Group 8: Chronic Papular and Fissuring Eczema.—These were 7 chronic cases of endogenous eczema. One showed some improvement, the remainder no change.

Group 9: Impetigo.—Five cases cleared satisfactorily with vioform cream.

Group 10: Infantile Eczema.—The results of the treatment of this condition were unsatisfactory. Eight cases improved equally on the cream and on the cream base, 5 showed no change, and 3 displayed some irritation on the vioform cream.

Group 11: Nummular Eczema.—Four showed marked improvement; two appeared to be irritated by the vioform and one by the cream base.

Group 12: Neurodermatitis.—One showed improvement, three showed equal improvement on both preparations, whilst in one patient the base proved the more effective.

Group 13: Lichen Hypertrophicus.—In three patients no change was noted.

Group 14: Psoriasis.—In two patients with classical psoriasis no improvement could be attributed to the vioform. In psoriasis involving the flexures (seborrhoeic) two appeared to be improved, and four showed no change which could be attributed to the vioform. Two patients with pustular psoriasis found the active preparation and the vehicle equally effective.

Group 15: Dermatitis Herpetiformis.—Lesions in two patients with this disorder responded equally to the vioform cream and base, one patient was aggravated by both, and one appeared to be aggravated by the vioform cream. In view of the sensitivity of these patients to potassium iodide locally, this measure was tried at first very carefully, but the action expected from an iodide preparation was not noted.

Comments

Unfortunately, vioform is yellow and it stains. When dispensed in a water-miscible base the staining tendency is less marked. It darkens on exposure to air; this does not seem to interfere with its therapeutic efficiency. A few patients stated that on application they experienced a mild burning sensation, which quickly disappeared; when there was intolerance this symptom was more pronounced.

Vioform proved antipruritic in eczematous conditions and soothing in the treatment of contact dermatitis; it showed a marked antiseptic action in pyoderma and secondarily infected dermatoses. In the media used it was found to be well tolerated.

Conclusions

The effect of 3% vioform in a water-miscible base or in petroleum jelly has been assessed under carefully controlled conditions on a variety of dermatoses in 176 patients. It proved a useful local application in the treatment of the following conditions: coccogenic sycosis barbae, seborrhoeic dermatitis, otitis externa, acute vesiculo-papular eczema, gravitational eczema, impetigo, and, occasionally, in nummular eczema. The incidence of cases of intolerance was low (4.5%). This investigation suggests that vioform merits more extensive use in these conditions.

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DISSEMINATED SCLEROSIS IN SOUTH AFRICA

ITS RELATIONSHIP TO SWAYBACK DISEASE AND SUGGESTED TREATMENT

BY

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It has long been believed, and rightly, that disseminated sclerosis very rarely occurs among the white population of South Africa and that it is most rare among those white people who have been born and bred there. Indeed, some physicians deny that it occurs at all in such people. These opinions, however, were based on individual clinical experience; there were no reliable and exhaustive figures. Therefore the available evidence relating to the incidence, geographical distribution, etc., of the disease among white South Africans during the last ten years has been collected and assessed. It may be said here that disseminated sclerosis is unknown in the native and other coloured peoples of the country.

Incidence and Geographical Distribution

In order to discover the incidence, etc., of the condition it was decided to search the medical records of all the main hospitals in the country for the last ten years. Records of a diagnosis of disseminated sclerosis could be found only at Groote Schuur Hospital, Capetown, at Johannesburg General Hospital, and at the Pretoria Hospital—in fact, at the three university teaching hospitals. In addition, three isolated cases—two at Port Elizabeth and one at Durban—have been reported. The cases are here summarized. In all cases, unless otherwise stated, the Wassermann reaction was negative and the blood count normal.

Groote Schuur Hospital, Capetown.—Between 1938 and 1948 eight patients were diagnosed as suffering from disseminated sclerosis. These cases are summarized in Table I. Two of these patients were white women born in South Africa (one lived at Capetown and one at Vryburg); one was a South African who had spent six months abroad; and two others were South Africans of German origin, though in the case of one of these the diagnosis was doubtful. Of the remaining three cases, in one the diagnosis was probably wrong and in the other two the records were incomplete.

Johannesburg General Hospital.—In the files at Johannesburg General Hospital 22 cases were classified as suffering from disseminated sclerosis between the years 1938 and 1948. The cases are summarized in Table II. Five of these patients were indigenous white South Africans, 14 were immigrants, and in the three others the diagnosis was in my opinion definitely

mistaken. These last three cases will not be considered further. Of the five indigenous patients, the diagnosis is considered to be correct in three and doubtful in two.

TABLE I.—Cases in Capetown

Case	Sex	Age	Race	Diagnosis
C.1	F.	46	S.A.	Symptoms of weakness in legs for 20 years, periods of remission; nystagmus, intention tremor; posterior column and pyramidal tract involvement. Seen by me. (DS)
C.2	F.	40	S.A.	Symptoms of paraesthesia on trunk, a visual episode; dragging of legs, with remissions, for seven years. Sensory loss, pyramidal tract and posterior column involvement. (DS)
C.3	F.	35	S.A.(a)	In England for six months when aged 8. A five-years history, with remissions, of diplopia, tingling in hands, and weakness of both legs. Nystagmus, temporal pallor, and pyramidal tract involvement. (DS)
C.4	F.	54	IMM.	Of German origin. Temporary blindness 20 years ago; tremor, clumsiness, weakness right leg, loss of sensation in foot, remissions. Pallor of temporal half of optic disks; cerebellar, sensory, and pyramidal tract involvement. (DS)
C.5	F.	30	IMM.	Of German origin. Giddiness and anaesthesia of right side of face. On examination no lesion of central nervous system. (Doubtful)
C.6	F.	47	S.A.	Twelve years ago had a temporary total paralysis and severe pain affecting both legs; a year later the right arm was similarly affected; a year ago blindness of right eye with pain. No physical signs. (Not DS)
C.7				Case records incomplete
C.8				Case records incomplete

S.A. = South African white who has never been abroad. S.A.(a) = South African who has been abroad. IMM. = Immigrant. DS = Disseminated sclerosis diagnosed.

Pretoria Hospital, Pretoria.—The records reveal one definite case of disseminated sclerosis in an immigrant who entered the country when he was 2 years old, and two doubtful cases (Table III).

Port Elizabeth.—Two cases were found here. The first was that of a woman, aged 60, who was born in South Africa but had spent the greater part of her life up to the age of 39 in England. First one eye then the other became affected by temporary loss of vision more than 20 years ago while in England. Since then she had had attacks of weakness in the

TABLE II.—Cases in Johannesburg

Case	Sex	Age	Race	Diagnosis
J.1	M.	39	S.A.	Weakness in right leg; nystagmus, optic pallor, intention tremor, posterior column and pyramidal tract involvement. (DS)
J.2	M.	40	S.A.	Unsteadiness of gait, hyperaesthesia of right thigh and weakness of right leg; nystagmus and pyramidal tract involvement. (DS)
J.3	F.	43	S.A.	Blurring of vision, paraesthesia, and weakness of both legs; remissions; urinary incontinence. Temporal pallor, intention tremor, sensory loss and pyramidal tract involvement. (DS)
J.4	F.	37	S.A.	A two-years history of difficulty with micturition and dragging of feet. No physical signs. (Doubtful)
J.5	M.	27	S.A.	Cramp in legs. (Doubtful)
J.6	M.	50	IMM.	Temporary blurring of vision six years ago; now drags the right leg. Nystagmus, temporal pallor and pyramidal tract involvement. (DS)
J.7	M.	30	IMM.	Loss of sensation in finger-tips of right hand; drags the right leg; remissions. Sensory loss in fingers and pyramidal tract involvement. (DS)
J.8	F.	37	IMM.	Weakness of both legs and precipitancy of micturition; remissions. Pyramidal tract involvement. (DS)
J.9	F.	45	IMM.	Twenty years ago had temporary blurring of vision in one eye. Weakness in right arm and right leg. Pyramidal tract involvement. (DS)
J.10	F.	47	IMM.	Blurring of vision a few years ago; weakness in right arm and right leg. Nystagmus and pyramidal tract involvement. (DS)
J.11	M.		IMM.	Insufficient details available
J.12	F.	31	IMM.	Insufficient details available
J.13	M.	38	IMM.	History suggestive of disseminated sclerosis, but details of examination are insufficient
J.14	F.	43	IMM.	Weakness right leg and difficulty in starting micturition. Insufficient details available
J.15	M.	35	IMM.	Not disseminated sclerosis
J.16	F.	40	IMM.	History suggestive of disseminated sclerosis, but details of her physical examination are insufficient for diagnosis
J.17	M.	48	IMM.	Insufficient evidence
J.18	M.	65	IMM.	Weakness in both legs and difficulty with micturition. Pyramidal tract and posterior column involvement. W.R. positive. A syphilitic cause for the symptoms has not been excluded
J.19	M.	31	IMM.	Weakness in left leg. Left Babinski response extensor; W.R. positive. Again a syphilitic cause has not been excluded

TABLE III.—Cases in Pretoria

Case	Sex	Age	Race	Diagnosis
P.1	M.	44	IMM.	Immigrated from Australia when 2 years old. In the last nine years attacks of transient blindness and hemiparaesthesia. Nystagmus, intention tremor, ataxia, and pyramidal tract involvement. (DS)
P.2	M.	53	S.A.	Fifteen years ago pyrexia and polyarthritis. Agglutinations for <i>Brucella abortus</i> positive. He developed ataxia with nystagmus and Rombergism, is occasionally incontinent of urine, and has transient paraesthesiae. There is pyramidal tract, posterior column, and cerebellar involvement. This seems to be a case of focal demyelination following brucellosis
P.3	M.	59	S.A.	Account translated from <i>Geneeskundige Tydskryf</i> . Thirty-five years ago he had weakness and loss of sensation in both legs, with retention of urine; lumbar laminectomy was performed; a short time later he had double vision for a few days. Six years ago he developed a sudden hemiplegia, since when he has been bedridden. On examination there was right-sided hemiplegia with bilateral pyramidal tract involvement; the posterior columns were normal. In my opinion there is not enough evidence to diagnose disseminated sclerosis. The hemiplegia was probably due to a cerebral thrombosis

The second patient was the brother of the first case. He was also seen by me. He developed a slowly increasing weakness, starting at the extremities, over a period of three years; this increased until he died following paralysis of his intercostal muscles. There was a bilateral extensor response, with absent reflexes and slurring of speech. In view of the fact that his sister had disseminated sclerosis this may have been a "forme fruste" of the disease.

Durban.—Dr. J. Drummond, medical specialist, has informed me that he treated a case of disseminated sclerosis in a South-African-born girl who had not been abroad. Unfortunately the records have been lost, and she has long been dead. As the records are not available, this case will have to be included in the "doubtful" group.

Comparison with England and Wales

My opinion, based on the above analysis, is that disseminated sclerosis does occur among South Africans who have never been abroad, but the incidence is extremely low. There have been two cases at Groote Schuur Hospital, Capetown, both women—one who had spent all her life in Capetown, and one from Vryburg—and three cases at Johannesburg General Hospital—one from Lichtenburg, one from Rouxville, Transvaal, and one of unknown address: five cases in all, scattered throughout the Union, and out of a white population of 2,400,000. This number is too small to enable one to draw any conclusions concerning the geographical distribution of the disease. There may of course have been other cases, but an obscure long-lasting disease would usually be discussed by the local doctor and eventually sent to a specialist in one of the big centres; and because of its rarity the specialist would normally send the case to hospital for investigation.

In South Africans who have been abroad two cases were diagnosed at Capetown, five at Johannesburg, one at Pretoria, and one at Port Elizabeth—making nine cases in all (Table IV). Of the doubtful cases in South Africans who have never been abroad there are two at Johannesburg, two at Pretoria, and one at Durban—five in all. There are ten doubtful cases among South Africans who have been abroad—one at Capetown and nine at Johannesburg. Probably the majority of these patients do suffer from disseminated sclerosis. A striking factor about all cases is the mildness of the disease; only one "doubtful" case is bedridden.

It has already been noted that the disease is unknown among the native and coloured peoples of South Africa. Two cases have been reported in East African natives.

TABLE IV.—*Classification of Cases Diagnosed as Suffering from Disseminated Sclerosis in South Africa*

	Capetown	Johannesburg	Pretoria	Elsewhere	Total
Disseminated sclerosis in South Africans who have never been abroad	2	3	0	0	5
Disseminated sclerosis in South Africans who have been abroad, or immigrated	2	5	1	1	9
Doubtful cases in South Africans who have never been abroad	0	2	2	1	5
Doubtful cases in South Africans who have been abroad, or immigrated	1	9	0	0	10

Brain (1940) gives statistics on the incidence of disseminated sclerosis. His figures are for the year 1930. He estimates the incidence as 160 per million for England and Wales. His method of computation, based on that of Bramwell, assumes an average expectation of life of eight years after diagnosis of the condition—in my opinion a very modest estimate. Therefore the annual number of deaths from disseminated sclerosis multiplied by eight million divided by the population is the incidence per million. Later figures give approximately the same incidence. Deaths from disseminated sclerosis in 1935 were 781; in 1938, 764; and in 1945, 760. The population of England and Wales in 1942 was 42,600,000. On the continent of Europe the disease is widespread in Germany, France, Scandinavia, Austria, and especially in Switzerland.

The figure of 160 per million in England and Wales may be compared with the South African figure of five cases out of an indigenous white population of 2,400,000.

Swayback Disease of Lambs

Campbell *et al.* (1947) reported the occurrence of four cases of disseminated sclerosis among seven workers engaged on research into swayback disease of lambs. This is a demyelinating disease, known to be associated with a deficiency of copper in the soil on which the affected lambs are bred, or with a defective absorption of copper.

In order to obtain some information at first hand about swayback disease I visited Onderstepoort Veterinary Research Station and consulted Dr. de Kock, the Director, and Dr. Schultz, one of the research workers concerned with this disease.

Swayback disease is, as far as present knowledge goes, confined in South Africa to three small areas—a small coastal strip around Mossel Bay from Knysna to Bredasdorp, along Saldanha Bay up to Vredenberg, and around Stellenbosch. It will be noticed that the five proved cases of disseminated sclerosis in native-born white South Africans are not confined to these "swayback" areas.

South African farmers call swayback disease "Lamkruis," which means paralysis of the lumbar region; also "Swaai-gat," or swaying back; and "Litsiekte," or joint disease. Adult sheep are seldom affected by it, but newborn lambs commonly are. The incidence is never high, although it does vary from year to year. Affected lambs are unsteady in their gait and spastic in their movements. Post-mortem examination often discloses cavities in the cerebrum filled with a yellowish fluid. The cerebellum is always involved, though there is never cavitation there. Histological examination of the central nervous system reveals a widespread round-cell infiltration and demyelination. Much more detailed work is still required on the pathology and histology of the disease in South Africa.

Relationship between Swayback Disease and Copper Deficiency.—For many years the farmers in the "swayback" areas have given pregnant ewes copper sulphate solution by mouth in order to prevent the occurrence of the disease in their lambs. Further, it has been shown at Onderstepoort that there is a relationship between the copper content of the grazing and the presence of the disease. In the Karroo areas, where swayback disease is unknown, the ruminal copper content (which is an index of the copper content of the soil) is 13 parts per million—a high proportion; but in the coastal areas, where the disease does occur, it is only 5 parts per million. This relationship has also been noted in England; and there, too, copper sulphate is given in certain areas to pregnant ewes as a means of prophylaxis against the disease. Less commonly, but successfully, it has been used in the treatment of established cases. A point of interest is that in the Karroo a form of jaundice occurs in sheep, known as enzootic icterus, with gross haemolysis and haematuria. Post mortem, the copper content of the liver is found to be over 200 times the normal. Enzootic icterus seems to be a form of copper poisoning, although why the copper, which is normally fixed in the bones, should be suddenly mobilized is not known. Experiments are now being carried out to find if copper can be mobilized in the same way as lead—by giving acid salts. This research is of particular interest because of the possible toxic results of copper therapy in man.

Relationship between Disseminated Sclerosis and Swayback Disease.—No explanation can yet be given for the occurrence, as reported by Campbell *et al.* (1947), of four cases of disseminated sclerosis among seven workers engaged on research into swayback disease. A low copper content of their food could hardly be more than a predisposing factor, as in a highly developed country such as England the copper content of the diet cannot be supposed to vary much from one region to another, while the incidence of the disease among the general public is certainly much less than it was among these research workers.

Margulis, Soloviev, and Shubladze (1946) claim to have isolated a virus responsible for disseminated sclerosis; and perhaps, therefore, an infective factor is common to both diseases, a relative copper deficiency in the host being the predisposing factor. A possible explanation of the observation of Campbell *et al.* would then be that a copper content of the body sufficient to protect against chance infections was not sufficient to do so when the infection was massive. This of course is purely hypothetical; but it is not at variance with the known facts and does provide a hopeful basis for future work on the problem.

Suggested Form of Treatment for Disseminated Sclerosis

Whatever the precise relationship between the two diseases may be, the observation of Campbell *et al.*, supported by the facts recorded in this article, does strongly suggest that there is some relationship, and that it resides most probably in there being a common causative factor (or factors). It is therefore reasonable to suppose that a form of treatment effective against one disease will be effective against the other. Also, there is at present no form of treatment for disseminated sclerosis that is of proved value.

Thus, while mass prophylaxis against disseminated sclerosis by ensuring an adequate copper content of the diet is neither indicated nor practicable at the moment, there is in my opinion much to be said for treating established cases with copper sulphate. A daily dose of $\frac{1}{4}$ gr. (16 mg.) is suggested. Even bigger doses than this have been found to be safe when given therapeutically over a period of many months to the four research workers who

contracted the disease. However, in view of the occurrence of enzootic icterus in sheep from copper poisoning, the possibility of overdosage with copper in man must be borne in mind.

Summary

Campbell *et al.* have recorded a striking incidence of disseminated sclerosis among workers engaged on research into swayback disease.

In South Africa disseminated sclerosis is very uncommon. So also is swayback disease; and except in those small areas where it occurs the copper content of the soil is high. Furthermore, treatment with copper is well known to be an effective means of preventing the disease and also of treating it.

It is therefore suggested that copper therapy of established cases be given a trial.

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THE USE OF PROCAINE PENICILLIN IN CHILDREN

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A penicillin preparation that will give a bacteriostatic blood level between infrequent injections has obvious advantages. There have been many attempts to increase the time interval between injections by suspending soluble penicillin in various media such as beeswax and peanut oil: these preparations have not been satisfactory.

Salivar *et al.* (1948) described the formation of an insoluble compound by the chemical combination of procaine and penicillin in equal molecular proportions. This substance dissociates gradually to liberate soluble penicillin, and when suspended in oil gives prolonged blood-serum penicillin levels in animals (Hobby *et al.*, 1948).

Several reports have been published on the use of an oil suspension of procaine penicillin in man. Since a single daily injection is aimed at, an analysis of the recorded 24-hour blood-serum penicillin levels has been made and is given in Table I. The only paper that is concerned with children is that of Carson *et al.* (1949). It will be seen that, in the larger adult series, 34 of 62 patients (Hewitt *et al.*, 1948), 33 of 78 patients (Thomas *et al.*, 1948), and 21 of 29 patients (Jones and Shooter, 1948) gave penicillin levels of over 0.03 unit per ml. 24 hours after the injection of procaine penicillin.

This communication reports an investigation at the Children's Hospital, Sheffield, of the serum penicillin levels

TABLE I.—Summary of Published Work

Reference	Dose in Units	Type of Patient	No. of Patients	No. of Patients with 24-hr. Serum Levels u./ml.			Ratio of Cases Giving 24-hr. Levels of Over 0.03 u./ml.
				<0.03	0.03-0.2	>0.2	
Herrell <i>et al.</i> (1947)	300,000	Mixed	10	1	6	2	8 of 9
Boger <i>et al.</i> (1948)	300,000	Ambulatory	9	8	1	0	1 of 9
	300,000	In-patients	10	4	6	0	6 of 10
	150,000	Normal children	10				9 of 10
Carson <i>et al.</i> (1949)	-11 Kg.; 300,000 over 11 Kg.	Bed cases	41				"The great majority" (240 of 41)
Hewitt <i>et al.</i> (1948)	300,000	In-patients	62	28	30	4	34 of 62
Hobby <i>et al.</i> (1948)	300,000	Ambulatory	3	0	3	0	3 of 3
Jones and Shooter (1948)	300,000	Mixed	29	8	16	5	21 of 29
Sullivan <i>et al.</i> (1948)	300,000	In-patients	17	1	14	2	16 of 17
	600,000	"	2	0	2	0	2 of 2
	900,000	"	1	0	0	1	1 of 1
Thomas <i>et al.</i> (1948)	1,200,000	"	1	0	0	1	1 of 1
	300,000	"	78	—	—	—	33 of 78

obtained in 50 children following the administration of procaine penicillin in arachis oil. Twenty-five were referred from the casualty department, and the remainder were cases in bed.

Method

The preparation of procaine penicillin in arachis oil is viscid, and accurate measurement is difficult. Accordingly empirical dosage on an age basis was adopted. The dosage was: 75,000 units (0.25 ml.) to children under the age of 18 months, 150,000 units (0.5 ml.) to children between 18 months and 5 years, and 300,000 units to older children. Eight of the youngest age group received 150,000 units. The injections were made into the vastus lateralis. The penicillin serum levels were estimated by the capillary-tube method described by Fleming (1943), using whole blood inoculated with Richard's strain of *Streptococcus pyogenes*. The blood used for assay was collected in Wright's capsules and was taken at varying times. In eight children the levels in the first few hours were followed to assess the rapidity of absorption. In all cases the serum levels were estimated at the end of 24 hours.

Results

In the eight cases which were observed during the first few hours after injection of procaine penicillin the penicillin serum levels rose rapidly, to reach a peak between two and six hours. Of a further eight cases in which the serum level was estimated 12 hours after injection, all had a concentration greater than 0.25 u./ml.

An analysis of the 24-hour penicillin serum levels is presented in Table II. In 29 of the 50 patients the 24-hour

TABLE II.—Analysis of the 24-hour Blood Penicillin Level on 50 Children Following Intramuscular Injection of Procaine Penicillin

Age	Dose Units of Penicillin	No. of Patients	Patients with 24-hr. Levels over 0.03 u./ml.
0-17 months	75,000	8	6
18 months-4 years	150,000	8	7
5 years-15 years	150,000	16	9
	300,000	18	7
In-patients: 18 months-15 years ..		19	11
Out-patients: 18 months-15 years		15	5

levels were above 0.03 u./ml. Of the 16 children under 18 months, six of the eight given 75,000 units and seven of the eight given 150,000 units had a 24-hour level over 0.03 u./ml. Of the children over the age of 18 months, 16 of 34 had a level above 0.03 u./ml. In this group 11 of 19 in-patients showed levels over 0.03 u./ml. compared with five of 15 out-patients.