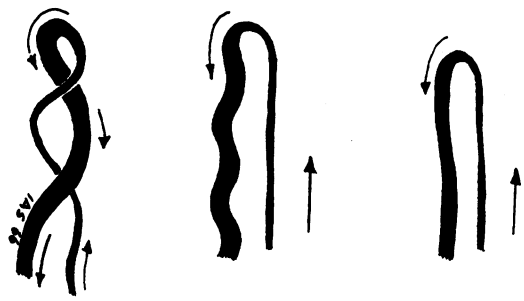

Fig 2 *Diagram illustrating the three basic morphological forms of microvessels of the posterior nail folds*

Fig 3 *Microvessels of posterior nail folds of R P S. None of the basic forms can be seen*

was observed which was not readily altered by constriction tests or applied temperature changes. However, in R P S none of the basic microvascular forms could be seen (Fig 3) in any of the posterior nail folds. All the microvessels were grossly dilated, and most showed unusual tortuosity. Anastomoses were frequent, not only between major limbs of the different planes of microvessels, but also between vessels of adjacent planes. Rapid changes in the direction of blood flow were readily observable and such changes could easily be produced by constriction or merely by the patient exerting pressure on his own finger tips.

The microvascular changes seen in this case have been observed in other cases of malignant disease, particularly of neoplasia involving mediastinal structures and the medulla of long bones. These associations at present defy interpretation, but they are worth detailed research.

Dr Louis Forman: The causal relationship between carcinoma in various situations and dermatomyositis is well accepted and the changes in the blood vessels, connective tissue and muscle are assumed to be mediated by an autoimmune mechanism. Recently a man aged 55 complained of a dusky erythema of the face and eyelids, with some œdema, and erythema of the chest and back of the hands. The condition had been present for three months. Although he felt quite well, dermatomyositis was diagnosed and admission to hospital advised, for the purpose of screening for a cryptic carcinoma. Within ten days he passed blood in the urine, and developed a rapidly progressing muscular weakness. At operation a large carcinoma of the bladder was removed. Dr Suzanne Alexander made an extract of this tumour and showed that the tumour extract with the patient's serum was able to fix complement. The dilution of serum required to fix complement rose from 1:8 to 1:320 during the ten days before operation. Again, Dr Alexander was able to demonstrate fluorescence in the papillary body of the skin, and between the muscle bundles suggesting globulin antibodies at these sites.

Dr M Feiwel: In a case of dermatomyositis associated with a bronchial carcinoma we found that the titre of antinuclear factor fell considerably after the neoplasm was resected. This coincided with a marked (though only temporary) remission in the severity of the dermatomyositis.

Behçet's Syndrome with Large Bowel Involvement

C A Ramsay MB MRCP
(for J S Pegum MD FRCP)
(*London Hospital, London*)

P W, male, aged 16. Journalist

History: Four and a half years' recurrent ulceration of the mouth. Eighteen months' recurrent skin lesions, consisting of erythematous areas with ulceration, and ulceration of the scrotum. The symptoms waxed and waned independently of treatment with various topical steroids. The mouth ulcers became very much worse, and at the end of February 1966 he was admitted to hospital.

On admission: Furred tongue; ulcers of tongue, palate, cheeks and inner aspects of lips with ulcerative lesions of thighs and scrotum. Infiltrated erythematous plaque at the back of the neck. Biopsy of scrotal lesion consistent with Behçet's disease.

Course and treatment: Treatment with prednisone 40 mg per day started on March 9, 1966, with some improvement in mouth ulcers initially but they became worse again on the same dose of steroid. Carbenoxolone pellets and ointment were

also used, but with little effect. The ulcers seemed to be following their usual course despite treatment, and the steroids were slowly withdrawn. On April 3, when the dose of prednisone was 20 mg a day, he developed diarrhœa. This was thought to be infective, a specimen was taken and phthalylsulphathiazole was given. The diarrhœa became bloody and the patient more ill with slight fever for two days. Stool culture was negative. Sigmoidoscopy under general anæsthesia showed anal ulcers, velvety mucosa and small ulcers in colon, i.e. the picture of acute ulcerative colitis.

The prednisone was increased to 50 mg a day. Prednisolone enemas b.d. and sulphasalazine were given. He improved slowly, the diarrhœa ceased and he was having only one bowel action a day. The enemas were reduced to one daily, the sulphasalazine was withdrawn and the oral steroids were slowly reduced. Mouth ulcers appeared again, but on this occasion responded well to hydrocortisone pellets.

On May 7 there was a further exacerbation of his colitis with bloody diarrhœa. Enemas were increased to two daily and oral steroids to 50 mg a day, producing improvement with one to two motions, containing some mucus, a day. The mouth ulcers healed and the mucosa now showed scarring only. The effects of systemic steroid therapy were apparent with increased weight, slight mooning of the face and the appearance of acne on the front of the chest.

Investigations: Hb 103%. WBC 7,800. ESR 32 mm in 1 hour (Westergren). Stool normal. Swab from mouth ulcer: normal flora, no viruses isolated. Blood urea 21 mg/100 ml. Prothrombin index 100%. Plasma proteins: total 6·5, albumin 4·6, globulin 1·9 g/100 ml. Plasma fibrinogen 300 mg/100 ml (normal). ECG normal. Intradermal test with 0·2 ml saline negative. Chest X-ray normal.

Biopsy from back of neck: Subacute purulent inflammation around vessels in dermis; one patch of parakeratosis and thinning of epidermis. Involvement of epidermis by inflammatory cells is very slight; no real spongiosis or folliculitis and no ulceration (Dr J W Landells).

Biopsy from scrotum: Acute purulent inflammation of dermis with ulceration of epidermis; one sweat gland involved. No evidence of primary vasculitis (Dr J W Landells).

Comment

The involvement of the gastrointestinal tract in Behçet's syndrome has been reported infrequently. Bøe *et al.* (1958) described 4 fatal cases of mucocutaneous-ocular syndrome with intestinal involvement. From the descriptions given 2 cases were of Behçet's syndrome. One patient died of central nervous system involvement and although there were no bowel symptoms post-mortem showed mucosal atrophy of the whole of the gastrointestinal tract. The other patient had severe bloody diarrhœa and colostomy was performed. Post-mortem showed colitis but no ulceration.

Oshima *et al.* (1963) described the clinical features of 85 cases of Behçet's syndrome seen in Japan. Gastrointestinal symptoms including nausea, abdominal pain and diarrhœa were present in approximately 40%.

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Bøe J, Dalgaard J B & Scott D (1958) *Amer. J. Med.* 25, 857

Oshima Y, Shimizu T, Yokohari R, Matsumoto T, Kano K, Kagami T & Nagaya H (1963) *Ann. rheum. Dis.* 22, 36

Dr Louis Forman: Disease of the bowel in association with Behçet's syndrome has been recorded by J S Jensen (1944) in 2 patients. The first was a man aged 29 who had ulcers of the mouth, joint swelling and hæmorrhagic ulcerative colitis. The second patient also died with ulcerative colitis. This author referred to papers by Bechgaard (1940) and T Jensen (1940) each describing a case of Behçet's syndrome with segmental ileitis, and hæmorrhagic ileitis and colitis respectively. The Japanese literature provides references for this association. Thus, Tsukada *et al.* (1964) describe a case of neuro-Behçet with perforating ulcer of the ileum and ileocæcal region. Dr Helen Curth (1964) in a paper read at the Academy of Dermatology, Chicago, detailed the history of a man diagnosed as having Behçet's syndrome with recurrent oral, genital and cutaneous ulcers. After five years he developed rectal ulcers, sloughing of the lining of the rectum and multiple perforations of the colon. Proctocolectomy was performed but the ulcers of the mouth and genitals recurred for a year up to the time of record. Crohn's disease may affect the colon primarily and be associated with the changes in the eye, mouth, skin and veins included under Behçet's syndrome. The wide spectrum of changes recorded in the small intestine and colon would suggest that different pathological mechanisms are responsible. This would reinforce the view that Behçet's syndrome, particularly when fragmentary, can be a manifestation of diverse pathologies.

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Jensen J S (1944) *Ugeskr. Laeg.* **106**, 176

Tsukada S, Yamazaki T, Iyo S, Nishio I, Hashimoto K & Matubara F (1964) *The Newest Medicine* (Japan) **19**, 1533

Dr C A Ramsay: Prior to the acute onset of colitis this patient had had no bowel symptoms and prednisone in a dose of 40 mg daily had had no effect on the mouth ulcers.

The mouth ulcers healed, but others developed later after the colitis was controlled, and these were less severe than usual. An exacerbation of his colitis when the prednisone was reduced was not accompanied by further mouth ulcers.

The following cases were also shown:
Pityriasis Rubra Pilaris Dr P F Borrie
Case for Diagnosis – ? Histiocytosis of Unknown Variety – ? Generalized Necrobiosis Lipoidica
Dr P W M Copeman (for Dr D G Freshwater, Dr P D Samman and Dr C F H Vickers)
Basal Cell Nævus Syndrome
Dr M Keir (for Dr R P Warin)
(1) **Congenital Skin Defect, Epidermolysis Bullosa and Nail Dystrophy**
(2) **Connective Tissue Nævi and Cerebral Atrophy**
Dr M McKelvie (for Dr P J Hare)
Cryoglobulinæmia
Dr M L Johnson and Dr H S Platt (for Dr P J Hare)
Acanthosis Nigricans
Dr T Robinson (for Dr R M B MacKenna)
(1) **Nodular Vasculitis**
(2) **Oculo-cutaneous Albinism with Multiple Solar Keratoses**
Dr T Robinson (for Dr P F Borrie)
Granuloma Annulare involving the Eyelids
Dr L Fry and Mr Barrie Jay
Giant Pigmented Nævus of the Scalp with Multiple Cutaneous Nævi
Dr A W McKenzie

A paper entitled **An Advance in the Treatment of Psoriasis using Triacetoxyanthracene – a New Dithranol Derivative,** was read by Dr F F Hellier, Dr H Yarrow and Dr M Whitefield.

Meeting June 16 1966

Cases

? Morphœic Basal Cell Carcinoma of Left Cheek
P Hall-Smith MD FRCPEd
(*Brighton General Hospital, Brighton*)

J F B, man aged 37
History: Since 1963 lesion left malar area which he thinks has extended in the past twelve months.
Past history and family history not relevant.
On examination: Pearly white, partly pigmented, indurated patch left malar area, 5 cm in diameter, extending to lower lid margin.
Histopathology of fragments of skin 0·5 cm long: The epidermis is thinned and the rete pegs have disappeared. There is some hyperkeratosis. The upper dermis shows changes in the collagen which is pale staining and has lost its fibrillary appearance. Beneath this is some chronic inflammatory infiltration, particularly around small vessels. The appearances are of lichen sclerosus et atrophicus rather than of scleroderma.

Comment
Christianson *et al.* (1956) undertook a clinical study of 235 cases of localized scleroderma, 191 of which showed linear or localized sclerodermatous plaques. In this group precipitating factors antecedent to onset of scleroderma included 14 instances of infection and 14 of trauma. Onset was usually slow and insidious. Arthralgia and obscure abdominal pain were common concomitant symptoms. Anomalies of the spinal column were noted in 32 patients, 20 of whom had spina bifida occulta. Many other cutaneous, neurological and congenital abnormalities were noted though none of these applies to our case. Complications and residua consisted of pigmentation, calcinosis, contractures and facial hemiatrophy and unilateral atrophy of one or more extremities. Duration of activity was three-and-a-half months to twenty-five years.

In the *Yearbook of Dermatology* (1957–8) a comment on this paper says that it is interesting to note that the series includes 2 patients who also had lichen sclerosus et atrophicus which appeared several years after onset of morphœa; also that it is sometimes extremely difficult to differentiate these cases, both clinically and histologically.

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Christianson H B, Dorsey C S, O'Leary P A & Kierland R R (1956) *Arch. Derm., Chicago* 74, 629
Yearbook of Dermatology and Syphilology (1957–8) p 190

Dr B Bentley Phillips: Has anyone had any further experience of the use of potassium para-amino benzoate in this type of condition? Would it be worth a trial?
Dr S C Gold: Regarding the effectiveness of potassium para-amino benzoate, I can remember asking, at the meeting of the British Association of Dermatology in 1965, if anyone present could recall a patient who had improved as a result of its use and there was no reply.
Dr Louis Forman: May I suggest the diagnosis of a morphœic basal cell carcinoma?
Dr Denis Sharvill: I would like to support the suggestion of possible basal cell carcinoma. A young man of 18 consulted me with clinical morphœa of the face, apparently confirmed by a small biopsy, and accepted by a senior colleague and by the plastic surgeon to whom we referred him for reconstruction of his lids. Only in the gross surgical specimens did we find very scanty strands of basal cells. Later he developed more typical, ulcerating rodent ulcers.
Dr H W Chadfield: I thought there was rather a lot of erythema and scaling in evidence and it seemed to me that this was a case of lupus erythematosus.
Dr E Wilson-Jones: The degree of scarring and hyalinization of the dermal collagen would be unusual in lupus erythematosus. We have had experience of a morphœic basal cell epithelioma in which no basal cells were detected in the initial biopsies.
Dr S C Gold: The patient mentioned by Dr Wilson-Jones was a man who complained of a scarring