degree of severity these lesions may attain. The two main problems of balantidiasis remain to be solved: first, its mode of spread, and, secondly, its treatment.

Opinion is divided on whether or not the pig plays any part in the spread of human balantidiasis. Walker (1913) is of opinion that pigs serve as a reservoir for human infection, and Craig and Faust (1940) state that 25% of cases give a history of contact with pigs. Neither, however, produces evidence of the transfer of the infection from a pig to man. The evidence to support the theory of infection being contracted from pigs is therefore only presumptive. Ostroumov (1947), after studies of abattoir workers, states that he is convinced that the pig plays no part in the epidemiology of human balantidiasis. Ferri (1943), from observations on a group of children in a mental hospital in Tomsk, concluded that the infection was passed from child to child. In my series of cases it is certain that pigs played no part in the transmission of the disease. It may therefore be concluded that balantidiasis may be contracted from pigs, but it is certain that the disease can be transmitted without the pig acting as an intermediate host.

In the group of children studied by Ferri no cysts were seen. Neither in my series were any cysts seen in numerous stool examinations in each of 87 cases. It therefore seems a logical conclusion that transmission can occur in the active ciliate state, although the generally accepted theory is that infection occurs by swallowing cysts.

The first experiment to produce human balantidiasis was carried out in 1896. Andrews (1930) cites Cassagrandi and Barnagallo as having in that year fed balantidia from man and pigs to human volunteers, but they failed to infect them. Ziemann, in 1925, again cited by Andrews, fed balantidia from chimpanzees to men, but also failed to infect them. In view of the number of different species of *Balantidium* described since these early experiments, however, we cannot be sure that it was *Balantidium coli* which was actually used. Knowles and Das Gupta (1934) fed trophozoites and cysts of balantidia from the monkey *Macacus rhesus* to two men. Neither developed balantidiasis. However, it is now known that the rhesus monkey is infected by *Balantidium simile*, which is not pathogenic to man, and balantidiasis would not therefore develop. Young (1950) fed cysts and vegetative *Balantidium coli* in gelatin capsules to two volunteers. Over a period of 10 years neither developed diarrhoea or passed cysts or trophozoites in the stools. The ciliates given were obtained from a human case of balantidiasis. No intermediate host was found in my series, and the mode of transmission of balantidiasis must remain open to debate.

Sweeney (1929) carried out experiments on guinea-pigs with balantidiasis in an endeavour to find a suitable drug for treatment. Of many substances tried, she found the arsenicals gave the best results, and more particularly acetarsol, tryparsamide, and "parosan." These drugs do not seem to have been used much in the treatment of human balantidiasis. Only one reference to the use of acetarsol was found (Atiles, 1943). Results in my series would indicate acetarsol to be of definite value. No success was obtained with mercury biniodide, as used by Shun-Shin (1947) and Pramanik (1947).

Summary and Conclusions

A series of 87 cases of balantidiasis among the workers of a large industrial concern in South Persia is described. The mode of infection is assessed, the method of the investigation outlined, and the result of treatment stated.

It is stated that pigs played no part in transmission of the disease, that man-to-man transmission is strongly suggested, and that acetarsol is an effective drug in the treatment of balantidiasis.

I should like to thank Dr. S. D. McClean, chief medical officer, Anglo-Iranian Oil Company, for permission to publish this paper, and Dr. I. S. Stewart, pathologist, Anglo-Iranian Oil Company, for much helpful criticism in its preparation.

BIBLIOGRAPHY

- Andrews, J. (1930). *Trans. 8th Cong. Far East Ass. trop. Med.*, 2, 194.
Atiles, A. D. (1943). *Puerto Rico J. publ. Hlth*, 18, 287.
Craig, C. F., and Faust, E. C. (1940). *Clinical Parasitology*, p. 217, Philadelphia.
Ferri, L. V. (1943). *Trop. Dis. Bull.*, 40, 459.
Knowles, R., and Das Gupta, B. M. (1934). *Indian med. Gaz.*, 69, 390.
Koppisch, E., and Wilking, V. N. (1947). *Puerto Rico J. publ. Hlth*, 23, 185.
Ostroumov, V. G. (1947). *Trop. Dis. Bull.*, 44, 904.
Pramanik, A. (1947). *British Medical Journal*, 2, 794.
Selimkhanov, K. A. (1947). *Trop. Dis. Bull.*, 44, 91.
Sewart, I. S. (1949). *British Medical Journal*, 1, 662.
Shun-Shin, M. (1947). *Ibid.*, 2, 417.
Strong, R. P. (1942). In *Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases*, 6th ed., vol. 1, p. 445. Philadelphia.
Stshensnovitsk, V. (1943). *Trop. Dis. Bull.*, 40, 313.
Sweeney, M. A. (1929). *Amer. J. Hyg.*, 9, 544.
Walker, E. L. (1913). *Philipp. J. Sci.*, B, 30, 333.
Young, M. D. (1950). *Amer. J. trop. Med.*, 30, 71.

EXCHANGE TRANSFUSION IN SEVERE ANAEMIA

BY

T. WARD, M.B., B.S.

(From the Royal Victoria Infirmary, Newcastle-upon-Tyne)

The object of this paper is to describe a technique for exchange transfusion in patients with severe anaemia. The technique has not, to my knowledge, been reported previously.

In exchange transfusion the patient's blood is exchanged for similar volumes of whole blood or packed cells. In the present method packed cells are used, and the general principle of combined exsanguination and transfusion is the same as that employed in the treatment of erythroblastosis and chronic leukaemia.

In severe prolonged anaemia, however, there is little cardiac reserve, and some cases exhibit cardiac failure with congestion. For such cases Victor Riddell (1939) suggested preliminary venesection before transfusion, but with the technique here described exsanguination and transfusion are done simultaneously.

Technique

The components of the transfusion apparatus are: (1) Packed cells; (2) giving set; (3) funnel calibrated in ml. for measuring volume of transfusion blood; (4) two lengths of 2-mm. bore polythene tubing; (5) cannula to unite giving set and polythene tubing; (6) cutting-down set; (7) measuring cylinder (1,000 ml.) calibrated in ml.; (8) heparin (5,000 units per ml.); (9) 50-ml. syringe with 3.8% sodium citrate or heparinized saline (5,000 units per litre normal saline); (10) sterile needle (for use in removing clots from tip of venous escape); (11) acriflavine-cetrimide skin preparation; (12) tourniquet. (Sterilization of polythene tubing: Farquhar and Lewis, *Lancet*, 1948, 2, 244.)

The apparatus for the transfusion is set up as shown in Fig. 1. Packed cells are run into the measuring funnel, and a little is allowed to run through the apparatus to clear it of air bubbles.

Cut-down.—A vein in the antecubital fossa is incised immediately distal to a venous valve. A transverse skin incision is made across the vein, the vein is mobilized, ligatures are passed beneath the vein, and the ends are brought out through the skin. A longitudinal incision is made into the vein. When the lumen is opened the haemorrhage is stopped by passing a haemostat beneath the vein. (The tension in the vein causes the walls to come together.)

FIG. 1.—Simple exchange transfusion.

The "polythene" tubing (2-mm. bore) is then inserted into the vein and pushed proximally for several inches. The transfusion is started at a rate of 10–15 drops a minute. The proximal ligature is tied.

Venous Escape.—A second polythene tube is washed through with 1 ml. of heparin (5,000 units) and inserted into the vein through the same incision in its wall. It is pressed distally down the vein for a few inches. Should a venous valve be encountered, it is better to withdraw the tubing a short distance, as a freer flow of blood results. The venous-escape tubing is secured with the distal ligature. Skin sutures are inserted and tied. The length of venous-escape tubing left outside the vein should be cut to about 6 or 8 in. (15 or 20 cm.). If it is longer, clotting within the tube is troublesome. The venous-escape tube is led into a measuring cylinder.

Control of Transfusion

The rate of transfusion depends on the rate of venous escape, both being measured volumetrically. The two rates should in most cases be equal. In the few cases of congestive failure needing venesection the venous escape may be allowed to exceed the volume transfused.

Venous escape should be rapid. Any tendency to slowing can be obviated by the use of a tourniquet to increase the venous pressure; palpation of the radial pulse will ensure that the tourniquet does not increase the systolic pressure. Once the rate of venous escape has been restored, the tourniquet should be released, as its prolonged application is painful.

Transfusion through polythene tubing is rarely troublesome, but clotting in the venous escape may occur. Clots form at the free end, and can be removed with a needle. A tourniquet applied distal to the proximal end of the transfusion tubing (in the vein) will increase the venous pressure and may expel such a clot. Otherwise, moving the tubing in the vein or syringing with heparinized saline (5,000 units per litre) may succeed. It is considered inadvisable to give systemic heparin, as the tendency to haemorrhage (already great in such patients) may be increased. Dicoumarol is, of course, contraindicated.

The physician in charge of the transfusion must remain at the bedside or within calling distance of the patient throughout the transfusion. A special nurse is needed for whole-time duty with the patient.

During transfusion the following are observed: (1) filling of neck veins; (2) half-hourly pulse; (3) blood pressure—hourly readings suffice; (4) drip rate and venous escape (drops/minute), half-hourly (used only as a rough check on exchange rates); (5) drip volume/unit time (half-hourly); (6) venous-escape volume/unit time (half-hourly); and (7) clinical observations.

The value of direct measurements of venous pressure during exchange transfusion has yet to be ascertained.

The following are illustrative cases. Case 1 particularly shows the need for simultaneous exsanguination.

Case 1

A man aged 59 suffering from pernicious anaemia came under our care on May 4, 1949. On admission he was very pale and very weak; he was unwilling to move, and was too ill to give a history. A blood count showed: haemoglobin, 3.4 g./100 ml. (23%); red cells, 970,000; colour index, 1.18; reticulocytes, 0.8.

FIG. 2.—Case 1. Exchange transfusion.

Replacement transfusion was started at 11.30 a.m. on May 5, using 1 mm. polythene tubing. The venous escape was troublesome. Fig. 2 shows the wide fluctuations in venous-escape rate. Nevertheless, after each reading the venous-escape rate and transfusion rates (in drops a minute) were made equal.

From the start of the transfusion until about 9 p.m. there was a progressive improvement. He lost his complete apathy, took an interest in things about him, and began to ask questions spontaneously and even to smile at times.

At 9 p.m. the venous escape blocked completely, and a slow direct transfusion was allowed to continue. There was no further improvement in his condition. Instead there was a statistically significant rise in pulse rate coincident with the blocking of the venous escape. The mean pulse rate before 9 p.m. was 74 (standard deviation 2.03); after 9 p.m. it was 86 (standard deviation 4.25). On May 6 the Hb was 6.8 g./100 ml. (46%) and red blood cells 2,480,000. Next day the Hb was 5.3 g./100 ml. (36%) and red blood cells 1,850,000. The total volume transfused was 900 ml.; the total venous escape was 630 ml.; and the duration of transfusion was 13 hours.

Case 2

A man aged 70 had suffered from symptoms of idiopathic steatorrhoea for three years, when in 1947 he developed a reticulum-cell sarcoma of the palate. Repeated recurrences of metastases in mediastinum and skin were treated successfully by radiotherapy. In July, 1949, he was admitted once more. He was very pale and weak, and had purpuric spots on his arms and legs. There was an apical systolic murmur; the pulse was feeble at 90; and the B.P. was 90/80. On July 6 a blood count showed: Hb, 3.99 g./100 ml. (27%); red cells, 1,430,000; colour index 0.91; platelets 20,000 per c.mm.

An exchange transfusion was carried out on July 7. This was the last of the present series of replacement transfusions, and is the most complete in data. Fig. 3 demonstrates the progress in the transfusion.

The blood pressure rose very gradually from 110/60 to 140/85 during the transfusion. The pulse pressure increased only by 5 mm. Hg. This is not a significant change. The pulse remained at 110 until 8.15, when the venous escape blocked. Thereafter it rose slightly to 120, reaching this rate at 9.15 p.m., when the transfusion was stopped. This rise is probably significant, but not certainly so.

The cumulative blood-drip volume and the cumulative venous escape show a marked discrepancy. (It will be noted that there were half-hourly readings of blood drip volume, but only two readings of venous-escape volume were taken.) This discrepancy was noted when the first cumulative venous escape reading was taken. The blood-drip rates and venous-escape rates in drops a minute before the first cumulative venous-escape reading had not given any indication of an inequality.

At the outset the patient was very pale and his respirations were quick and sighing. He preferred to be supported on a back rest. He was very weak. His colour improved quite soon after the transfusion was begun. His breathing became easy and unhurried by 4.45 p.m., but he remained very weak. There was never any congestion of the neck veins. The total volume transfused was 1,020 ml., the total venous escape was 1,080 ml.; and the duration was seven hours. On July 8 a blood count showed: Hb, 5 g./100 ml. (34%); red cells, 1,880,000; colour index, 0.9. In a few days he became weaker and was given a direct transfusion. His progress was relentlessly downhill. He was constipated until July 11, when melaena occurred, so it is possible that he was bleeding into the bowel at the time of his transfusion. Death occurred on July 14. At the post-mortem examination the clinical diagnoses were confirmed. There was, in addition, oedema of the left

lower lobe, but no dilatation of the atria or ventricles. A recent subdural haemorrhage and gastro-intestinal haemorrhages were present.

FIG. 3.—Case 2. Exchange transfusion.

Case 3

A frail old lady was admitted with profound pernicious anaemia. Her feet had been swollen in the evenings throughout the previous two months, though this had gone since she had been confined to bed. There was no evidence of congestive heart failure.

It was decided to carry out an exchange transfusion; 2-mm.-bore polythene tubing was used. The transfusion lasted six and a half hours, and in that time 2,300 ml. was transfused, and 2,900 ml. removed (to the nearest 100 ml.).

The pulse remained between 80 and 90 throughout the transfusion. The total amounts exchanged were large in comparison with previous cases. Throughout the transfusion, however, the patient gradually improved; she became warmer and her colour returned. She was cheerful while awake and untroubled in her sleep. She was never distressed in any way, and never showed filling of the neck veins.

Slight puffiness of the eyelids was noted in the early hours of the morning when the transfusion was stopped.

At that time the pulse rose to 88. With the exception of one reading (at 9.30 p.m.) the pulse had not exceeded 84 prior to this last reading.

She was well next day. The change was very gratifying. The blood counts before and after transfusion were:

		Hb g./100 ml.	Hb %		R.B.C.	Retic.	C.I.
July 17	..	3.3	22	..	1.12	14.8	0.99
.. 18	..	3.4	23	..	1.07	15.2	1.08
Transfusion							
July 19	..	10.1	68	..	4.02	2.2	0.84
.. 24	..	8.4	60	..	3.444	1.0	0.87

The following night she developed dyspnoea, due to pulmonary oedema, which by 9 a.m. next day was severe. It responded to oxygen and rest, and by the afternoon she was perfectly well. No further complications occurred. She was resistant to treatment with liver extracts, and underwent a further transfusion at a later date. The transfusion was a slow direct transfusion without any venous escape and was well tolerated. Her haemoglobin when this transfusion was carried out was 32% (4.7 g./100 ml.).

Comment

It is suggested that exchange transfusion by the method described is indicated in (1) cases of severe anaemia with congestive heart failure, and (2) cases of severe anaemia (Hb 25% or lower) of many weeks' duration but not associated with venous congestion.

The advantages of the method are: (1) There is a greatly reduced risk of circulatory overloading, since the blood volume is controlled; and (2) the transfusion is more quickly done than with the usual practice of giving several small slow transfusions over the course of a few days.

Summary

A technique for exchange transfusion, in which exsanguination and transfusion are simultaneous, is described.

Three cases of severe anaemia are reported in which exchange transfusions were performed.

Indications for, and advantages of, exchange transfusion are mentioned.

My thanks are due to Dr. C. C. Ungley without whose help this paper would not have been written. They are due also to Mr. H. Campbell for the statistical interpretation of results, to Professor Duguid for the pathological data given, and to the medical and nursing staff of wards 10 and 12, Royal Victoria Infirmary, Newcastle-upon-Tyne, for their help and encouragement.

BIBLIOGRAPHY

- Altschule, M. D., and Gilligan, D. R. (1938). *J. clin. Invest.*, 17, 401.
 Caughey, J. L., Jun. (1935). *Proc. Soc. exp. Biol., N.Y.*, 32, 973.
 Drummond, R. (1943). *British Medical Journal*, 2, 319.
 Marriott, H. L., and Kekwick, A. (1940). *Ibid.*, 1, 1043.
 Riddell, V. H. (1939). *Blood Transfusion*. London.
 Sharpey-Schafer, E. P., and Wallace, J. (1942). *British Medical Journal*, 2, 304.
 Watson, L. (1943). *Lancet*, 1, 107.

On March 19 the first party of 25 British children left for Vordinborg, in Denmark, under the Anglo-Danish Society's scheme for their treatment at a Danish sanatorium. The children were chosen by the medical committee of N.A.P.T., and the British Red Cross helped to make the travel and welfare arrangements. Funds to meet the cost of transport and maintenance of this and subsequent parties of British children suffering from tuberculosis will come from the special fund created for the purpose by the Anglo-Danish Society. The ages of the children range from 8 to 15; there are 14 boys and 11 girls, mainly from London and the Birmingham and Oxford regions. It is expected that treatment in most cases will last about six months. The scheme will do something to reduce the waiting-list for beds in this country.

HOMOLATERAL PYRAMIDAL SIGNS WITH TUMOURS OF THE POSTERIOR FOSSA

BY

LESLIE C. OLIVER, F.R.C.S.

Neurosurgeon, West End Hospital for Nervous Diseases
and Royal Northern Hospital, London; Surgeon in Charge
of Neurosurgical Centre, Oldchurch Hospital, Romford

AND

J. R. GIBBS, F.R.C.S.Ed.

Senior Surgical Registrar, Neurosurgical Centre, Oldchurch
Hospital, Romford

It is well known that tumours and other displacement lesions of the cerebral hemispheres sometimes give rise to homolateral pyramidal signs. Kernohan and Woltman (1929) made a careful study of this phenomenon. The expanding lesion causes displacement of the brain stem, so that indentation of the *opposite* cerebral peduncle is produced by the edge of the tentorium; the affected pyramidal fibres cross below, in the decussation of the pyramids, to the same side as the lesion. The following case is cited as an example.

Illustrative Case

Case 1. Subdural haematoma of the right hemisphere: weakness of the right hand and arm, and equivocal right plantar response.—A man aged 47, who suffered from sleepiness for three months and headache and irritability for one month, became suddenly unconscious on June 22, 1946. There was no history of a head injury. On admission to hospital the same day he was conscious but very drowsy. There was no papilloedema and there were no localizing signs. The cerebrospinal fluid contained 80 mg. of protein per 100 ml., and it was yellow after treatment in the centrifuge. During the next few days the drowsiness fluctuated. Two weeks after admission the patient was referred to us. At that time there was *weakness of the right hand and arm* and the *right plantar response was equivocal*. Ventriculography, performed on July 6, showed displacement of the anterior part of the ventricular pattern to the left. A right frontal burr-hole revealed a subdural haematoma, which was solid and adherent to the membranes. A bone-flap was turned down and the haematoma exposed after wide reflection of the dura mater. The whole of the superolateral surface of the hemisphere was involved. There was no haematoma on the opposite side.

Comment.—It is not so well known that a closely similar phenomenon may occur with tumours in the posterior fossa, though Kernohan and Woltman found records of five examples in the series of intracranial tumours which they investigated, and Gould (1928) stressed the occurrence of *homolateral* hyperreflexia with some tumours of the posterior fossa. Tumours of the frontal region may produce "cerebellar" signs, and, conversely, slowly growing tumours in the posterior fossa may cause little or no disturbance of cerebellar function. Therefore the possible occurrence of pyramidal signs on the same side as a posterior fossa tumour may cause further difficulty in diagnosis.

In the following five cases various homolateral pyramidal signs were observed.

Case Reports

Case 2. Cyst of the left cerebellar hemisphere: paresis of the lower half of the face and weakness of the upper extremity on the same side.—A woman aged 46 was admitted to hospital in April, 1949, with a six-weeks history of intermittent headaches, giddiness, and vomiting. She showed a