by Perls's reaction for iron. The tubules of the kidney show cloudy swelling but no other lesion, and iron could not be demonstrated. The only other significant finding in the viscera was necrosis of the Malpighian corpuscles of the spleen.

Discussion

Although previous reports have dealt with the toxicology of this condition, this new case raises some points in the clinico-pathological synthesis that are of interest.

It appears from the microscopical examination that once the gastric mucosa is damaged the iron escapes from the lumen into the tissues and leads to thrombosis of the submucosal veins; this thrombosis would seem to act as a block, in that comparatively little iron has been found in the liver. None the less, it is difficult to escape the conclusion that the gross fatty change in the liver is due to absorption of substances from the lumen through the areas denuded of mucosa, or possibly from the breakdown products of the mucosa itself. The extent and degree of the change in the liver seem scarcely sufficient to explain the fatal issue, but in view of the failure to find any other cause of death one might assume that the damaged liver, although capable of sustaining life for some time, was not capable of maintaining its detoxicating process in the continued presence of this absorption. The final collapse of the liver, by allowing access of the absorbed substances to the rest of the body, led to the sudden exitus. The necrosis of the Malpighian corpuscles is probably associated with that generalized toxaemia.

The present case emphasizes again two important clinical points. First, it is of the utmost importance to begin treatment urgently for the sake of the gastric mucosa, as it seems probable that the gastric mucosa is the first and main line of defence of the liver. In this respect it is of interest to note that a recent paper described a case of recovery after the ingestion of similar pills in which treatment was started at once by the patient himself, and little evidence of liver damage could be found (Roxburgh, 1949). The histological findings in the present case fail to provide any suggestions for specific therapy, and one can merely repeat the probable value of early administration of alkali to form insoluble hydroxides. It would seem that dimercaptopropanol (BAL) is unlikely to be of help (McCance and Widdowson, 1946) in such cases; it was used by Roxburgh, who makes no claim, however, for its value.

The second point of clinical importance is the occurrence of a misleading period of improvement and apparent well-being before a rapidly fatal termination. A similar false recovery occurred in one of Forbes's cases, and the fact should perhaps be stressed that in treatment of such cases this period of betterment neither lessens the gravity of the prognosis nor permits any relaxation of therapeutic activity.

Summary

A case is described of fatal poisoning of an infant by pills containing iron, copper, and manganese.

The clinical course showed a period of apparent recovery followed by sudden death: this misleading sequence occurred also in one of the previously reported cases.

The histological findings suggest that the destruction of the gastric mucosa leads to lethal hepatic damage.

It is the duty of doctors and pharmacists to warn parents of the grave risk to infants presented by these visually attractive pills.

I wish to thank Professor A. C. Lendrum for his help and advice in the preparation of this paper and Mr. A. Dargie, Dundee City Analyst, for the analysis of the specimens of gastric and caecal content, bile, and urine. I am also grateful to Dr. J. Thomson, Royal Infirmary, Dundee, for the clinical details, and to Mr. J. W. Corkhill for the photomicrography.

References

Celsus, A. C. (? A.D. 200). *De Medicina.* Liber 4, Cap. 9.
Forbes, G. (1947). *British Medical Journal*, **1**, 367.
Hiyeda, K. (1939). *Jap. J. med. Sci.*, Sect. 5 (Path.), **4**, 91.
McCance, R. A., and Widdowson, F. M. (1946). *Nature*, **157**, 837.
Roxburgh, R. C. (1949). *Proc. R. Soc. Med.*, **42**, 85.
Scott Park, S. D., Scott, L. D. W., and Lendrum, A. C. (1947). *Brit. J. Radiol.*, n.s. **20**, 100.
Thomson, J. (1947). *British Medical Journal*, **1**, 640.

SYNOVIAL OSTEOCHONDROMATOSIS

TWO UNCOMMON EXAMPLES

BY

J. F. CURR, M.D., F.R.C.S.Ed.

Formerly Temporary Assistant Surgeon, Royal Infirmary, and Leith Hospital, Edinburgh; late Orthopaedic Specialist, R.A.M.C.

[WITH PHOTOGRAVURE PLATE]

Synovial osteochondromatosis is a well-recognized condition, but the two cases reported here presented some unusual features.

Case 1

The patient, a man aged 23, had a history of stiffness of the right shoulder for about six months, associated with occasional slight pain, but with no obvious cause beyond some trivial injuries in the past. On examination there was a complete absence of any movement in the right shoulder-joint.

Radiological examination disclosed enormous numbers of opaque loose bodies, of varying size and density, occupying the entire shoulder-joint and extending into the subscapular bursa and the synovial sheath of the long head of the biceps. Synovial osteochondromatosis was diagnosed (Plate, Fig. 1). There was no abnormality elsewhere.

At operation the right shoulder-joint was exposed through a long anterior incision. On opening the bulging synovial membrane loose bodies were scooped out by the dozen, but many

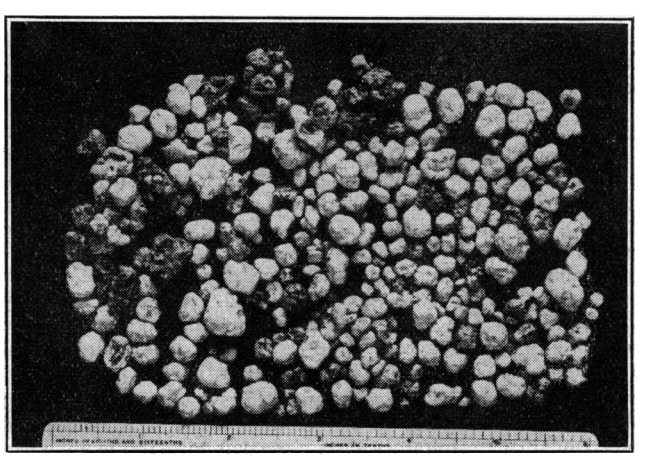

FIG. A.—Case 1. Photograph of loose bodies after removal.

still attached to it had to be avulsed or dissected out. An attempt at synovectomy was abandoned owing to tough adhesions and haemorrhage, but the whole of the synovial sheath of the long head of the biceps was excised and also as much as possible at the site of the reflection of the synovial membrane on the humerus. All loose bodies were removed after a complete exploration.

Pathological Condition.—The entire synovial membrane was red, rough, and oedematous. The earliest lesions were tiny nodules and small sessile excrescences, and were most pronounced at the reflection of the synovial membrane and in the bicipital groove. Larger bodies were hanging in clusters, and other solitary bodies possessed a mere hairlike pedicle. The largest bodies, up to 2 cm. in diameter, were lying completely detached and mobile within the joint. Those with any degree of pedicle were red and still retained a blood supply, but the actual loose bodies were yellow and more polished; all were somewhat rounded, with a pitted irregular surface. There were more than 350 osteochondromata, including several clusters not yet independent (Fig. A); very tiny ones still embedded in the synovial membrane were difficult to count accurately. Their total weight was 67.5 g. There was marked osteoarthritic lipping at the edge of the articular cartilage of the humerus, together with an erosion of the cartilage in the centre of the head where a loose body had been impacted.

Histological examination of a portion of the synovial membrane with the earliest changes demonstrated well-marked cancellous bone formation, as well as fibrous tissue and cartilage; there was extensive proliferation of the synovial tissue (Fig. 2).

Several of the loose bodies were examined radiologically after removal and showed a central opaque nucleus, surrounded by more dense peripheral laminae of calcification.

Progress.—Healing was uninterrupted, and no remaining loose bodies were found on subsequent radiological examination. Two months after operation a moderate degree of all movements had been restored, but further follow-up proved impossible. The formation of the osteochondromata appeared so active that it is almost certain that they will develop again, necessitating a second operation. A stiff shoulder is the probable final result, especially as osteoarthritis is already established.

Case 2

This patient, a man aged 55, sustained a trivial injury to the left ankle, and two weeks later was seen on account of swelling and moderate disability. Radiological examination revealed several opaque loose bodies, posterior to the ankle-joint, which were typical of synovial osteochondromata (Fig. 3). There were also early changes of osteoarthritis in the ankle-joint.

At operation a postero-medial incision was made and the loose bodies were found lying in a large bursa communicating with the synovial tendon sheath of flexor hallucis longus. Some were quite loose and others were attached in clusters to the synovial lining, which was otherwise healthy. All the osteochondromata were removed and the entire bursa was excised. A good recovery followed.

Comments on the Condition

Site of Origin.—Henderson and Jones (1923) observed that the site of reflection of the synovial membrane is the common site of origin of osteochondromata within a joint; this is the region of differentiation in the foetus, and is the most favourable situation for disarrangement of cells which in later life may take on properties not normally possessed. The findings in Case 1 are thus clearly in confirmation.

Nature of the Condition.—Osteochondromatosis is now generally accepted as being a benign neoplasm, developing from embryonic rests or from a metaplasia of connective-tissue cells. Although malignant synovial tumours are well known, it is seldom appreciated that malignant change may supervene on osteochondromatosis on very rare occasions; Geschickter and Copeland (1936) and Brailsford (1948) refer to cases in which a sarcoma developed. Bennett (1947) showed the necessity for following up all benign neoplasms or other tumour-like lesions of synovial tissue because of the risk of subsequent malignancy and also because there may be difficulty at first in distinguishing histologically between benign and malignant conditions. In Case 1 recurrence seems almost certain owing to the activity of the growth of osteochondromata, and it is interesting to speculate on the possibility of ultimate malignant change.

Growth of Bone.—The growth of bone and the increase in size of the osteochondromata are in keeping with the opinions of Greig (1931). He maintained that bone forms before a loose body becomes detached, for the reason that bone formation requires an adequate blood supply, not available after the loose body is once free. Any increase in size after detachment is then due to increase in the amount of cartilage, which can derive nutrition from synovial fluid. Calcification, as distinct from ossification, may occur thereafter in the periphery of the loose body.

Involvement of Shoulder-joint.—This is a very rare site, coming in order of frequency after knee, elbow, ankle, and hip joints. Albee (1927) removed 64 loose bodies, and Henderson and Jones reported another case, both being followed by recurrence. The condition was also demonstrated by de Lorimier (1943), Behrens (1947), and Brailsford.

Involvement of Bursae and Tendon Sheaths.—Being closely related to joints embryologically, these are subject to the same pathological conditions and may become the site of loose bodies. Osteochondromatosis is not common in them and may occur independently or in association with a similar joint lesion. References have been made, amongst others, by Henderson and Jones, Geschickter and Copeland, Buxton (1923), Janik (1927), and Shepherd (1943).

Numbers of Loose Bodies.—A solitary osteochondroma may occur. Very large numbers, however, have been recorded, such as 700 by Timbrell-Fisher (1921), 1,047 by Berry (quoted by Timbrell-Fisher), and 415 by Boyd (1947), all in the knee, and 1,000 by Brailsford in the hip. As a rule the number is much smaller, and the figure of at least 350 in Case 1 is well above the average.

Treatment

Operation is required if there is to be any hope of recovery of function of the joint or prevention of osteoarthritis. According to Timbrell-Fisher, the process of loose-body formation may end as suddenly and mysteriously as it began, and the results of operation are good provided the condition is not then progressive; otherwise there is recurrence and a second operation becomes necessary. Henderson and Jones extracted ten loose bodies from a shoulder-joint through a very small posterior incision, but, though possibly effective in certain instances, this limited type of exposure does not seem applicable in the presence of large numbers, especially if still attached to or involving synovial extensions of a joint. Wilmoth (1941) performed a partial synovectomy, with removal of loose bodies, in most of his cases, but was uncertain regarding the prevention of recurrence. A total synovectomy is the best method of ensuring against recurrence in a case with extensive involvement, but it is not attainable in all joints nor under such conditions as in Case 1.

Involvement of a bursa is easily treated by its complete excision. In a tendon sheath, simple removal of loose bodies is the usual treatment, but occasionally total excision of the synovial lining is possible.

Summary

Two uncommon examples of synovial osteochondromatosis are reported. In one case the shoulder-joint was involved most extensively, over 350 loose bodies being removed. In the other the loose bodies were in a bursa in direct communication with

a tendon sheath at the ankle: recovery followed removal of the osteochondromata and excision of the bursa.

I wish to acknowledge my indebtedness to Messrs. A. H. Baird, who carried out the photography.

REFERENCES

Albee, F. H. (1927). *J. Bone Jt Surg.*, **9**, 601.
Behrens, C. F. (1947). In *Clinical Radiology*, edited by G. V. Pillmore, vol. 2, p. 417. Davis, Philadelphia.
Bennett, G. A. (1947). *J. Bone Jt Surg.*, **29**, 259.
Boyd, W. (1947). *Surgical Pathology*, 6th ed. Saunders, Philadelphia.
Brailsford, J. F. (1948). *Radiology of Bones and Joints*, 4th ed. Churchill, London.
Buxton, St. J. D. (1923). *Brit. J. Surg.*, **10**, 469.
de Lorimier, A. A. (1943). *The Arthropathies*. Year Book Publications Inc., Chicago.
Geschickter, C. F., and Copeland, M. M. (1936). *Tumours of Bone*, rev. ed. American Journal of Cancer, New York.
Greig, D. M. (1931). *Clinical Observations on the Surgical Pathology of Bone*. Oliver and Boyd, Edinburgh.
Henderson, M. S., and Jones, H. T. (1923). *J. Bone Jt Surg.*, **5**, 400.
Janik, A. (1927). *Ann. Surg.*, **85**, 897.
Shepherd, J. A. (1943). *Brit. J. Surg.*, **30**, 179.
Timbrell-Fisher, A. G. (1921). Ibid., **8**, 493.
Wilmoth, C. L. (1941). *J. Bone Jt Surg.*, **23**, 367.

A STUDY IN VISUAL DEFECTS IN YOUNG CHILDREN

BY

P. A. TYSER, M.D., B.S., D.P.H.

Senior Assistant Medical Officer, Area No. 3 (Tottenham), Middlesex County Council

AND

T. W. LETCHWORTH, M.D., F.R.C.S.

Visiting Ophthalmic Surgeon to the Local Education Authority

It is evident from the annual returns of the school health service that amblyopia and strabismus form a large proportion of cases of defective vision. It is the purpose of this paper to describe a simple method whereby gross errors of refraction in children under the age of 5 can be ascertained. We demonstrate that this method is accurate enough to be used on a wide scale, that a large number of children in the age group discussed suffer from gross errors of refraction, and that it is important from a preventive aspect that they should be detected and treated as early as possible in order to achieve good results and prevent deterioration and the development of other conditions. Except in cases of suspected strabismus, hypermetropia under 2D and astigmatism under 0.75D was not corrected; in all other cases correction was made, the conditions being referred to as gross errors of refraction.

We are not concerned here with the finer analysis of the degrees of the errors of refraction and their age distribution; this is of secondary importance to this investigation.

The sight of children is usually examined when they are 7 to 8 years old by the Snellen type test in the course of routine medical inspections. Before this age treatment may be sought only when attention is drawn to the child's eyes by the occurrence of squint or because defective vision is thought to exist. A squint of the ordinary concomitant type associated with hypermetropia is usually manifest between the ages of 18 months and 5 years. At the age of 12 months a child should have acquired the power of fixation. Now, if something prevents that being acquired, nystagmus will occur, but during the next few years the full visual acuity will have to be developed. In cases of a high degree of astigmatism a distinct image is not formed on the retina and full visual acuity cannot be developed. Also with anisometropia the child will not focus the ametropic eye and the same will follow.

It is therefore of great importance that such conditions should be recognized as soon as possible, since by correcting such errors at the earliest opportunity the final result will be much better. After the age of 8, if no correction has been carried out, there will be in these cases a considerable degree of amblyopia, which may be difficult or impossible to overcome. Furthermore, by correcting high degrees of hypermetropia many cases of potential squint may be forestalled. Experience has also shown that if one child in a family has a gross error of refraction it is likely that some of the other children will also have errors requiring correction, and it is the custom of one of us (T.W.L.) to inquire into family histories and to examine all the children of a family in which one child is found to have a gross error of refraction.

The number of cases of amblyopia discovered in school entrants indicates that a far more intensive investigation should be made of the eyesight of the pre-school child. Some special test, easily applied and quickly performed, is required to ascertain those children who are suffering from grosser errors of refraction. One of us (P.A.T.) undertook the retinoscopy examination of all children present in the day nurseries, nursery schools, and classes in the borough.*

Method and Scope of Eye Investigation

Retinoscopy was performed with a plane mirror without using a mydriatic. The children were examined in a darkened room seated on a chair at arm's length. First the movement of the red reflex was noted in vertical and horizontal meridians. Movement against the mirror in any meridian in either eye was noted as "myopia" and referred to one of us (T.W.L.) for further examination. If movement occurred with the mirror then the procedure was repeated using a +2D lens held in turn immediately in front of the child's eyes, movement in any meridian in either eye being noted as "hypermetropia" and referred for further examination. It was found that having the children attend in batches of ten alleviated anxiety and that it was possible to examine 60 children in the course of an hour.

The upper age limit was taken as the age of 5 in January, 1949. Accommodation for 567 children exists in the day nurseries, nursery schools, and classes. Owing to illness and other reasons 87 were absent at the time of the survey and 10 became too emotionally disturbed to be examined. Of the 470 children examined, 10 were excluded from the final results, eight being over the age of 5 and two who were ascertained to have defects failing to keep several appointments for further examination. The results of the remaining 460 examinations are considered here.

Results

Table I shows the analysis by sex and age of the examinations made. The numbers in the age groups are considered too small for further analysis. The 460 children comprised 247 boys and 213 girls, and in each case 19

*We wish to acknowledge our indebtedness to Dr. G. Hamilton Hogben, to the borough education officer, and to the head teachers and matrons of the nursery schools and classes and day nurseries in Tottenham for their invaluable assistance.

C. H. BEST, W. STANLEY HARTROFT, C. C. LUCAS, AND JESSIE H. RIDOUT: LIVER DAMAGE PRODUCED BY FEEDING ALCOHOL OR SUGAR, AND ITS PREVENTION BY CHOLINE

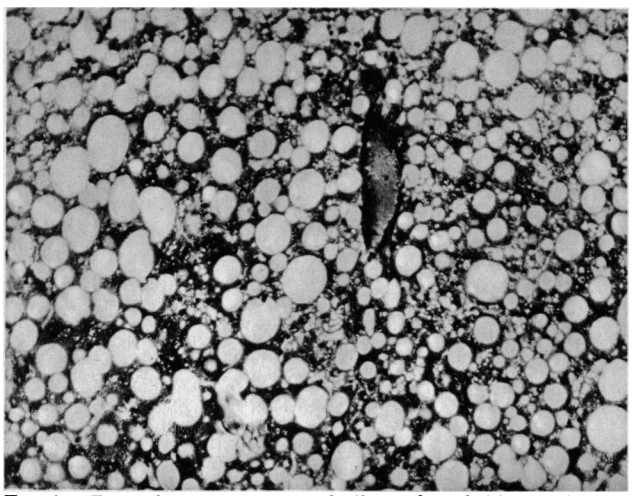

FIG. 1.—Fatty change (++++) in liver of rat fed basal diet and given 15% alcohol in place of drinking-water. Paraffin section; azocarmine, aniline blue, and orange G. (× 80.)

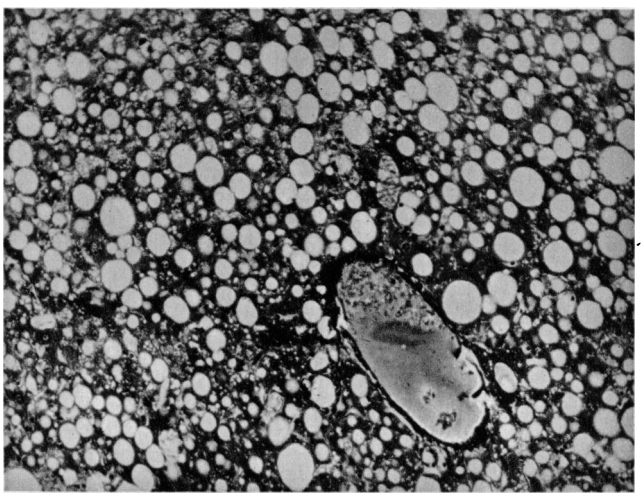

FIG. 2.—Fatty change (++++) in liver of rat fed basal diet and sugar. Paraffin section; stain as in Fig. 1. (× 80.)

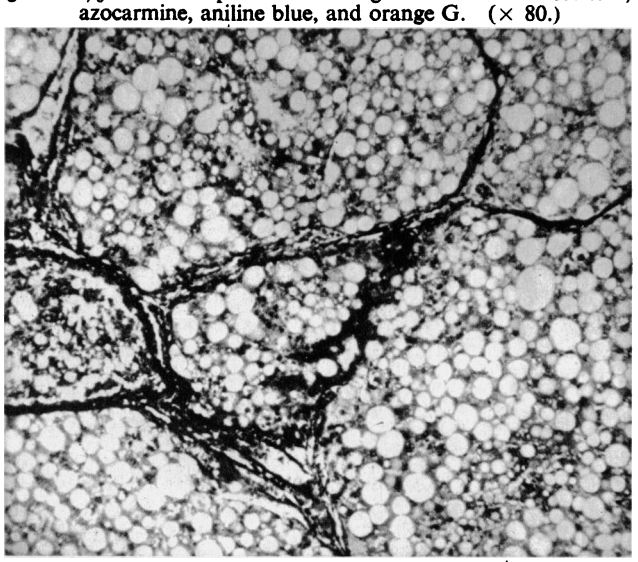

FIG. 3.—Well-defined fibrosis in liver of rat fed basal diet and given 15% alcohol in place of drinking-water. Paraffin section; stain as in Fig. 1. (× 80.)

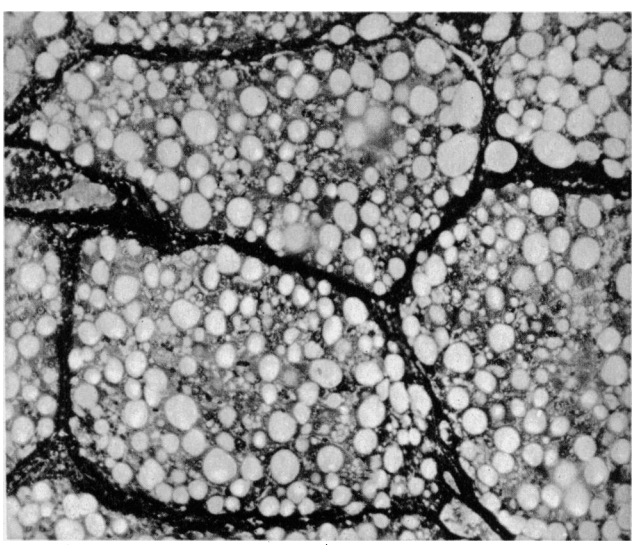

FIG. 4.—Well-defined fibrosis in liver of rat fed basal diet and sugar. Paraffin section; stain as in Fig. 1. (× 80.)

J. F. CURR: SYNOVIAL OSTEOCHONDROMATOSIS

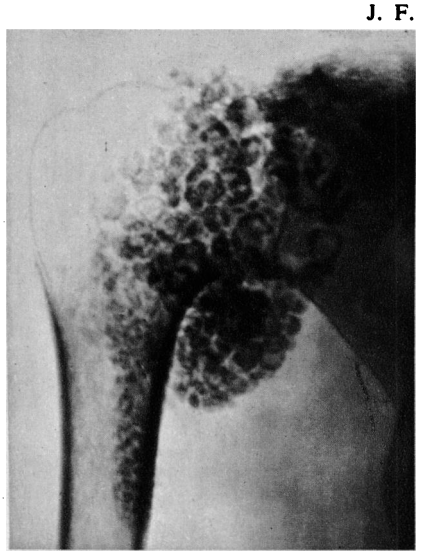

FIG. 1.—Case 1. Right shoulder: large numbers of osteochondromata.

FIG. 2.—Case 1. New bone formation. (× 60.)

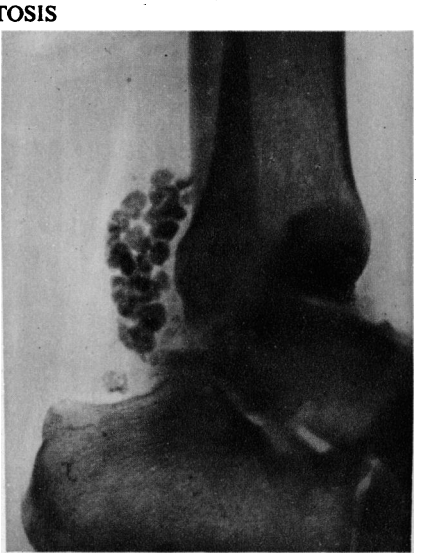

FIG. 3.—Case 2. Left ankle: osteochondromata posterior to joint.