20 to 30 minutes and two to three hours after its administration. Immediately after dipyridamole a pronounced reduction (Fig. 3) in the cumulative number of emboli was found (21.5% of control; $P \leq 0.001$). However, the effect was transient, and injuries inflicted 20 to 30 minutes after dipyridamole showed platelet activity comparable to that seen in the control tests (91% of control; $0.3 > P > 0.2$).

Neither of the two animals given prostaglandin E_1 (25 and 125 $\mu\text{g./kg.}$ body weight) intravenously and tested before, immediately after, and 45, 75, and 120 minutes after its administration showed an inhibitory effect on platelet activity.

DISCUSSION

With the technique used in the present investigation it is possible to study the effects of different materials on different days in the same animal. Each animal acts as its own control, thus overcoming the problem of the individual nature of platelet reactivity. The technique has additional advantages over the methods hitherto available. The animals are not anaesthetized, endothelial injury is precise and reproducible, and the observations are made at high magnification. Furthermore, platelet activity at sites of fresh laser injury at different times is remarkably constant (Arfors *et al.*, 1968).

Dextran 70 was shown to have a pronounced inhibitory effect on platelet activity at sites of endothelial trauma. Indeed, the changes were highly significant even in a single animal. Since four to six hours elapse before the effect of dextran becomes maximal it cannot be ascribed to dilution of platelets after volume expansion or to hyperkinetic blood flow. It is of interest that a similar delay is found in man after dextran infusion before the maximum effect on platelet behaviour as tested in vitro (Bennett *et al.*, 1966; Bygdeman *et al.*, 1966). This suggests the possibility that a time-dependent interaction between dextran and some plasma factor underlies the observed inhibition of platelet activity.

In contrast to dextran, other materials of current interest were found to be disappointing in this preliminary study. Heparin had no significant effect. The effect of dipyridamole was transient; though pronounced suppression of platelet activity was seen immediately after its administration, the activity had returned to control values after 20 to 30 minutes. The transient

effect of dipyridamole on platelet behaviour at sites of injuries inflicted after its administration has already been noted (Emmons *et al.*, 1965). However, there is evidence that platelet activity at sites of injuries inflicted before the administration of dipyridamole is profoundly suppressed (Emmons *et al.*, 1965). The reason for this difference is not clear.

The lack of effect after a single intravenous dose of prostaglandin E_1 given before injury may be accounted for by the rapid inactivation of prostaglandins in the lungs (Bergström and Samuelsson, 1965). Nevertheless, Emmons *et al.* (1967) claim that platelet activity is suppressed in vivo after similar single doses. Further work using single doses and continuous intravenous and intra-arterial infusion of prostaglandin E_1 is clearly necessary.

We are indebted to Dr. D. A. van Dorp, of Unilever Research Laboratories, Vlaardingen, Holland, for supplies of prostaglandin E_1 , and to Mr. J. W. Robertson for technical assistance.

K.-E. ARFORS, PH.D.,

Department of Clinical Physiology, Academic Hospital, Uppsala, Sweden.

H. C. HINT, M.D.,

Department of Experimental Medicine, Pharmacology AB, Uppsala, Sweden.

D. P. DHALL, M.B., CH.B., F.R.C.S.ED.,

N. A. MATHESON, CH.M., F.R.C.S., F.R.C.S.ED.,
Department of Surgery, University of Aberdeen, Scotland.

REFERENCES

- Arfors, K.-E., Dhall, D. P., Engeset, J., Hint, H. C., Matheson, N. A., and Tangen, O. (1968). *Nature (Lond.)*, **218**, 887.
Bennett, P. N., Dhall, D. P., McKenzie, F. N., and Matheson, N. A. (1966). *Lancet*, **2**, 1001.
Bergström, S., and Samuelsson, B. (1965). *Ann. Rev. Biochem.*, **34**, 101.
Bygdeman, S., Eliasson, R., and Gullbring, B. (1966). *Thrombos. Diathes. haemorrh. (Stuttg.)*, **15**, 451.
Emmons, P. R., Harrison, M. J. G., Honour, A. J., and Mitchell, J. R. A. (1965). *Nature (Lond.)*, **208**, 255.
Emmons, P. R., Hampton, J. R., Harrison, M. J. G., Honour, A. J., and Mitchell, J. R. A. (1967). *Brit. med. J.*, **2**, 468.

Medical Memoranda

Uncommon Complication of Anaphylactoid Purpura

Brit. med. J., 1968, **4**, 431-432

Anaphylactoid purpura, the Henoch-Schönlein syndrome, is a common condition in childhood and one in which abdominal pain frequently occurs. This may on occasion be severe enough to mimic an acute surgical abdomen, and an unnecessary laparotomy may be performed. Recently two children suffering from anaphylactoid purpura were admitted to the Mansfield General Hospital, and in the course of this illness both boys developed testicular pain and scrotal swelling clinically simulating acute testicular torsion. In one patient exploration excluded torsion, and in both patients it was assumed that their symptoms were associated with the underlying condition and were not purely unrelated incidents.

CASE 1

A boy aged 5½ years was admitted to the children's ward on 10 February 1968 with symptoms typical of the Henoch-Schönlein syndrome. He had been well until two weeks before admission, when he developed a cough and signs of an upper respiratory tract infection. During the following week he complained of pains in his legs, and on the evening before coming into hospital it was noted that his ankles were swollen and he was unable to walk. His previous health had been good and he had been in hospital only once before, when he had a circumcision in 1967.

On examination there was a characteristic rash on his elbows, buttocks, and legs. Both feet were puffy and his ankles painful, the right more than the left. His temperature orally was 99° F. (37.2° C.) and the E.S.R. was reported as 20 mm. in the first hour. A throat swab was reported subsequently as showing no evidence of haemolytic staphylococcus. Urine examination showed no abnormality. The antistreptolysin O titre was 500 Todd units.

The child was prescribed penicillin and his progress over the next few days was satisfactory. The swelling of his feet gradually sub-

sided and his urine remained free of albumin and red blood cells.

Six days after admission he complained of pain in his scrotum, which on examination was found to be slightly oedematous. No other abnormality was noted, but during the next two hours the pain worsened, the scrotum became very much more swollen, and there was extreme tenderness of the right testis with bluish-red discoloration of the overlying scrotal skin. There was also some tenderness along the spermatic cord. His temperature was normal but the pulse rate was 110.

Clinically it seemed likely he had torsion of the right testis, and exploration was carried out the same evening. At operation the scrotal oedema and discoloration were noted, but there was no evidence of torsion and the body of the testis was normal. There were, however, purpuric spots scattered over the epididymis, similar to those found on the bowel wall in patients with anaphylactoid purpura requiring laparotomy on account of abdominal pain. The scrotal swelling was presumed similar in nature to the swelling that may occur in relation to joints or over the dorsum of the hands and feet in this condition, but no definite cause for the testicular pain could be found. Presumably oedema in or around the nerve endings on the surface of the testis could have been responsible. Prednisolone was given, and by the following day the scrotal oedema and pain were very much less, completely disappearing during the ensuing 72 hours. Despite the use of steroids the boy had an attack of colicky abdominal pain, and his stool 48 hours later showed a positive reaction for occult blood. His subsequent progress, however, was satisfactory, his urine remained clear, and when seen at the follow-up clinic one month after discharge he was well and both testes appeared equal in size and of normal consistency.

CASE 2

Two weeks after the above patient had been discharged from hospital a boy aged 5 years was admitted to the same ward. The history and clinical findings in this patient were again typical of anaphylactoid purpura, with the usual rash and joint pains following an upper respiratory tract infection. The patient had abdominal pain before admission, and four days after admission complained of scrotal pain and was found to have generalized oedema of the scrotum with marked tenderness and pain on palpation of the left testis. The scrotal skin over the left testis was discoloured. The clinical findings were once again similar to those of acute testicular torsion. In view of the progress in the above case it was decided to observe this child, and he was prescribed steroids. By the following day the pain had disappeared, and within four days the swelling had subsided. At subsequent follow-up the boy was well and both testes appeared normal.

Meningomyelitis due to Brucellosis

Brit. med. J., 1968, 4, 432-433

Neurological complications of brucellosis have been recognized for a long time (Tomaselli, 1879). Hughes (1897) was the first to describe a case of meningomyelitis with necropsy findings. Cases of sciatica, brachial neuritis, and meningitis due to brucellosis have been recorded in India (Mathur, 1954, 1955; Pathania *et al.*, 1966). A case of meningomyelitis occurring as a complication of brucellosis is now reported.

CASE REPORT

A 40-year-old wife of an officer was admitted to hospital with a one month history of irregular fever. Diagnosis did not pose a problem, as her husband had been under treatment for brucellosis a few months previously. *Brucella abortus* was grown in culture from the blood, and the agglutination tests were repeatedly positive in significant titre, the highest being 1 in 1,000. She responded

COMMENT

During discussion about these patients a surgeon who was not involved mentioned that on one occasion in the past he had performed an unnecessary operation on a child thought to have torsion of the testis, but in fact no torsion had been found and the child subsequently developed symptoms and signs of the Henoch-Schönlein syndrome. A surgical registrar involved with Case 2 also mentioned having seen this complication of anaphylactoid purpura on a previous occasion. It would appear, therefore, that this is an unusual complication, but probably commoner than the literature would suggest. There is in fact no mention of it in standard paediatric textbooks.

It is obviously desirable to avoid unnecessary operations at any time, and for this reason, if a child presents with symptoms of testicular torsion during the course of an illness, proved or suspected to be anaphylactoid purpura, then observation rather than exploration would appear reasonable. Whether prednisolone helps is doubtful; it did not prevent the first patient from developing typical abdominal pain, but it may possibly be of value in treatment of the oedema.

ADDENDUM.—In July of this year a 5-year-old boy was admitted to Mansfield General Hospital with acute right testicular pain and scrotal swelling. On examination on admission he was noted to have a purpuric rash on his legs and he undoubtedly had anaphylactoid purpura. His scrotal pain was, however, of such severity that the consultant surgeon in charge felt that exploration was necessary to exclude torsion. As in the other cases, there was no sign of torsion at operation. The cord was oedematous and congested and so also was the testis and epididymis. The appendix testis was haemorrhagic, and histology of a small portion of the appendix testis and epididymis showed some haemorrhage with an intense vasculitis, characteristic of anaphylactoid purpura. The child was not given steroids and his subsequent progress was satisfactory. When seen at follow-up clinic he appeared to have a normal right testicle and no evidence of sequelae.

I am grateful to Dr. A. C. Blandy for permission to publish details of Case 2 and to Mr. M. J. Ball for his help with the third patient. I thank the consultants and registrars at Mansfield and District General Hospital for their assistance in the management of these patients.

J. S. FITZSIMMONS, M.B., M.R.C.P.ED., D.C.H.,
Consultant Paediatrician, Nottingham and Mansfield
Clinical Area.

rapidly to treatment with tetracycline and was discharged in six weeks.

She was readmitted two weeks later for persistent vomiting, nausea, and giddiness, and was found to have low-grade pyrexia (99.4° F.; 37.4° C.). No abnormal physical findings were elicited on examination. An E.N.T. examination proved negative. The blood culture was sterile and brucella agglutination test was negative (under 1 in 25). X-ray examination of the chest, the Venereal Disease Research Laboratory test, serum biochemistry, peripheral blood picture, and urine examination were non-contributory. She was found normal by a psychiatrist and was given symptomatic treatment.

The symptoms proved troublesome and resistant. Three weeks after admission she complained of unsteadiness of gait, but did not have any positive neurological signs, and was given physiotherapy. After another four weeks she developed slight weakness of the lower limbs and was noted to drag her left foot while walking. On examination she had a flaccid areflexic paresis of the lower limb and impairment of all forms of cutaneous sensibility below the L3 level in the right lower limb. The C.S.F. contained 132 cells per cu. mm. (predominantly lymphocytes) and 400 mg. of protein per 100 ml. No specific therapy could be started, and she continued to deteriorate under observation. Four weeks after