short period described; probably all part of a single attack.	Regularly recurrent attacks a feature of some cases. Recurrence after long period of freedom is rare.
Age at Onset	Young people. Highest incidence between 20 and 30	Young people 15–25.	Young people 15–25
Seasonal Distribution	Commonest in the colder months.	As for P.R.	Not applicable.
Epidemics	'Epidemics', apart from seasonal fluctuations, noted by many observers. Increased number of cases seen in Transvaal 1950–52 and again in 1955.	Cases have occurred in sporadic outbreaks in various parts of the world since the first cases were described in Vienna. 8 cases seen in Transvaal 1950–52, 4 in 1955.	Sporadic occurence of cases mentioned by Drake. 4 cases seen in Transvaal 1950–52, 2 in 1955.

I have gratefully to acknowledge the help of the following collaborators; Dr. J. M. Martin, Institute for Pathology, University of Pretoria; Dr. James Gear, Dr. H. H. Malherbe and Miss R. Harwin, Poliomyelitis Research Foundation, Johannesburg; Dr. I. J. Venter, Pretoria General Hospital and Mr. T. Marais, Photographic Department, Pretoria University Medical School.

REFERENCES

1. Percival, G. H. (1932): Brit. J. Derm., **44,** 241.
2. Gougerot, H. (1936): in *Nouvelle Pratique Dermatologique*, Vol. 4. Paris: Masson.
3. Benedek, T. (1936): Acta derm.-venereol., **18,** 151.
4. Klauder, J. V. (1924): J. Amer. Med. Assoc., **82,** 178.
5. Degos, R. (1953): *Dermatologie*. Paris: Flammarion.
6. Haxthausen, H. (1927): Brit. J. Derm., **39,** 141.
7. Wile, U. J. (1927): Arch. Derm. Syph., **16,** 185.
8. Niles, H. D. and Klumpp, M. M. (1940): *Ibid.*, **41,** 265.
9. Darier, J. (1928): *Précis de Dermatologie*, 4th ed. Paris: Masson.
10. Gourvitch, E. I. (1936): Ann. Derm. Syph., Sér. 7, **7,** 488.
11. Hollström, E. (1948): Acta derm.-venereol., **28,** 325.
12. Schirren, C. G. (1954): Hautartzt, **5,** 457.
13. Hissard, R. (1927): Bull. Soc. franç. Derm. Syph., **34,** 105.
14. Thomson, M. S. and Cumings, J. N. (1931): Brit. J. Derm., **43,** 617.
15. Joyeux, Burnier and Duché (1930): Bull. Soc. franç. Derm. Syph., **37.** 1128.
16. Civatte, A. (1948): Arch. belges Derm. Syph., **4,** 75.
17. *Idem* (1951): Ann. Derm. Syph., **23,** 33.
18. Gross, P. (1931): Arch. Derm. Syph., **23,** 33.
19. Lapière, S. (1948): Arch. belges Derm. Syph., **4,** 40.
20. Joulia, P., le Coulant, P., Sourreil and Texier, L. (1953): Bull. Soc. franç. Derm. Syph., **60,** 202.
21. Gougerot, H. and Mathieu, R. (1937): *Ibid.*, **44,** 48.
22. Touraine, A., Golé, L. and Carteaud, A. (1947): Ann. et Bull. Soc. franç. Derm. Syph., Ser. 8, **7,** 451.
23. Joulia, P. and le Coulant, P. (1954): Minerva derm., **29,** 172.
24. Drake, J. A. (1930): Brit. J. Derm., **42,** 72.
25. Macleod, J. M. H. (1933): *Diseases of the Skin*, 2nd issue. London: H. K. Lewis.
26. Drake, J. A. (1932): Brit. J. Derm., **44,** 85.
27. Basset, H. *in* de Graciansky, P. and Boulle, S. (1952): *Atlas de Dermatologie*. Paris: Maloine.
28. Touraine, A., Solente, G., Lortat-Jacob, E. and le Sourd (1942): Bull Soc. franç. Derm. Syph., **52,** 59.
29. Duperrat, B. (1950): *Ibid.*, **57,** 32.

DISTRIBUTION OF INTERNS *

P. N. SWANEPOEL, M.B., B.CH. (RAND)

Boksburg

In the post-war years the world entered the era of shortages. Wherever one turns one finds that the demand far exceeds the supply. This applies not only to raw materials but also to man-power, the shortage of which is evident everywhere. The employee can therefore dictate the conditions of employment to the employer. The demand for interns also exceeds the supply, and the intern today is in a position to dictate the conditions under which he will work. At some hospitals this dictator-like attitude has only been overcome with great difficulty.

The distribution of interns is so closely inter-related with the shortage of interns, that one has included the causes of the shortage of interns in this paper.

The shortage of interns may not be felt in teaching hospitals and other large general hospitals, but it is evident in the smaller hospitals and the platteland hospitals. It is of vital importance to the smooth administration of a hospital, for the intern forms the backbone of the full-time staff, and a shortage of interns directly affects the medical care of the patient. This shortage has become so acute in some hospitals that drastic steps will have to be taken to relieve it, and will have to be taken immediately.

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

CAUSES OF THE SHORTAGE IN INTERNS

It is only since compulsory internship was introduced that the shortage of interns has arisen. In the days of voluntary internship the hospitals were well staffed although housemen were paid much lower wages. At that time there were not so many hospitals as today, but on the other hand the medical schools have increased since those days and are producing more medical graduates. In my opinion the present shortage of interns is not a true shortage, but only an apparent shortage, which was created in the post-war years. To estimate the true shortage of interns one must first remove the causes of this pseudo-shortage, which are as follows:

(*a*) Directly after the cessation of World War II a number of ex-volunteers applied to the Cape Town and Witwatersrand medical schools for admission as medical students. These medical schools already had their