total blood volume, and by the picture in the bone marrow. The normal arterial oxygen saturation is a significant point against any secondary cause of polycythaemia.

The most interesting facets of the case are the following: Firstly, the patient presented with portal venous thrombosis and the consequences of portal hypertension. Secondly, she had portal cirrhosis of the liver. This appeared mild and all the liver-function tests, with the exception of a slight elevation of serum bilirubin, were normal: it was not considered likely to have contributed significantly to the portal hypertension. An episode of jaundice in 1961 was probably caused by an intercurrent infective hepatitis. Thirdly, signs of mastocytosis appeared at the same time as the first symptoms of portal hypertension. The mastocytoses are generally regarded as members of the group of orthoplastic reticuloses. Their cutaneous manifestations are always the most prominent, but visceral and osseous involvement, often occult, may be associated. In most cases the disease is entirely benign, and in children it may disappear spontaneously, but rare cases of malignant cutaneo-visceral mastocytosis have been reported.

Minor inconstant changes in the blood picture have often been reported in patients with benign mastocytosis, and tissue mast cells have been found in the peripheral blood in a few patients with malignant mastocytosis.[3]

Mastocytosis has occasionally been associated with overt diseases of the haemopoietic system, e.g. with Cooley's anaemia,[4] lymphocytic leukaemia,[5] myeloblastic leukaemia,[6] monocytic leukaemia,[7] Hodgkin's disease,[8] and polycythaemia.[1]

It has long been recognized that PRV is not a disease of the erythropoietic system alone. Leukocytosis is a feature of PRV, and immature cells of the leukocyte series are occasionally discovered. Parkes Weber[9] coined the term 'erythroleukaemic chain' to define the trail of transition that could be followed between PRV and myeloid leukaemia, and Schwarz[10] summarized 104 cases from the world literature in support of the theory. Merskey,[11] in a comprehensive review of all available material up to 1946, concluded that, although some degree of myeloid hyperactivity is the usual finding in PRV and some patients may show a leukaemoid blood picture, true polycythaemia is very rarely associated with true leukaemia. PRV occasionally progresses to chronic non-leukaemic myelosis, a condition whose relationship to leukaemia is not disputed.

The association of portal cirrhosis of the liver with polycythaemia has previously been reported by Mosse,[12] but it is not clear whether the relationship is more than casual. Kratzeisen's[13] suggestion that portal-vein thrombosis may cause polycythaemia has nothing to support it.

This case, in which an orthoplastic reticulosis accompanied a myeloproliferative process, adds weight to the unitary theory of the diseases of the reticuloendothelial and haemopoietic systems.

We are indebted to Dr. L. van R. Oosthuizen, Port Elizabeth, Dr. W. C. Harington, South African Institute for Medical Research, Port Elizabeth, and to Prof. H. W. Weber, Department of Pathology, University of Stellenbosch, for assistance in the investigation of this case.

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SKIN DISEASE IN THE WESTERN CAPE PROVINCE

A SURVEY OF THE INCIDENCE OF SKIN DISEASES IN 2,500 WHITE AND 1,500 COLOURED PATIENTS

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Surveys of the incidence of skin diseases in the White[1] and Bantu[2] populations of the Transvaal and Orange Free State have recently been published by Findlay, Scott and Schultz. The present study serves to illustrate the position of the Coloured people in relation to the other two major racial groups in this country. As the number of Bantu patients attending our clinics is too small to allow analysis, we quote Findlay's figures from Pretoria.[2]

In the addendum to this article (Table I) the common dermatoses (those with an incidence of 1% or more) are shown in order of frequency; the rarities, relative or actual, are then listed alphabetically.

Column 1 gives the diagnoses in 1,000 consecutive patients seen in private practice (J.M.) in Cape Town during 1962 and 1963. The patients came in about equal numbers from the city of Cape Town and from the countryside of the Western Cape. About 70% were Afrikaans-speaking, 30% English-speaking.

Column 2 refers to 1,500 outpatients who attended the Karl Bremer Hospital, Bellville, CP, during 1962 and 1963. Most were Afrikaans-speaking.

Column 3. The 1,500 Coloured patients attended the Karl Bremer Hospital during 1962 and 1963. They all came from Cape Town and its immediate vicinity (e.g. Paarl, Stellenbosch).

Column 4. The figures here are taken from Schultz, Findlay and Scott's report on dermatoses in the Bantu.[2] The 2,000 cases were seen at the Pretoria General Hospital between 1959 and 1961.

Notes

Eczema. Table II shows a breakdown of the eczemas. It will be noted that infective eczema is the variety most common in the Coloured people. The high incidence of eczema (27%) in Bantu patients in Pretoria, is unexpected. Eczema has always been reputed to be relatively rare

in the Negro, and the available statistics bear this out; e.g. in Bloemfontein 9·7%[2]; in Lagos, Nigeria, 6·1%[3]; in Lourenço Marques, Moçambique, 4·4%[4] (5,803 cases 1956 - 1960, Farrajota Ramos); and in Dakar, Senegal, 16·1%[5] (mainly infective eczemas—1,633 cases, 1961, Basset). The tendency of the urban Negro to develop disease patterns akin to those of the White has been noted elsewhere.[6,7]

Acne vulgaris is rare in Coloured people, and the sex incidence, 1·6 to 1 in males and females, is the reverse of that in Whites.

Fungus infections. Scalp ringworm, caused by *Trichophyton violaceum* in the vast majority of cases, is the commonest fungus infection in the Coloured population (children); athlete's foot predominates in Whites (adults) with *T. mentagrophytes* as the most frequent cause. *T. rubrum* infections are rare and, in this series, were encountered only in White immigrants from Europe. None of the deep mycoses figure in this particular group of cases.

Tumours. The incidence of solar and senile keratoses, rodent ulcer, squamous carcinoma, moles and birthmarks is notably lower in the Coloured and the Bantu than in Whites. Seborrhoeic warts took the form of dermatosis papulosa nigra in our Coloured patients. Keratoacanthoma was not encountered in the Coloured, but we are informed by Belisario[8] and by Oettlé[9] that cases have now been discovered in Negroes.

The presence of seborrhoeic warts (benign basal-cell papillomas) on the white skin seems to imply a fairly high degree of immunity to rodent ulcer and squamous carcinoma.

Light eruptions. The darkly pigmented skin offers protection against all the diseases in which photosensitivity is a factor, with the exception of chronic discoid lupus erythematosus. Among the Whites, fishermen are the most liable to develop chronic solar dermatoses and skin cancer, with farmers next. The 2 Coloured men presenting with solar cheilitis were both fishermen.

Lichen planus presents much the same picture in all races in South Africa, and the florid forms seen in Negroes in tropical Africa are rarely encountered. Our observation that the buccal mucosa is very rarely affected in Negroes is confirmed by Basset[10] in Senegal.

Malnutrition. The customs and food habits of the Coloured people have been much the same as those of their White neighbours of similar economic status for the last century, which explains their low incidence of pellagra as compared with that in the Bantu.

Parasitoses. Only scabies and lichen urticatus, caused almost always by fleas, are common. Sandworm (larva migrans) infestation is rarely contracted in the Western Cape, and the solitary case of myiasis was imported from Southern Rhodesia.

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ADDENDUM

TABLE I. PERCENTAGE ENUMERATION OF DERMATOSES

Column number	*1*	*2*	*3*	*4*
Number of cases	*1,000*	*1,500*	*1,500*	*2,000*
Source	*Cape, private practice*	*Cape, hospital*	*Cape, hospital*	*Pretoria, hospital*
Group	*White*	*White*	*Coloured*	*Bantu*
	%	%	%	%
1. Eczemas	27·8	32·0	36·7	27·0
2. Acne vulgaris	11·9	9·3	1·7	9·15
3. Fungus infections	7·9	8·1	8·3	1·85+2·4
4. Solar and senile keratoses	4·9	2·2	—	—
5. Psoriasis	4·9	5·0	3·3	1·45
6. Rodent ulcer	4·5	2·5	0·06	—
7. Warts	3·0	4·5	5·0	2·45+1·45
8. Pityriasis rosea	2·3	1·6	1·2	2·4
9. Moles	2·2	1·0	—	—
10. Seborrhoeic warts	2·0	0·6	0·1	—
11. Pruritus ani et vulvae	1·8	0·3	0·06	—
12. Vitiligo	1·6	0·3	1·2	0·15
13. Urticaria	1·5	2·5	1·9	1·7
14. Light eruptions	1·5	0·6	0·4	0·2
15. Solar cheilitis	1·5	0·6	0·1	—
16. Impetigo	1·3	3·0	11·6	9·4
17. Alopecia areata	1·2	0·6	0·8	0·2
18. Lichen planus	1·2	0·6	1·0	0·9
19. Lupus erythematosus, chronic discoid	1·2	0·6	1·2	0·6
20. Pyoderma (excluding impetigo)	1·1	3·0	5·6	3·1+1·6
21. Lichen urticatus	1·0	2·5	1·0	2·05
22. Scabies	0·6	2·6	6·0	2·4+1·5(?)
23. Erythema multiforme	0·2	1·3	1·0	2·1
24. Varicella	0·1	0·1	1·0	—
25. Pellagra	—	—	1·3	6·3
26. Syphilis	0·1	0·06	1·2	4·35
Acne necrotica	0·2	—	—	
Acrodermatitis perstans	—	0·06	0·2	
Acrokeratosis verruciformis	0·1	0·06	—	
Acrosclerosis	—	0·06	0·06	
Alopecia, male type, in women	0·6	0·06	—	
Anetoderma (Schweninger — Buzzi)	—	—	0·06	
Angiokeratoma (Fordyce)	—	0·06	—	
Aphthosis, buccal	0·3	0·20	0·06	
Aphthosis, buccal and genital	0·1	0·06	—	
Balanoposthitis	0·1	0·06	0·06	
Capillaritis, allergic	0·1	0·3	—	
Carcinoma, squamous	0·6	0·8	—	
Chancroid	0·2	—	0·06	
Cheilitis glandularis	—	0·06	0·06	
Chloasma	0·3	0·2	—	
Cimex lectularius	0·1	—	—	
Clavus and callus	0·2	1·0	0·2	
Cutaneous horn	0·3	0·1	0·06	
Dermatitis herpetiformis	—	0·4	0·5	
Dermatofibrosarcoma	0·1	—	—	
Dyskeratosis congenita	—	—	0·06	
Ectodermal defect, congenital	0·1	—	—	
Epidermolysis bullosa	—	—	0·06	
Ephelides	0·1	0·06	—	
Erythema chronicum migrans	—	0·2	—	
Erythema marginatum	0·1	—	0·1	
Erythema nodosum	—	0·4	0·3	
Erythema induratum and nodular vasculitis	—	—	0·1	
Erythroderma, congenital ichthyosiform	0·1	—	—	
Fixed eruption	0·3	0·1	0·2	
Génodermatose en cocardes (Degos)	0·1	—	—	
Gonorrhoea	—	0·06	0·1	
Granuloma annulare	0·3	0·5	—	
Granuloma pyogenicum	—	0·2	0·1	
Haemangioma	0·9	0·3	0·2	
Hair-fall, cyclical	—	—	0·2	
Herpes simplex	0·8	0·7	0·3	
Hidrosadenitis suppurativa	0·1	—	0·2	
Histiocytoma	—	0·2	—	
Hyperhidrosis	0·1	0·3	0·06	
Hypertrichosis	0·2	—	—	
Ichthyosis vulgaris	0·1	0·2	0·2	

Column number	*1*	*2*	*3*
Number of cases	*1,000*	*1,500*	*1,500*
Source	*Cape, private practice*	*Cape, hospital*	*Cape, hospital*
Group	*White* %	*White* %	*Coloured* %
Incontinentia pigmenti (Bloch-Sulzberger)	—	—	0·06
Keloid	0·3	—	—
Keratoacanthoma	0·2	0·06	—
Keratosis follicularis	0·6	—	0·06
Keratosis palmo-plantaris	—	0·2	0·2
Keratosis pilaris	0·1	—	—
Leprosy	—	—	0·1
Leukoplakia, buccal	0·5	0·5	—
Leukoplakia, genital	—	0·06	—
Lichen sclerosus et atrophicus	0·1	0·2	0·1
Lipoid proteinosis	—	0·06	0·1
Livedo reticularis	0·1	—	—
Lupus erythematosus, systemic	0·2	—	0·1
Lupus vulgaris	0·2	0·2	—
Lymphadenosis benigna cutis	0·1	0·06	—
Lymphangioma	0·1	—	—
Melanoma, malignant	0·4	—	—
Milia	0·1	—	—
Miliaria	0·5	0·3	0·06
Molluscum contagiosum	0·2	0·06	0·5
Mongolian spot	—	0·06	—
Morphoea	0·1	0·06	—
Mycosis fungoides	—	0·06	—
Myiasis, *Cordylobia anthropophaga*	0·1	—	—
Nail dystrophies	—	—	0·3
Necrobiosis lipoidica	—	0·06	—
Neurofibromatosis	—	0·06	0·06
Painful nodule of ear	0·1	0·2	—
Paget's disease of nipple	0·1	—	—
Panniculitis (Weber-Christian)	—	0·6	—
Parapsoriasis lichenoides	0·1	—	—
Pemphigus vulgaris	0·1	—	—
Perléche	—	0·1	—
Pernio	—	0·2	—
Pigmentation	—	0·1	0·1
Pili incarnati	0·3	—	0·06
Poikiloderma (Civatte)	0·2	0·06	—
Polyfibromatosis (Touraine)	0·1	—	—
Porphyria	—	0·6	0·3
Porokeratosis (Mibelli)	0·2	—	—
Progressive hemiatrophy	—	—	0·06
Pruritus, generalized	0·2	1·0	0·3
Pseudopelade	0·2	—	0·06
Pseudoxanthoma elasticum	—	—	0·06
Purpura	0·3	0·2	—
Radiodermatitis	0·2	0·06	—
Rhinophyma	—	0·1	—
Rosacea	0·7	0·6	—
Sandworm (larva migrans)	—	—	0·8
Skin tags	0·6	0·3	—
Striae distensae	0·1	—	—
Sycosis barbae	—	1·0	0·7
Telangiectasia, all types	0·2	0·3	—
Thrombophlebitis migrans	0·1	—	—
Tinea amiantacea	0·2	0·1	0·06
Tongue, geographical	0·2	0·2	0·06
Tongue, hairy black	0·1	—	—
Trichotillomania	—	0·06	—
Urticaria pigmentosa	0·4	—	—
Vaccinia	—	0·06	—
Xanthelasma palpebrarum	0·1	—	—
Xanthomatosis	0·3	—	—
Zoster	0·3	0·6	0·5

TABLE II. ANALYSIS OF THE ECZEMAS

Number of cases	*278*	*479*	*538*
Source	*Cape, private practice*	*Cape, hospita*	*Cape, hospital*
Group	*White* %	*White* %	*Coloured* %
Atopic	20·7	17·5	14·1
Nummular	15·7	8·1	9·7
Seborrhoeic	12·8	9·8	6·7
Infective	10·7	17·3	26·8
Lichen simplex chronicus	6·1	3·8	5·8
Pityriasis alba	5·0	4·8	8·4
Housewife's hands	3·9	7·1	4·5
Varicose	2·8	7·1	4·5
Lichen striatus	0·4	0·2	—
Unclassified	—	6·5	8·9
Contact:			
Cosmetics	6·1	1·3	0·2
Medicaments	4·3	4·8	2·8
Clothing	3·9	3·1	1·3
Plants, flowers	1·4	0·4	0·2
Industrial	1·4	1·7	1·3
Factitious	—	0·6	0·2
Various	3·9	5·8	4·5

THE BICKERSTETH MEDICAL SOCIETY : DIE BICKERSTETH MEDIESE VERENIGING

CLINICAL EVENING, SOMERSET HOSPITAL, TUESDAY 19 NOVEMBER 1963

Cases Presented

1. Mr. L. Blumberg presented a 48-year-old man with a parotid calculus and superimposed infection. As infection continued recurring in spite of conservative management and antibiotic therapy, it was necessary to perform partial parotidectomy with removal of the calculus.

Rose's well-known series of salivary calculi was quoted, and it was stressed that the parotid is a relatively rare site for the formation of calculi. It was affected in only 3 out of the 75 cases in Rose's series.

The clinical corollary is that the presence of a purulent discharge from the submandibular gland frequently indicates an underlying calculus, whereas the presence of a purulent discharge from the parotid duct is accompanied by a calculus in only a minority of cases.

2. Dr. H. Edelstein presented the preliminary findings in a 25-year-old woman who posed a serious therapeutic problem. She had suffered from diabetes mellitus for at least 8 years and was now 28 weeks pregnant. She had signs of diabetic neuropathy. The presence of cataracts prevented examination of the fundi for signs of diabetic retinopathy. She was oedematous, the blood pressure was 150/100 mm.Hg, and the urine contained 3+ albumin, the blood urea was 21 mg./100 ml., the serum albumin was reduced to 2 G./100 ml., and the serum cholesterol raised to 370 mg./100 ml.

Investigations were being undertaken to try to determine whether her renal lesion was due to pre-eclamptic toxaemia in a diabetic, or whether she had the histological lesions of the Kimmelstiel-Wilson kidney. Renal biopsy was being undertaken, and constant consideration was being given to the time for undertaking termination of the pregnancy.

3. Dr. O. Meyers outlined in detail the case-history of the first uraemic patient successfully treated with peritoneal dialysis at Somerset Hospital.

Appreciation was expressed of the interest and support of Dr. T. H. L. Jones, Acting Medical Superintendent.

The aetiology of the anuria and uraemia was acute intravascular haemolysis. The patient was a bright, cheerful 13-year-old African girl. On 21 October she awoke complaining of abdominal pain, and was given 'epsom salts'. That day she felt sufficiently ill to go to bed, and her father, a medical orderly, noticed that her nails were blue. There was an indefinite history that, prior to her illness, she had played with a packet in the street. The packet was practically empty but contained some 'powder'. Its nature could not be determined.

On examination her nails, lips and buccal mucosae were blue. The serum and the urine were black. Investigations performed by the Department of Chemical Pathology, University of Cape Town, revealed that there was methaemoglobin and methaemalbumin in the serum, and methaemoglobin in the urine.

This intravascular haemolysis was accompanied by anaemia and by albuminuria. Severe oliguria developed and eventually the urine volume fell to 25 ml. in 24 hours. The blood urea rose progressively to 490 mg./100 ml., the serum potassium started rising, mild acidosis developed, and she became drowsy. The situation was critical, and on the 8th day of her renal 'shut-down' it was decided to undertake peritoneal dialysis.

This treatment, which uses the peritoneum as a dialysing membrane, was initially introduced in 1923. It was vir-