diseases). These cells are widely spread in the dermal collagen and associated with lymphocytes and more ordinary histiocytes form a fairly dense perivascular infiltration; there is much streaky fibrin, but doubtful collagen necrosis.

The appearances suggest connective tissue breakdown taking an unusual biochemical course and exciting an unusual reaction; the great quantity of lipid and the absence of gross collagen necrosis in the dermis suggest that the primary change is breakdown of subcutaneous fat and that the dermal changes are due to the percolation of irritant breakdown products.

Very little of fat doubly refractile. Schultz test for cholesterol: negative. Test for mucin: very small amount present.

Comment

These lesions appear to be closely akin to those described by Shaffer (1938) as examples of liquefying panniculitis, and the histology, though not identical with that he described, is compatible with this diagnosis. Meanwhile a noncommittal name has been given, liquefying lipoid necrosis.

REFERENCE
Shaffer B (1938) *Arch. Derm. Syph., Chicago* **38**, 535

Dr N R Rowell: I would suggest that these lesions are artifacts. These patients are very clever and the lesions, even on the back, could have been caused by the injection of some chemical substance using a syringe and needle.

Dr R H Seville: I had a similar looking case where one or two of the lesions were arranged in lines, central leaking points were present, and at biopsy a strong smell of Dettol came from the oozing fluid. The relatives did not accept my verdict, so I asked Dr M Garretts to see her and he is investigating further.

Arthritis in Secondary Lues

I Sarkany MRCP

P M, female, aged 24 years

History: This woman from the West Indies was admitted to hospital on 18.1.65 with a three days' history of a rash on the trunk, a month's history of pains in knees and ankles followed by aching in both shoulders and severe pain and swelling of the right sternoclavicular joint. She had felt tired and weak for about one month. It was established that her blood WR was negative in an antenatal clinic one year previously, and her husband was found to have a positive WR in February 1965.

On examination: At the time of her admission, she was listless and looked ill. Her temperature was 99° F. There was a widespread papular eruption over most of the body surface, except the face, hands and the feet. There were no condylomata. There was a visible tender swelling of the right sternoclavicular joint and tenderness over both acromioclavicular joints.

She had pain on movement of both elbows and knees and effusions in both knees. She had a few small axillary lymph nodes and one small tender left occipital lymph node. The mucous membranes were normal and the liver and spleen could not be felt.

Investigations: Hb 82%. WBC 8,900. ESR 86 mm in 1 hour (Westergren). Protein: total 6·4, albumin 2·4, globulin 4·0 g/100 ml. WR, Reiter protein and fluorescent antibody: all positive. Rose's test and ANF: negative. Slide latex test: weak positive. Alkaline phosphatase: 29 K–A units.

Histology of the skin showed perivascular infiltration by lymphocytes, fibroblasts, histiocytes and plasma cells. The capillary endothelium was prominent. There was the formation of early granulomata composed predominantly of histiocytes.

X-rays showed marked destruction of the medial end of the right clavicle and partial destruction of the lower border of the medial end of the left clavicle. Disappearance of the outer ends of the right and left (Fig 1) clavicles was also noted. There was also effusion into both elbow and knee joints.

ECG showed marked myocardial changes.

Progress: The eruption began to improve before penicillin therapy (1 million units daily for fourteen days) was started on 28.1.65. On the night

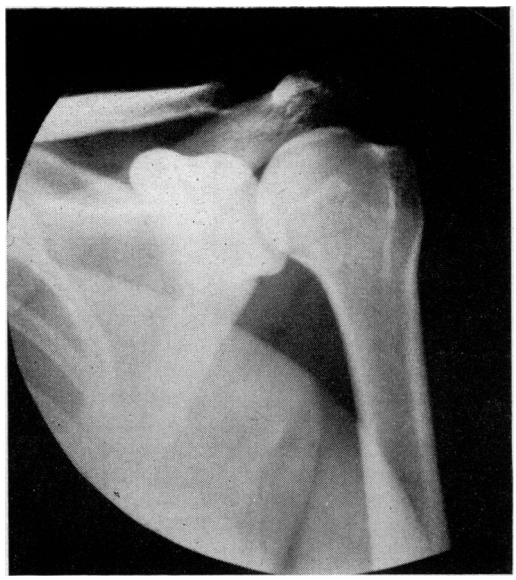

Fig 1 *X-ray changes in acromioclavicular joint*

after the first injection her temperature rose to 103° F and fell to normal next morning. The joints became less painful and the effusions disappeared. Complete clearing of the rash followed, there was gradual improvement of her general condition and the swelling over the right sternoclavicular joint disappeared. One month after the start of treatment the sedimentation rate was down to 35 mm in 1 hour (Westergren) and there was slight improvement in the X-ray appearances of the right sternoclavicular joint. At the end of two months the X-ray appearances of the joints showed a very marked degree of repair.

Comment

There is no doubt that this patient has secondary syphilis in view of the recently acquired positive blood Wassermann reaction, the skin changes and hydrarthroses. Although the bilateral symmetrical destructive arthritis could conceivably be an incidental associated finding due to another cause, such as rheumatoid arthritis, hyperparathyroidism or some infiltrative process, this is in fact unlikely, particularly because of her good response to penicillin treatment. These destructive osseous lesions are therefore considered to be due to the severe secondary luetic infection.

Destructive bony lesions in early syphilis though unusual are well documented. Reynolds & Wasserman (1942) found that the bones of the skull were most often involved and the sternoclavicular region was not infrequently affected. The long bones were more rarely attacked.

REFERENCE
Reynolds F W & Wasserman H (1942) *Arch. intern Med.* **69**, 263

Dr F J V Jenner: Joint involvement is commoner in gonorrhœa: could there be a double pathology in this case?

Dr E Wilson Jones: The histology was unusual for secondary syphilis. The infiltrate was becoming granulomatous and even tuberculoid in some of the foci. Their features are more in keeping with late than early syphilis.

Dr W B Reed: The X-rays do not show arthritis but rather lysis of the bone. A similar patient with secondary syphilis and a large lytic lesion in the skull which responded also to penicillin was recently presented at the Los Angeles County Hospital. Dr Marjorie Bauer found two references in regard to this phenomenon which Stokes did not describe in his very comprehensive book on syphilis. Perhaps in these patients there is less resistance to syphilis with greater destruction.

REFERENCES
Newman B & Saunders H C (1938) *N. Y. St. J. Med.* **38**, 788
Reynolds F W & Wasserman H (1942) *Arch. intern. Med.* **69**, 263

Dr Sarkany: In reply to Dr Jenner, the patient's GCFT on admission to hospital was negative.

The following cases were also shown:

(1) **Cystic Acne of Infancy**
(2) **Hansen's Disease**
(3) **Mycosis Fungoides with Unusual Skin Histology**
Dr F Bor

(1) **Basal Cell Carcinoma in both Axillæ**
(2) **Vagabond's Disease**
Dr M Feiwel

Follicular Mucinosis (Alopecia Mucinosa)
Dr H W Chadfield

Mycosis Fungoides
Dr P E Kilby (for Dr R H Meara)

Cutaneous Leishmaniasis
Dr J G Holmes

Scleroderma and Pancreatitis
Dr A B Shrank (for Dr F R Bettley)

Pyoderma Gangrenosum
Dr A B Shrank (for Dr R H Meara)

Yaws. Secondary Stage
Dr L Fry (for Dr J S Pegum)

Meeting March 18 1965

Cases

Basal Cell Nævi with a Neurological Syndrome
G A Caron MRCP (for Professor C D Calnan FRCP)

Mrs D M, aged 27

History: First investigated by neurologists at the age of 9 years because of rapid growth during childhood. Birth weight was 12 lb and at 8 years she was excessively tall and weighed 16 st 8 lb. The menarche occurred at this time. A craniopharyngioma was suspected and a ventriculogram performed. It was entirely normal and there was no intracranial calcification. Following this she was put on a diet and anorectic drugs and gradually lost weight.

At 24 years she developed a disturbance of micturition, passing urine every one and a half hours. Incontinence with dribbling then occurred. These symptoms are relieved by oral carbachol. Neurological examination revealed no abnormal physical signs but X-ray studies revealed extensive intracranial calcification which is considered to be causally related to the micturition difficulties.

Recently she was noted to have punctate lesions on the palms and several basal cell carcinomata on