Orthopaedic Consultation.—Congenital absence, multiple, in extremity bones; left upper and both lower limbs markedly shortened. Cleft palate, harelip repaired. Later in childhood, if indicated at that time, prosthetic considerations may be given.

Ophthalmological Consultation.—Right eye: Pupil 3^s regular and active homatropine—media clear; disks clear, good colour; vessels and maculae normal. Left eye, similar to right eye. Examination of media normal. No disease seen.

Psychological Consultation.—Gross motor patterns fall between the eight- and sixteen-week levels. Standing, unable to support any weight, below twelve weeks; supine, pulled to sitting, complete head-lag twelve weeks; prone, head zone III 16-week level. Adaptive patterns are somewhat higher; that is, radial palmar grasp 28 weeks; and transfers adeptly, 28 weeks. No verbalization elicited. Sphincter control absent; cannot sit up or feed herself. Chronological age: three years seven months; mental age, eight and two-tenths of a month; intelligence quotient 19. Classification: idiocy.

CONCLUSIONS

In view of the above findings, it appears that this child manifests the congenital malformation described as phocomelia, which is complicated by a severe degree of intellectual defect (idiocy), cleft-palate, hare-lip, skull anomalies, pedal syndactylism, and pilonidal sinus.

I am indebted to Mr. James Dutremaine, an employee at Letchworth Village, for the photograph of the patient.

FREDERICK EDWARD KRATTER, M.B., B.Sc., D.P.M.,
Senior Clinical Psychiatrist, Letchworth Village, Thiells, N.Y.

REFERENCES

Aschoff, L. (1928). *Pathologische Anatomie*, 7th ed. Fischer, Jena.
Church, A., and Peterson, F. (1911). *Nervous and Mental Diseases*, 7th ed. Saunders, Philadelphia.
Kaufmann, E. (1929). *Pathology*. Blakiston, Philadelphia.
Patten, B. M. (1946). *Human Embryology*. Philadelphia and Toronto.

Scorbutic Arthritis Complicating Triamcinolone Therapy

A case is described below in which fleeting muscle and joint pains followed by arthritis and effusion in the knee-joint associated with vitamin-C deficiency occurred in the course of triamcinolone therapy.

CASE REPORT

A man aged 50 was admitted to hospital on February 27, 1958, for investigation of bilateral basal pulmonary fibrosis. He gave a one-year history of progressive breathlessness, but nothing referable to any other system, and no past history of rheumatic trouble. He said that he had always taken a good mixed diet, and was fond of green vegetables and fresh fruit.

On examination he was well nourished, and there were no signs of avitaminosis. Physical examination was negative apart from the presence of marked finger- and toe-clubbing and central cyanosis. In addition crepitations were present at the bases of both lungs. Detailed examination of his other systems revealed nothing abnormal, and all laboratory investigations were within normal limits. Lung biopsy was carried out and the diagnosis of Hamman-Rich syndrome was confirmed on microscopical examination.

On May 9 he was started on triamcinolone, 16 mg. a day, in the hope of preventing extension of the pulmonary fibrosis, and on June 26 the dose was reduced to 8 mg. a day because of the improvement in his general condition. By July 25 marked moon-face had developed, but the blood picture and electrolytes remained normal. In view of this, and because of the absence of any other physical abnormality and of any deterioration radiologically, it was decided to continue with triamcinolone. By September 4 his condition had deteriorated. On examination he was more breathless and he had a marked tachycardia. His weight had fallen from 11 st. to 10 st. 2 lb. (70.3 to 64.4 kg.), but, apart from his moon-face, no fresh abnormality was found on clinical examination. His blood chemistry and electrolytes remained as before. By September 25 he was *in extremis*. His triamcinolone was increased to 16 mg. t.d.s., and on this massive dosage he improved dramatically. The blood chemistry remained normal.

He developed a purpuric rash on the legs and feet on October 2 and fleeting muscle and joint pains on October 26. His dose of triamcinolone was slowly reduced. On October 30 pain was localized to the left knee, which was stiff and swollen. There were clinical signs of effusion into the joint, but *x*-ray examination was negative. The knee was treated with support and rest, but remained swollen and painful. The rash also persisted. At this time the dose of triamcinolone was 4 mg. t.d.s.

On November 13 ascorbic-acid-saturation tests were started, 650 mg. of vitamin C being given each morning and the urinary excretion of vitamin C being measured in the 10 a.m.–12 noon collection. The normal excretion value (greater than 50 mg.) was not reached for seven days, by which time the rash was fading and the joint pain and swelling had disappeared. Triamcinolone had not been discontinued.

COMMENT

Arthritis has been reported by Polley (1956) as a complication of corticosteroid therapy. Indeed, he quotes Slocumb (1953), who described characteristic fleeting muscle and joint pains as a symptom of hyper-cortisonism. Arthritis has also been described as a complication of triamcinolone administration (Wells, 1958), while the development of scurvy has been observed in patients on long-term corticosteroid therapy (Stefanini and Rosenthal, 1950; Holley and McLester, 1951). The case described above would appear to combine all these abnormalities. It is not recorded whether or not there was any evidence of scurvy in the earlier patients suffering from arthritis.

Chamberlain and Addison (1958) described the development of scurvy after bilateral adrenal-ectomy. It is highly probable that the same mechanism operated in the case described above, in that the massive doses of triamcinolone given would totally suppress the patient's own adrenocortical function.

In view of the findings in this case it is probably desirable that patients given long-term corticosteroid therapy, and in particular patients receiving triamcinolone, should be given vitamin-C supplements. The patient described above has been kept symptom-free despite continuing triamcinolone therapy on 50 mg. of ascorbic acid twice daily.

I thank Dr. C. Kelman Robertson for his permission to describe a case under his care, and for his encouragement in the writing of this report.

T. G. JUDGE, M.B., Ch.B.,
Senior House Officer, Industrial Chest Unit, Bangour General Hospital.

REFERENCES

Chamberlain, D., and Addison, N. V. (1958). *Brit. med. J.*, **2**, 1128.
Holley, H. L., and McLester, J. S. (1951). *A.M.A. Arch. intern. Med.*, **88**, 760.
Polley, H. (1956). *Brit. med. J.*, **2**, 1253.
Slocumb, C. H. (1953). *Proc. Mayo Clin.*, **28**, 655.
Stefanini, M., and Rosenthal, M. C (1950). *Proc. Soc. exp. Biol. (N.Y.)*, **75**, 806.
Wells, R. (1958). *Lancet*, **2**, 498.