astinitis and it is felt that in this case it was of great value.

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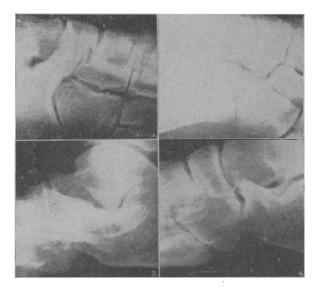
CALCANEO-SCAPHOID BAR

By Major R. K. Magee, R.C.A.M.C. and Major R. A. Benson, R.C.A.M.C.

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The patient was a thirty-seven year old male who had had no foot complaints as carpenter and salesman in civilian life, nor on 20-mile route marches in the army. Only once after marching 29 miles in 24 hours had he had pain and swelling over the extensor tendons of the right ankle lasting three or four days. He was otherwise well until eight weeks prior to our seeing him he sustained a kick over the instep of the right foot while playing soccer. It was better in three or four days but he had some slight residual pain on long patrols, and as it was not improving he came to be examined.

On examination of the right foot there was slight prominence of the medial malleolus and immediately below in the region of the talus. There was no edema or swelling. Dorsal and plantar flexion appeared normal. Abduction and adduction were limited to about half their normal range, and eversion and inversion were nil, but there was no pain on forced movements



1.--Oblique view showing calcaneo-navicular bar. Fig. 2.—Normal foot. Figs. 3 and 4.—Lateral views of same.

There was one-half and no local tenderness. inch atrophy of the right calf muscles. Examination of both feet was otherwise normal.

The diagnosis, unsuspected clinically as this was the first case we had seen, is evident when the x-rays are compared with those of a normal foot. In the oblique x-ray (Fig. 1) the solid bar of bone uniting the antero-internal angle of the calcaneus with the navicular is evident where normal joint lines are seen in the normal foot x-ray (Fig. 2). In the lateral x-ray (Fig. 3) the same bar may be seen though it is easily overlooked unless compared with a normal (Fig. 4).

The condition is a congenital ossification in mesenchyme which would ordinarily form the lateral part of the short plantar ligament. The x-rays showed no other bony injury and the condition responded to rest and he returned to full duty and will have no obvious disability.

A second example of this condition came to the attention of one of us (R.A.B.) three months after the above-described case had appeared. He was a 27-year old, Category A soldier who complained of stiffness in his left ankle region. The diagnosis was not suggested by the examination but x-ray examination revealed a calcaneonavicular bar almost identical in appearance with the one described above.

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HYPOPLASTIC ANÆMIA TREATED WITH TRANSFUSIONS AND FOLIC ACID FRACTION*

By G. B. Peat, M.D. and Arnold Branch, M.D.

Saint John, N.B.

In 1937 Peat¹ reported a case of aplastic anæmia in a nine-year old girl, whom he had treated for six years with multiple transfusions. The present report brings the case up to date.

The patient is now 18 years old, rather small for her age and with a definite brownish pigmentation of the skin, which has considerably lessened in recent months. She is normal in her activities, i.e., attends school and parties. She has not yet menstruated. Her blood group is international O, Rh 1 and 2.

Small transfusions of 125 c.c. were admin-

^{*} From the General Hospital, Saint John, N.B. and the Provincial Bureau of Laboratories.

istered weekly for a long time, with infrequent variations up to 500 c.c. From August, 1940, she was given 250 c.c. of blood weekly with the hæmoglobin staying around 40 to 52%, and a red blood count varying from 2,000,000 to 3,500,000, and a white count of from 1,800 to 3,200.

In May, 1942, she had a severe infection, apparently the "flu", with a bad attack of tonsillitis. In the late part of 1942, her skin had a peculiar darkness almost as if a solution of ink had been washed under it. In October, 1943, eschatin, 1 c.c. was given for a time hypodermically with each transfusion to see if it would make her more energetic and if there would be any effect on the skin pigmentation, with no definite results. At this time the number of transfusions was reduced, with rapid deterioration in the blood picture.

Early in 1944, she began developing some indolent ulcers on the head and shoulders. A number of the ordinary remedies were tried, with no effect, but they healed with tyrothricin applications. She also had a persistent hoarseness. Penicillin became available in July of that year and 100,000 units produced a dramatic effect on the throat condition. She has since had two other courses of penicillin (200,000 units each). This also seemed to have a curative effect on some violent headaches that she was apt to get and which did not seem to be entirely due to a sinus condition.

In July, 1944, she was started on folic acid fraction of liver² kindly supplied by the Lederle Laboratories, and has been on this product since. For the first two months 16 capsules $(7\frac{1}{2})$ gr. size) were given daily and since then 20 capsules a day. There was no improvement in the blood picture but the level was retained with less frequent transfusions. In February, 1945, she was changed to 6 capsules a day of a concentrated folic acid fraction with apparent deterioration in the blood picture, so she was again put on the original fraction first used. The blood picture continued on a level with few transfusions. In June and July there was a sudden upgrade in the percentage of hæmoglobin and in the red cell count, not accompanied by a reticulocytosis, but the leucocytes continued low, around 3,000 per cubic millimetre. About this time some copper was added to her iron and ammonium citrate capsules and she also had another 200,000 units of penicillin for headache of undetermined origin.

Of technical interest is the continued use of one vein for all transfusions. Preparations were made for intra-medullary transfusions, but this has not been necessary. A further point is the repeated use of the same donors. One donor has given over 30 transfusions and the others 15, 10, and 5 or less. Reactions have only occurred on four occasions, 3 of which were from the same donor and consisted of a severe general pruritus. This all happened in the early days, when she was getting larger amounts (400 to 500 c.c.).

It is difficult to evaluate the part that each method has played in this case. The clinician (G.B.P.) is convinced that the long continued transfusions have kept her going, and that penicillin has controlled any infection, and the combined use of folic acid fraction and iron and copper has stimulated hæmatopoiesis. There is also the possibility that destruction of red cells had been decreased.

TABLE I.

TABLE SHOWING NUMBER AND VOLUME OF
TRANSFUSIONS PER YEAR

Year	Number	$Volume\ c.c.$
1932–36	87	11,250
1937	46	5,750
1938	43	5,500
1939	43	6,125
1940	33	6,300
1941	33	8,250
1942	30	8,350
1943	24	5,600
1944	13	3,050
1945	3	775
1932-45	355*	60,950 c.c

^{*}Exclusive of those given elsewhere.

Table I shows the number of transfusions she has received in 13 years with a total volume of 61 litres. Group O donors, compatible in both the major and minor reactions on crossmatching are employed irrespective of whether they are Rh positive or negative. The usual amount per transfusion for several years has been 250 c.c.

The blood picture. — Since the initiation of folic acid fraction in 1944 the reticulocyte count has not varied materially, usually running between 1 and 2%. Very occasionally a normoblast has been seen. There is usually irregularity in the size of the cells, polychromatophilia and sometimes stippling. The colour index is about 1. Differential count of the white blood cells is about 45% polymorphonu-

TABLE II.

Hæmatological Studies on Blood Taken at 10.30 a.m., August 7, 1945, Three Months After the Last Transfusion

Capillary Blood:

Hæmoglobin—8.84 gm. or 61%
Red blood cells—2,660,000
White blood cells—2,400
Platelets—126,000
Reticulocyte count—1.3%
Clotting time (capillary tube)—7 minutes

Smear:

Red cells large, and well stained. Some poikilocytes and small forms.

Occasional polychromatophilia and stippling.

Differential of white cells.

Neutrophiles, segmented—19%
Neutrophiles, unsegmented—15%

Eosinophiles—3%
Lymphocytes—61%
Diameter of red blood cells. Average 8 microns.

Venous blood in oxalate:

Hæmatocrit—32.7%
Red cell fragility—
Complete hæmolysis—0.3-0.35% NcCl
Beginning hæmolysis—0.4-0.45% NaCl
Prothrombin time—(Smith bedside method with Abbott

Thromboplastin) 40 seconds.
Clot retraction time—Beginning in less than 1 hour.
Sedimentation rate—30.5 in 1 hour (Cutler method)
Van den Bergh—negative

Van den Bergh—negative Icteric index—6.3

Wet preparation of whole blood shows many rouleaux cells are biconcave, few poikilocytes.

Calculations:

Colour index—1.13
Volume index—1.45
Mean corpuscular volume—121 cubic microns.
Mean corpuscular hæmoglobin—32.7 micro-milligrams.
Mean corpuscular hæmoglobin concentration—27.3%.

Van Slyke Gravity Method:

Whole blood specific gravity—1.047 Plasma specific gravity—1.0265 Plasma protein—6.7% Hæmatocrit—29% Hæmoglobin—9.9 gm.

clear leucocytes, of which 5% are segmented, with 50% lymphocytes, 2% eosinophiles and 2% monocytes. Absolute numbers of neutrophiles vary from 1,200 to 1,900 and of lymphocytes from 1,500 to 1,800.

No bone marrow examination has been made since that previously reported which was diagnosed as hypoplastic anæmia. Furthermore, it did not appear that detailed studies on the blood would be capable of interpretation while she was having weekly transfusions.

SUMMARY

A case of long standing hypoplastic anemia is presented to illustrate the practicability of keeping these cases going by frequent small blood transfusions. It would also appear that folic acid fraction has some effect in reducing the number of transfusions necessary in treating

these cases. Whether this fraction alone is responsible for the improvement cannot be determined as other factors including age, penicillin and copper have been additive.

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SEVERE, DELAYED PROCAINE POISONING

By Arthur Vogelsang, B.A., M.D.

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Miss A.R., a 19 year old, well nourished girl, had a tonsillectomy under local procaine anæsthesia at 9 a.m. on March 9, 1946. The preoperative medication was morphine sulphate gr. 1/6, atropine sulphate gr. 1/100 and sodium pentobarbital gr. 11/2 at 7.30 a.m. The operation was quite uneventful and scarcely any blood was lost. At 9 a.m. she was returned to the ward and her condition was reported as good when the surgeon visited her. At 2 p.m. she began to complain of pain in the epigastrium and a hot water bottle was applied. She became more restless and still complained of pain in the epigastrium and throughout the abdomen. Morphine gr. 1/6 was given, followed by an aspirin phenacetin compound with $\frac{1}{2}$ gr. codeine. At 8.30 p.m. she started to vomit and still complained of the same pain in the abdomen. Codeine gr. ½ was given.

I was first called to see her at 11 p.m. At that time she was obviously in marked shock, with a rapid, thready pulse (160), temperature 96.0° F., bloodless, slate-coloured lips, and no blood pressure was obtainable. She was thrashing about the bed, vomiting spasmodically, and retching almost continuously. Her feeble movements were inco-ordinated and she was disoriented and incoherent. Immediately treatment for shock was instituted, including codeine, adrenalin, and caffeine sodium benzoate parenterally, as well as 1,000 c.c. 5\% glucose in saline, 500 c.c. of blood plasma intravenously, and sodium luminal intramuscularly. An attempt to administer brandy by mouth was unsuccessful on account of the vomiting.

Two hours later (1 a.m.) her colour was slightly better and her systolic blood pressure by palpation was 70 mm. Her pulse was 120, stronger and of more even quality. She still had "a sinking feeling" and her abdomen was