On the 22nd day after admission the nail and surrounding tissue were removed to allow egress of this material. Repeated blood counts during this time showed a range of 8,500 to 11,500 leucocytes per c.mm., with 54-69% neutrophils. Subsequent urinalyses were all normal. The other gangrenous areas gradually separated after the 30th day in hospital, and by the 42nd day they had all separated, leaving clean granulating surfaces (Fig. 2). In none of these areas was separation deeper than the subcutis. They were dressed with petroleum jelly gauze, and all healed over the course of the ensuing 10 weeks. The gland in the right inguinal region did not break down, and gradually subsided. The patient was discharged from hospital on the 58th day after admission, surveillance being continued in the out-patient department. A small protrusion of the distal phalanx of the big toe sequestered 10 weeks later. No regrowth of the nail has occurred.

Comment

In the case here reported no association or predisposing factors could be determined. The history did not suggest a previous generalized infection. There was some support for a diagnosis of local infection in the presumed fever before admission to hospital and the convulsion, which must be assumed to have been febrile in origin. The lymphangitis in the leg and the later development of an enlarged tender lymph node in the groin also lent support to this diagnosis, but these may have been secondary to the pustules on the foot and leg. The distribution of the gangrene did not, however, suggest a local infection. The normal leucocyte count was also against an infection, and osteomyelitis of the tarsus was excluded by radiography.

Arteriosclerosis and thromboangiitis obliterans could be excluded in view of the age, and there had been no previous episodes to suggest a diagnosis of Raynaud's disease. The course of the illness did not suggest a diagnosis of periarteritis nodosa. Diabetes and syphilis could be almost certainly excluded. There was no haemoglobinuria and the serum was negative for cold haemagglutinins. No history whatever of trauma could be obtained, and frost-bite was out of the question.

Thus none of the accepted associated or predisposing factors in the causation of peripheral gangrene could be demonstrated in this case. The evidence leans more towards a local infection than any other aetiological factor.

Thanks are due to Professor R. S. Illingworth, who gave permission for the publication of the case report and for his encouragement and advice in the preparation of this paper.

M. G. PHILPOTT, M.B., M.R.C.P., D.C.H.

Department of Child Health,
University of Sheffield.

REFERENCES

- Craig, J. D., and Muns, W. E. (1931). *Amer. J. Dis. Child.*, **41**, 126.
 Hoyne, A. L., and Smollar, L. (1941). *J. Pediat.*, **18**, 242.
 Johnson, G. (1894). *British Medical Journal*, **1**, 900.
 Khautz, A. von (1914). *Z. Kinderheilk.*, **11**, 35.
 Lawrence, R. D., and McCance, R. A. (1931). *Arch. Dis. Childh.*, **6**, 343.
 Martin, W., and Shore, B. R. (1928). *Ann. Surg.*, **88**, 725.
 Stats, D., and Bullowa, J. G. M. (1943). *Arch. intern. Med.*, **72**, 506.
 Stoesser, A. V., and Lockwood, W. W. (1938). *J. Pediat.*, **12**, 641.
 Uhr, J. S. (1938). *Amer. J. Dis. Child.*, **56**, 1317.
 Watson, T. M. (1934). *J. Amer. med. Ass.*, **102**, 2179.
 Webb, B. D., Dubs, Elizabeth J., and Conrad, Elizabeth (1947). *J. Pediat.*, **30**, 76.
 Winstead, J. A. (1935). *Ibid.*, **6**, 382.

An Anomaly of the Sedimentation Rate in Rheumatic Diseases

In view of the extent to which the Wintrobe sedimentation rate is now being used in the rheumatic diseases, in particular for the assessment of steroid hormone treatment, it was thought that it would be useful to record in a preliminary note an anomaly we have observed, since it may be very misleading in such trials. A full account, it is hoped, will be published in the near future. It would appear from the paper published recently by Gilmour and Sykes (1951) that the phenomenon may not be confined to the rheumatic diseases.

In this unit during the past 10 months, largely owing to our participation in the Medical Research Council United States-United Kingdom scheme for the treatment of rheumatic fever with cortisone, etc., we have determined simultaneously the sedimentation rate by the Wintrobe and the Westergren methods. In some cases an anomaly in the sedimentation rate has been noted in that the Westergren method has given a very high recording, whereas, with blood taken from the same syringe, the Wintrobe method has given readings falling within the normal range or only slightly above it. The Westergren rate was a better index of the clinical state. The Westergren readings were virtually unaltered when dry sodium citrate was used instead of citrate solution as the anticoagulant. The following simultaneous weekly readings taken from a patient with disseminated lupus erythematosus illustrate the type of discrepancy that has been encountered.

Westergren readings (mm./hr.): 75, 84, 65, 43, 35, 36, 57, 98, 60, 90, 90.

Wintrobe readings (mm./hr.): 6, 5, 6, 5, 5, 14, 5, 16, 12.

The haematocrit readings on these occasions ranged from 40 to 43.

As most cases here are in-patients for prolonged periods, we have been able to observe the incidence of this anomaly and have attempted to correlate it with the clinical state. Cases showing this anomaly are those of Still's disease, rheumatoid arthritis, and disseminated lupus, and, very occasionally, acute rheumatic fever at the onset of the attack. In some cases the anomaly occurs consistently over several weeks. In other cases the phenomenon occurs sporadically. When it is noted, sedimentation rates are repeated in duplicate.

The clinical state of patients showing this discrepancy would lead one to expect a high sedimentation rate. In the example quoted the patient was febrile, had arthritis and a lupus rash, was losing weight, and had leucopenia.

In the cases showing these discordant readings, similar low values are obtained when heparin is used as an anticoagulant and the blood set up in either a Wintrobe or a Westergren tube. The addition of dry citrate to the previously oxalated or heparinized blood causes the sedimentation rate to rise to a level approximating that obtained with the Westergren method. Further investigation of the mechanism is proceeding. It appears probable that it depends on the anticoagulant's effect upon the pH.

The cases in this unit belong to a selected group (rheumatic diseases) in whom it is apparent that this phenomenon is not uncommon (10 out of 86 cases). It is not known yet to what extent this phenomenon occurs in other diseases. In the rheumatic diseases, at least, the assessment of activity by the Wintrobe method may be misleading.

SYLVIA GOLDBERG, M.R.C.P.

L. E. GLYNN, M.D., M.R.C.P.

E. G. L. BYWATERS, F.R.C.P.

Special Unit for Juvenile Rheumatism,
Canadian Red Cross Memorial Hospital,
Taplow, Bucks.

REFERENCE

- Gilmour, D., and Sykes, A. J. (1951). *British Medical Journal*, **2**, 1496.