America; and of foxes in Denmark, Finland, Norway, Holland, and the far west of the United States of America (for references to the literature, see Wilson and Miles, 1946). The apparent absence of this particular salmonella from the extensive eastern and mid-western parts of the United States is quite remarkable (Edwards *et al.*, 1948).

Raw milk from an infected cow is the usual vehicle for human *Salm. dublin* infections, and large outbreaks of food-poisoning from this source have often been reported. Symptoms of the infection vary from a mild gastro-enteritis to a fulminating septicaemia, with a special predilection for fatal meningeal involvement in young children. In Britain *Salm. dublin* has been found more invasive for humans than the other food-poisoning salmonellae (Report of Ministry of Health, 1950). The present carrier had no access to cow's milk; but at the time when he had his bout of fever he was part owner of a water buffalo and customarily drank its milk raw. Since *Salm. enteritidis* (Gaertner) infection of water buffaloes had been described (Wolff, 1930) before this closely related organism was generally differentiated from *Salm. dublin*, they may well be natural hosts, as cattle are, and the patient's febrile episode was perhaps a *Salm. dublin* infection resembling enteric fever, conveyed by buffalo milk, followed by the chronic carrier state. This man's unhygienic family environment emphasizes the possibility that his infant's death at weaning age was due to a *Salm. dublin* infection conveyed from the father.

Chronic human carriers of salmonellae other than typhoid and paratyphoid are relatively uncommon, and those that have been reported were almost invariably faecal. I have failed to find any other chronic human carrier of *Salm. dublin* described in the literature. Ledingham and Arkwright (1912), however, mention two cases of chronic urinary infection with *B. enteritidis* Gaertner, which at that time was indistinguishable from *Salm. dublin*.

ADDENDUM (January 15, 1952).—This man continued to be a profuse urinary excreter of *Salm. dublin* until September, 1951, when he was given 30 g. of chloramphenicol in the course of 10 days. In 100 post-treatment days all of 31 urine and 14 stool specimens have been negative for salmonella organisms, despite uncured urinary bilharziasis.

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The Archbishop of Canterbury said at a meeting of the Society for the Promotion of Christian Knowledge in London recently that the rising costs of printing meant that very soon we should become a nation of illiterate people (*Manchester Guardian*, March 7). This was not because we could not read intelligently—though he added that there were not so many who could—but because there would no longer be money left to print anything for people to read. This mounting cost was a real threat to our cultural life.

BALANTIDIASIS IN SOUTH PERSIA*

BY

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Balantidiasis is a rare disease in man, and it is therefore considered that the following 87 cases found in South Persia between April 1 and December 31, 1948, are worth placing on record. The only previous records of balantidiasis in Persia are by Stshensnovitsh (1943) and Selimkhanov (1947).

Local Conditions

The cases occurred among the population of Abadan, an oil refinery town on the island of Abadan, South Persia. This island is situated in the estuary of the Shatt-el-Arab river, at the head of the Persian Gulf, and is some 30 miles (48 km.) long and 5 miles (8 km.) wide at its broadest part. It is a flat desert, with small areas under cultivation. The temperature in winter seldom falls below freezing-point, and the summer shade temperature averages 110–118° F. (43.3–47.8° C.), with usually low humidity. The winter rainfall is about 15 in. (38 cm.). Rain does not fall during the summer.

The population of the town at the time consisted of about 200,000 Persians, 2,000 Indians and Pakistanis, and 5,000 British. About 100,000 of the Persians were either employed in the refinery operated by the Anglo-Iranian Oil Company or were dependants of employees. Much was done by the oil company to control and prevent the spread of disease by providing public lavatories and baths, clean piped water supply, an efficient sewerage system, and collection and disposal of refuse. It was sometimes difficult, however, to persuade the uneducated section of the populace to use these facilities, or to make them realize the value of simple hygiene.

In an effort to reduce the incidence of the dysenteries in Abadan a scheme was instituted on April 1, 1947, by which all artisan employees reporting sick and regarded as cases of dysentery on clinical grounds or from stool examination were admitted to the company's isolation hospital. Any employee found to be a carrier of dysentery was also admitted. This scheme was described by Stewart (1949), and led to the collection of this series.

An excellent account of the pathology, with necropsy reports, and a review of the literature on balantidiasis, is given by Koppisch and Wilking (1947).

Mode of Infection

All patients in this series were Mohammedans. In accordance with their religion they had no contact with pigs, nor did they eat pork. The pig can be excluded as the source of infection. This is in accordance with the conclusion reached by Ostroumov (1947) following examination of pigs and slaughterhouse workers in Soviet Central Asia.

No cysts were found in the stools in the Abadan series, and no source of infection, apart from the human cases, was discovered. Intestinal contents of sheep and cattle killed in the town slaughterhouse were examined, but no balantidia were found. The excreta of goats and samples of river water revealed no balantidia. After this investigation, which ended on December 31, 1948, cases occurred with much less frequency, but all continued to be admitted to hospital for treatment. Dr. I. S. Stewart later informed me in a personal communication that balantidiasis is now rare. A man-to-man transmission of the infection therefore seems to be strongly suggested.

*This paper is condensed from a thesis presented for the M.D. degree of the University of Glasgow, 1950.

Method of Investigation

The geographical location of the patient and any contact with animals over the two years prior to admission to hospital were investigated. Neither of these factors was found to be significant. A history of previous attacks of diarrhoea and of the present attack was taken. A history of previous diarrhoea was found in 65% of cases.

A routine physical examination, inspection of the lower 7 in. (17.8 cm.) of bowel by sigmoidoscopy, macroscopic and microscopical stool examinations, and stool culture were made. Each case was re-examined after six months, and if the stool was positive had further treatment and was followed up.

Clinical Features

Common complaints were diarrhoea, abdominal pain, tenesmus, and the passage of blood and mucus. In uncomplicated cases the symptoms in general were mild, though 22% had severe pain and diarrhoea with much blood and mucus. Seven cases which had concomitant bacillary dysentery and one with concomitant amoebic dysentery had severe symptoms.

Small discrete ulcers were found in the lower bowel in 11 cases. Scrapings of these ulcers invariably revealed the presence of numerous balantidia. These ulcers healed in each case after successful treatment of the infection. Physical examination commonly revealed tenderness and patchy thickening of the colon. No death occurred in the series.

Treatment

Until it became evident that acetarsol ("stovarsol") was a specific treatment, other drugs were given a trial. Two cases were admitted to hospital with positive stool reports, but no balantidia were found after admission, and no treatment was given.

Sulphaguanidine.—A daily dose of 20 g. was given to seven cases of uncomplicated balantidiasis. Three cases received a total of 20 g. each, one case 40 g., one 60 g., one 80 g., and one 100 g.

Mepacrine.—As mepacrine had proved successful in the treatment of giardiasis, it was given a trial. Five cases were each given 0.1 g. thrice daily. One case in this group had previously received sulphaguanidine, 80 g.

Mercury Binioidide.—In view of Shun-Shin's (1947) success with mercury binioidide, five cases were given two intramuscular injections of $\frac{1}{2}$ gr. (32 mg.) with an interval of two days between injections.

Acetarsol.—Eleven cases were given 4 gr. (0.26 g.) thrice daily for five days, a total of 60 gr. (4 g.). Fifty cases were given 4 gr. (0.26 g.) thrice daily, until the stools became negative, when treatment was stopped. Of these 50 cases, 3 had one day's treatment—12 gr. (0.8 g.), 11 had two days' treatment—24 gr. (1.6 g.), 27 had three days' treatment—36 gr. (2.4 g.), and 9 had four days' treatment—48 gr. (3.2 g.). Twenty cases which were positive at the follow-up or had relapsed within six months were also treated with acetarsol until the stools became negative. Six cases had two days' treatment—24 gr. (1.6 g.), 11 had three days' treatment—36 gr. (2.4 g.), and 3 had four days' treatment—48 gr. (3.2 g.) (see Table II).

TABLE I

No. of Cases Treated	Drug Used	Daily Dose	Days of Treatment	Method of Administration
7*	Sulphaguanidine	20 g.	1-5	Oral
5*	Mepacrine	0.3 g.	5	"
5*	Mercury binioidide	$\frac{1}{2}$ gr. (32 mg.)	2	I.M. injection
11	Acetarsol	12 gr. (0.8 g.)	5	Oral
50 plus 20 followed up	"	12 gr. (0.8 g.)	1-4	"

* Two cases had no treatment.

* Some of these cases were treated with two different drugs.

Thirteen further cases were treated with acetarsol, but as these men left the employ of the company, or were non-employees and could not be traced, they are excluded from further mention in this series. The treatment is summarized in Table I.

Table II summarizes the number of days of treatment and the quantity of acetarsol administered before the stools became negative in the last 50 cases of the series and in those cases which were positive at the follow-up or which relapsed.

TABLE II

No. of Cases	Days of Treatment	Amount of Acetarsol
3	1	gr. 12 0.8
17	2	24 1.6
38	3	36 2.4
12	4	48 3.2

Average no. of days of treatment = 2.84.

Average quantity of acetarsol given = 34.1 gr. (2.3 g.).

Immediate Response to Treatment

Of the seven cases treated with sulphaguanidine, the stools became negative in four, and remained positive in three. In no case treated with mepacrine or with mercury binioidide did the stools become negative. One case with concomitant amoebic dysentery received 10 intramuscular injections of emetine hydrochloride, 1 gr. (0.65 mg.), but *Balantidium coli* persisted in the stools.

Acetarsol was given to the 61 original cases, to the two that relapsed within six months, and to the 18 that were positive at the follow-up. No case failed to respond to this treatment, the stools becoming negative in every case. In two untreated cases the stools spontaneously became negative.

Result of Follow-up

One of the two untreated cases had a positive stool at the follow-up six months later. Acetarsol was given until the stools became negative, and six months later they were still negative. Sulphaguanidine, mepacrine, or mercury binioidide, or a combination of these drugs, was used in the treatment of 17 cases. All these had a positive stool at the follow-up after six months, and all were negative six months later, after treatment with acetarsol.

Of the 61 cases treated with acetarsol at the outset, two relapsed, one in a month and the other in two months, four had a positive stool on follow-up, and 55 a negative stool. The two relapses and the four which were positive at the follow-up were treated again with acetarsol, and all had a negative stool six months later.

All cases in the series were permanently cured by acetarsol, although two treatments were required in six cases. Table III summarizes the immediate response to treatment and the result of the follow-up.

TABLE III

No. of Cases	Treatment Given	Immediate Result of Treatment		Relapses	Follow-up	
		Stool Pos.	Stool Neg.		Stool Pos.	Stool Neg.
2	None	—	2	?1	1	1
7	Sulphaguanidine	3	4	—	7	—
5	Mepacrine	5	—	—	5	—
5	Mercury binioidide	5	—	—	5	—
61	Acetarsol	—	61	2	4	55

Discussion

The pathology of balantidiasis has been thoroughly investigated by several workers (Walker, 1913; Strong, 1942; Koppisch and Wilking, 1947). There is no divergence of opinion on the type of lesion produced or the

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.

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EXCHANGE TRANSFUSION IN SEVERE ANAEMIA

BY

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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.)