fifth and sixth ribs, which showed callus formation. The wound was well healed and firm, and did not show any paradoxical movements with respiration. A radiograph of the chest (Fig. 5) showed a clear lung field, but slight elevation of the diaphragm owing to adhesions. There was no pleural thickening adjacent to the wound. The physical findings were essentially normal over the whole chest and no abnormalities in the heart could be detected.

An electrocardiogram was taken, and particular attention paid to the leads over the tantalum implant. All tracings showed no abnormalities of the heart or pericardium and there were no changes attributable to the metal implant.

Summary and Conclusions

A case is presented in which tantalum mesh was used to repair a large gunshot wound defect of the chest.

Tantalum mesh is eminently suitable for use in potentially infected chest wounds to effect closure, and, provided supportive antibiotic therapy is employed, it is possible to obtain primary closure.

Tantalum mesh has no demonstrable effects on the electrical charges or their transmission from the heart as recorded by electrocardiography.

I would like to thank Dr. Henry Lee for his co-operation and permission to publish this report, and Miss Dot Brown for the drawing.

BIBLIOGRAPHY

- Burke, G. L. (1940). *Canad. med. Ass. J.*, **43**, 125.
 Douglas, D. M. (1948). *Lancet*, **1**, 936.
 Fulcher, O. H. (1943). *J. Amer. med. Ass.*, **121**, 931.
 Gardner, W. J. (1945). *Surg. Gynec. Obstet.*, **80**, 303.
 Jefferson, N. C., and Dailey, U. G. (1948). *Amer. J. Surg.*, **75**, 575.
 Koontz, A. R. (1948). *Ann. Surg.*, **127**, 1079.
 Pudenz, R. H. (1943). *J. Amer. med. Ass.*, **121**, 478.
 Throckmorton, T. D. (1948). *Surgery*, **23**, 32.

THROMBOCYTOPENIC PURPURA COMPLICATING INFECTIOUS MONONUCLEOSIS

BY

R. FINLAYSON, M.B.

Registrar, Department of Pathology, St. Bartholomew's Hospital, London

The occurrence of thrombocytopenic purpura with infectious mononucleosis is rare. Tidy (1934) stated that he had found only five recorded cases of infectious mononucleosis complicated by purpura; in one case the thrombocytes were lowered, and in the others the platelets were not estimated. More recently Angle and Alt (1950), in addition to reporting a case of thrombocytopenic purpura complicating infectious mononucleosis, reviewed the literature on this subject. They found only six detailed reports of a definite association between thrombocytopenic purpura and infectious mononucleosis, and twelve other cases in which the two conditions were possibly associated. This series included not only those cases in which the thrombocytopenic purpura paralleled the attack of glandular fever, but also two case reports in which a pre-existing haemorrhagic diathesis was exacerbated by an attack of infectious mononucleosis.

When glandular fever is associated with thrombocytopenic purpura the disease is often first diagnosed erro-

neously as acute leukaemia, the correct diagnosis being made either after a belated study of the heterophil antibody titre of the patient's serum or in retrospect, when the patient has recovered from the illness. A report of a case of infectious mononucleosis complicated by thrombocytopenic purpura may therefore be of interest.

Case Report

A 28-year-old man was admitted to hospital on November 24, 1950, complaining of "blood spots" in his mouth and small dark spots on his body. He had been in good health until one week previously, when he developed a headache and generalized aches and pains. Two days before admission he noticed some "blood spots" on his lips and in his mouth, and that his sputum was streaked with blood. The following day he observed that the glands in his groins were swollen, and on the morning of the day of admission he cut himself repeatedly while shaving, the bleeding from these multiple cuts being difficult to stop. About the same time he noticed numerous small dark spots scattered over his body.

There was no recent history of a sore throat or of other haemorrhagic symptoms, such as epistaxis, haematuria, or intestinal bleeding. There was no previous or family history of haemorrhagic or allergic conditions.

On admission his temperature was 98.2° F. (36.8° C.), pulse rate 90, and respirations 15 a minute. There was no jaundice; the conjunctival mucosae were of a good colour and the ocular fundi were normal. The fauces were injected, but showed neither exudate nor membrane. Numerous purpuric spots were seen on the lips and on the inside of the cheeks; similar lesions were present on the lateral margins of the tongue, and a large haemorrhagic bleb was seen at the junction of the hard and soft palate. The trunk and limbs were blemished by a widespread petechial rash, the legs being affected the most. The tonsillar, cervical, supraclavicular, axillary, inguinal, and femoral lymph nodes were either slightly or moderately enlarged. The lymph nodes were discrete and, with the exception of the right tonsillar node, were neither tender nor painful. The spleen was enlarged, extending two fingerbreadths below the left subcostal margin; it was soft in consistence and not tender to palpation. The liver was not enlarged. The heart, lungs, and central nervous system were normal. The blood pressure was 108/60 mm. Hg. The urine and stools showed no macroscopic abnormality.

Investigations.—Examination of the blood on admission showed: Hb, 90% (Haldane), or 12.4 g. per 100 ml.; red cells, 5,100,000 per c.mm.; colour index, 0.88; white cells, 15,000 per c.mm.; platelets (Lempert's method), 10,000 per c.mm. A differential white-cell count showed: neutrophil polymorphs, 2,175 per c.mm. (14.5%); lymphocytes, 5,400 per c.mm. (36%); monocytes, 150 per c.mm. (1%); eosinophils, 75 per c.mm. (0.5%); basophils, 75 per c.mm. (0.5%); and atypical mononuclears, 7,125 per c.mm. (47.5%). The atypical mononuclears were oxidase-negative and showed the typical features of "glandular fever" cells. The stained blood film confirmed the marked thrombocytopenia. The bleeding-time (Duke) was longer than 10 minutes. The clotting-time (Dale and Laidlaw) was two minutes. The erythrocyte sedimentation rate (Westergren) was 6 mm. in one hour. The heterophil antibody test (Davidsohn) was positive in a serum dilution of 1:1,024. The bone marrow was not examined. The diagnosis was infectious mononucleosis complicated by thrombocytopenic purpura.

Course and Treatment

During the first two days in hospital the patient's condition showed little change. He was slightly febrile—maximum temperature 99.4° F. (37.4° C.)—on November 25, and, although his temperature was normal on the following day, the fever returned on November 27 (three days after admission). On that day "aureomycin" therapy was started; 750 mg. was given six-hourly to a total dose of

12 g. Coincident with chemotherapy, his temperature returned to normal. On November 27 he developed a sore throat, which persisted for three days. A blood count showed: white cells, 13,000 per c.mm.; platelets, 8,000 per c.mm. That evening he had an epistaxis after blowing his nose. Dry "stypven" and adrenaline packs failed to stop the bleeding; five hours after the onset of the epistaxis a transfusion of 1 pint (570 ml.) of fresh blood was given. On the next day the bleeding ceased, although a platelet count showed no significant alteration. On November 29 a further transfusion of 1 pint (570 ml.) of fresh blood was given. A blood count (before transfusion) showed: Hb, 92% (Haldane), or 12.7 g. per 100 ml., and 4,000 platelets per c.mm. Microscopical examination of the urine revealed a slight excess of red cells. This was the only occasion on which any urinary abnormality was detected.

December 2 (Eight Days After Admission).—Although epistaxis had not recurred, the purpuric lesions and the glandular enlargement persisted, and the sputum was still blood-stained. The spleen was no longer palpable. A blood count showed Hb, 96% (Haldane), or 13.2 g. per 100 ml.; white cells, 9,050 per c.mm. (including 23% atypical mononuclear cells); platelets, 7,000 per c.mm. The heterophil antibody titre was 1:996 (presumptive test), 1:996 (guinea-pig absorption), and 1:28 (beef-cell absorption).

December 12 (18 Days After Admission).—The lymph nodes were now of normal size. Although the petechiae had almost disappeared and bleeding from the respiratory tract had ceased, a blood count showed only 3,000 platelets per c.mm. The bleeding-time had shortened to six minutes. A tourniquet test was strongly positive. On the following day a few fresh petechiae appeared on the lips and buccal mucosa. These were the last haemorrhagic manifestations to develop during the course of the illness, although the thrombocytes showed little numerical change until December 27 (33 days after admission), when the platelets numbered 30,000 per c.mm. By that date the patient was symptom-free and feeling well.

January 2, 1951 (39 Days After Admission).—The patient was discharged from hospital. A blood count showed a persistence of the thrombocytopenia, although the platelets had increased to 66,000 per c.mm. The total and differential white blood cell counts and the bleeding-time were normal; a tourniquet test was negative.

Follow-up.—A blood count on January 16 showed 164,000 platelets per c.mm., and on January 31 (70 days after the onset of purpura) 215,000 platelets per c.mm. When last examined on September 7, 1951, the patient was in excellent health, his blood picture was normal, and he had had no recurrence of the purpura.

Comment

Laboratory-proved cases of infectious mononucleosis complicated by thrombocytopenic purpura, such as the one reported, are rare. I have found only eight similar cases recorded in the literature (Magner and Brooks, 1942; Lloyd, 1944; Read and Helwig, 1945; Goldbloom and Denton, 1948; Angle and Alt, 1948, 1950; Kutzer and Allen, 1950, two cases; Wallerstein and Madison, 1950). In a further 13 cases a similar diagnosis is probable, although the clinical and haematological evidence is less convincing.

From a study of these 21 case records it would appear that both the purpura and the thrombocytopenia developed during the early stages of the attack of glandular fever. In addition to purpura, a high proportion of the cases showed other haemorrhagic manifestations, most commonly haematuria and/or epistaxis. The thrombocytopenia persisted from one to ten weeks, with an average duration of slightly less than five weeks.

Haemorrhagic symptoms persisted for a considerably shorter time, with an average duration of slightly less than two weeks. In at least nine cases recovery occurred without any active form of treatment. In five cases blood transfusions were given, and, although such therapy may have temporarily decreased the tendency to bleed and the bleeding-time, it did not ameliorate the thrombocytopenia. None of the cases gave a previous history of a haemorrhagic tendency; all but one recovered and none recurred. In the fatal case reported by Smith and Custer (1946) death was caused by splenic rupture.

I wish to thank Dr. E. F. Scowen for permission to study this case and Dr. H. F. Brewer for his advice.

REFERENCES

- Angle, R. M., and Alt, H. L. (1948). *J. Lab. clin. Med.*, **33**, 1652.
 — (1950). *Blood*, **5**, 449.
 Goldbloom, A., and Denton, R. (1948). *Canad. med. Ass. J.*, **58**, 189.
 Kutzer, M., and Allen, E. G. (1950). *N.Y. St. J. Med.*, **50**, 1131.
 Lloyd, P. C. (1944). *Amer. J. med. Sci.*, **207**, 620.
 Magner, W., and Brooks, E. F. (1942). *Canad. med. Ass. J.*, **47**, 35.
 Read, J. T., and Helwig, F. C. (1945). *Arch. intern. Med.*, **75**, 376.
 Smith, E. B., and Custer, R. P. (1946). *Blood*, **1**, 317.
 Tidy, H. L. (1934). *Lancet*, **2**, 180.
 Wallerstein, R. S., and Madison, L. (1950). *Amer. Practit., Phila.*, **1**, 624.

PRODUCTION OF A BLOCKING ANTI-D ANTIBODY BY INJECTION OF D^u RED CELLS

BY

JACQUES RUFFIÉ

AND

M. CARRIÈRE, M.D.

(From the Haematological Laboratory, Medico-Legal Institute, Faculty of Medicine, and the Laboratory of the Regional Blood Transfusion Centre, Toulouse)

In 1947 J. J. van Loghem reported that on injecting red cells r'r' and r'r into a donor of group O rr with a view to obtaining anti-C antibody, there had appeared in the serum of the recipient not only anti-C but also blocking anti-D. Race (1947) gave the explanation of this. He proved that the two donors had in fact the D^u antigen on their red cells, and that this antigen was responsible for the appearance of the anti-D antibody.

We have since observed the same result in a series of immunizations of normal male volunteers which were planned with the intention of preparing Rh subgrouping antisera. One of these series of recipients comprised four individuals—Rum., O rr; Lat., A rr; Mas., O rr; and Cusc., B rr—each of whom received twice weekly an injection of 1 to 2 ml. of whole blood from a donor of O group who was thought to be r'r. In fact, this person proved to have a weak D^u antigen in his red cells. Three out of these four sensitized persons gave a reaction and produced an anti-C antibody + anti-D antibody which from the beginning was of a blocking type. A specific D^u antibody did not occur.

The donor Cusc. reacted at the 13th injection, Rum. at the 22nd injection, and Mas. at the 36th injection. The table summarizes our results.