A Study of Balantidiasis coli[1]

REPORT OF TWO CASES IN CHILDREN SUCCESSFULLY TREATED WITH STOVARSOL

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GENERAL CONSIDERATIONS

BALANTIDIASIS COLI, also known as balantidiosis, balantidial dysentery, and ciliate dysentery, is a parasitic disease caused by the presence of the ciliate protozoan *Balantidium coli* in the intestinal tract, and it presents a clinical picture very similar, in general, to that of an amoebic dysentery. However, not infrequently *Balantidium coli* is found in the intestinal tract of man, with no clinical manifestations of disease on the part of the host; in some localities this aspect of the disease is the more common occurrence. Therefore we wonder whether some distinction such as has been made when dealing with cases of amoebic infection should not be established between balantidiasis (or balantidiosis) and balantidial dysentery.

The disease was first recognized and the parasite first described in Stockholm in 1857 by Malmstem, who named the parasite *Paramecium coli*. Five years later Stein noticed the remarkable resemblance of the newly discovered parasite to the already known *Balantidium entozoön*. After studying the former, Stein concluded that both belonged to the same genus and named it *Balantidium coli*. Since then numerous investigations have been undertaken with regard to the morphological and physiological properties of balantidia, particularly those found in the pig, monkey, ape, and man.

Balantidiasis is widely distributed throughout the world,[2] though the incidence of dysenteric symptoms resulting from its presence in the intestinal tract of man is relatively low. Approximately three hundred cases have been reported to date in medical literature from almost every country in Europe and Asia, as well as from the Sudan and Egypt in Africa, from Canada, the United States, the West Indies, Brazil, and Uruguay in the Americas. In the United States alone, forty-three cases have been reported. According to Zinsser,

1. Received for publication June 23, 1942.

2. E. B. McKinley, *A Geography of Disease* (Washington: George Washington University Press, 1935).

Pinto found about 1 percent of the population parasitized in southern Brazil; Nendergaard, a little more than ½ percent in eastern Cuba. In Puerto Rico in 1931, Dr. Américo Serra reported four cases of balantidiasis out of five thousand stool examinations; out of these four, only one had symptoms of dysentery.

The disease is contracted by the ingestion of contaminated food or drinking water, the source of contamination usually being the intestinal contents of the domestic pig.[3]

MORPHOLOGIC CONSIDERATIONS OF THE *Balantidium coli*[4]

Balantidia, of which there are about fifty different forms, fall into the general class-group Infusoria. Protozoa in this class are equipped with numerous hairlike appendages, or cilia, as organs of locomotion, and present typically a macronucleus and a micronucleus. There are two subclasses: the Suctoria, which lose their cilia on entering the adult stage, and the Ciliata, which retain them during the entire life cycle. *Balantidium*, a genus of the latter subclass, is the only species among the Infusoria of medical importance. The *Balantidium coli* is the largest protozoan parasite and probably the only ciliate pathogenic to man.

The organism is very actively motile when obtained from fresh stools or from lesions in the intestinal wall of the host. Its body is ovoid in shape and covered with cilia arranged in parallel, longitudinal lines. The more narrow anterior end is somewhat pointed and presents a subterminally situated funnel-shaped structure, the peristome. The parasite varies in size from thirty to two hundred micra, or more, in length, and twenty to seventy micra in breadth. Its body surface is covered with a dense cuticle, beneath which lies a clear layer of ectoplasm, contrasting sharply with the granular endoplasm in which may be seen food vacuoles containing food particles. The macronucleus is large and kidney-shaped, while the micronucleus is small and usually situated in the indentation of the former. The cytoplasm also shows two contractile vacuoles as well

3. E. Schumaker, "*Balantidium coli;* Host Specificity and Relation to Diet of Experimental Host," *Am.J.Hyg.*, XII (1930), 341; "Relation of *Balantidium coli* Infection to Diet and Intestinal Flora of Domestic Pig," *Am.J.Hyg.*, XIII (1931), 576.

4. C. W. Rees, "Studies on Morphology and Behaviour of *Buxtonella sulcata* from Cattle and of *Balantidium coli* from Pig," *Parasitology*, XXII (1930), 314.
H. L. Ratcliffe, "Intestinal Lesions Associated with Amebic and Balantidial Infection in Man and Lower Animals," *Am.J.Hyg.*, XIX (1934), 68.
R. Hegner, "Specificity in Genus: Balantidium Based on Size and Shape of Body and Macronucleus, with Descriptions of six New Species," *Am.J.Hyg.*, XIX (1934), 38.

as an opening in the cuticle at the posterior extremity, which serves as a cytopyge for the extrusion of indigestible residue food matter.

Multiplication takes place through both the vegetative and sexual processes, that is, through binary division and conjugation, as shown by Nelson's[5] experiments. Cultivation of the parasite *in vivo* and *in vitro* has been achieved by several workers[6] in the past few years. Nelson points out, however, that cultures *in vitro* will not thrive in the complete absence of bacteria.

Balantidial cysts are spherical and present a protective, transparent double wall. They measure about fifty to sixty micra in diameter.

ANIMAL HOSTS

Species of Balantidia have been found in the intestines of pigs, monkeys, chimpanzees, guinea pigs, and rats. The parasite may be present in the stools for years without apparent harm to the host, yet sometimes it has been found to cause severe and rapidly fatal dysentery.

Minor morphological and cultural differences have been observed among the various strains which characterize them, and therefore they have been named according to their particular natural host. Experimental interinfection[7] has been possible in some instances, but no other strain than the *Balantidium coli* of the domestic pig has been known to infect man, not even that of *Balantidium suis*, also a habitual guest of the intestines of the pig.

THE DISEASE IN MAN

In man the disease may manifest itself in two ways, rather, the patients may well be divided into two groups. In one the parasites

5. E. C. Nelson, "Observations and Experiments on Conjugation of Balatidium from Chimpanzee," *Am.J.Hyg.*, XX (1934), 106.
F. O. Atchley, "Effects of Environmental Changes on the Growth and Multiplication in Populations of Balantidium," *Am.J.Hyg.*, XXI (1935), 151.
A. Marques da Cunha and J. Muñiz, "Conjugação e endomixis em ciliados do genero Balantidium," *Mem.Inst.Oswaldo Cruz*, XXXII (1937), 75 (94 e).

6. E. C. Faust, "Method for Obtaining Pure Culture of *Balantidium coli*," *Proc.Soc.Exper. Biol.and Med.*, XXVII (1930), 648.
E. C. Nelson, "Intestinal Content Cultivation Medium; Methods of Preparation and Use and Data Obtained in Cultivation of *Balantidium coli* from Pig," *Am.J.Trop.Med.*, XX (1940), 731.

7. E. C. Nelson, "Cultivation and Cross-Infection Experiments with Balantidia from Pig, Chimpanzee, Guinea Pig, and Macacus rhesus," *Am.J.Hyg.*, XXII (1935), 26.
A. Gabaldón, "*Balantidium coli:* Quantitative Studies in Experimental Infections and Variations in Infectiousness for Rats," *J.Parasitology*, XXI (1935), 386.

are found consistently or intermittently in the stools, but the host has no complaint and is entirely free from symptoms, intestinal or otherwise, except for perhaps an occasional loose bowel movement or the periodic appearance of small amounts of mucus in the feces. A patient in this group is not a sick individual but actually a carrier, though his potentialities for transmitting the disease are still unknown. It is known that the disease never has occurred in epidemic form and that there is no conclusive evidence to support the theoretical possibility of its transmission among human beings. This has been suggested from time to time and has been in some instances (as in Young's cases[8]) a logical assumption. Nevertheless, apart from their epidemiological aspect these cases present a clinical problem, technically identical with that of amoebiasis. The condition should rightly be termed balantidiasis or balantidiosis, in contradistinction to balantidial dysentery.

The other group will then comprise those patients who, in addition to the presence of *Balantidium coli* in the stools, show local and sytemic symptoms, usually referable to the large intestine—a clinical condition almost indistinguishable from that of amoebic dysentery. This should be regarded as true balantidial dysentery.

The severity of symptoms will depend largely upon: (*a*) massiveness of infection; (*b*) state of nutrition of the host; (*c*) nature of the host's diet; and (*d*) nature of the intestinal flora at the time of infection. Intermittent diarrhea, with or without blood, generalized abdominal tenderness, and sometimes colicky pains are the most common symptoms. In severe, untreated cases the systemic detriment inherent to any protracted diarrhea eventually will be evident as dehydration, weakness, loss of weight, anorexia, anemia, and finally cachexia. The usual case runs a slow chronic course, although a few rapidly fatal infections have been reported. One such case was recently lost in the University Hospital. In unusually severe cases symptoms such as nervousness, fever, vague diffuse pains, and marked mental depression have been recorded.

Brea and Nieto[9] have reported patients in Uruguay free from intestinal symptoms, but with various systemic manifestations, such as fever, urticaria, pruritis, delirium, and eosinophilia. This, however, has not been the experience of other observers,[10] and it is generally accepted that the leukocytic picture remains unchanged unless the host is harboring other parasites capable of producing eosinophilia, as happened in the cases reported herein.

8. M. D. Young, "Balantidiasis," *J.A.M.A.*, CXIII (1939), 580.
9. R. J. Brea and C. A. Nieto, "Balantidiasis humana en el Uruguay; contribución a su estudio," *Arch.urug.de med., cir.y especialid.*, XI (1937), 720.

PATHOLOGY

Balantidium coli is far more pathogenic to the human being than it is to its natural host, the pig. In man the parasite frequently invades the wall of the large intestine, though it is probably confined in many instances to the lumen of the bowel. As it pierces the mucosa, the parasite forms small, shallow ulcers with undermined irregular borders, or it may wander from the initial point of invasion and form small fistulous tracts. Groups of organisms may form flask-shaped ulcers, which extend into the submucosa. Very little tissue reaction can be demonstrated at autopsy, except around disintegrating parasites where a moderate infiltration with lymphocytes and plasma cells may be exhibited. Walker reported ulcers of the small intestine due to *Balantidium coli;* Stokes and Masson saw abscesses of the liver; and Winogradow reported a case in which a lung abscess developed due to the presence of the parasite in the lung.

DIAGNOSIS

The diagnosis of balantidial dysentery is simple enough if the disease is kept in mind; it rests entirely upon the presence of *Balantidium coli* in the stools of the patient. A history of close association with pigs, a low-grade chronic diarrhea, and an occasional appearance of blood and mucus in the stools should strongly suggest the disease. The parasite is not always present in the stools, however, so repeated examinations may be necessary. The actively motile organisms are more likely to be found in the mucoid portions, while the more resistant encysted form is usually encountered in harder portions of the stools. The large size of the parasite and its conspicuous ciliated body and marked activity make it easily recognizable.

10. B. Siffert de Paula e Silva, "Human Balantidiasis: Clinical and Therapeutic Aspects," *Brazil Med.*, LII (1938), 1005.
D. Yered, "Algumas considerações sobre um caso de balantidiose intestinal e seu tratamento," *Brazil Med.*, XLIX (1935), 1093.

TREATMENT

As in any infectious disease, treatment resolves itself into prophylaxis, specific treatment, if any, and general supportive measures. Prophylaxis consists mainly, as far as is known, in preventing ingestion of food and drinking water contaminated with the excreta of pigs.

Opinion concerning drug treatment of balantidial infection is widely divergent. In his *Textbook of Medicine* Cecil[11] states that "no drug specific for this disease has been discovered." Manson-Bahr, in his recent book, *Dysenteric Disorders*,[12] likewise states that the therapy is purely empirical and limits himself to listing innumerable remedies recommended by different workers. In 1928 Greene and Scully[13] already had argued that from the large number of different drugs advocated it was apparent that no single one was of benefit, and they encouraged the use of the diet treatment which they had successfully used in four cases.

There is no denying that the large number of drugs recommended for treating this disease has brought no little confusion and distrust to the mind of the physician seeking guidance in literature as to the management of these cases. On the other hand, one is forced to admit that enough evidence has been brought forth to support the value of some of the therapeutic agents advocated. This state of affairs may be due partly to the fact that because of the relative infrequency of the disease no particular therapeutic regime has been tried on a sufficiently long series of cases to prove its undisputed merit. Nevertheless, the fact that one case fails to respond to treatment by a certain drug does not invalidate the usefulness of the drug if other cases have been helped by it, any more than failure to save one case of malaria under quinine treatment belies its specificity. One must also bear in mind that most drugs used in treating balantidial infection are potent ones and that success will depend largely on the experience and ability of the physician to achieve optimum therapeutic results without causing harm to the patient.

For the purpose of description, the treatment of balantidial dysentery may be divided into: (1) drug therapy by mouth, (2) injection therapy, (3) medicated enemas, and (4) diet treatment.

Among drugs that have been used by mouth are: thymol, calomel,

11. R. L. Cecil, *A Text-Book of Medicine* (Philadelphia: W. B. Saunders Co., 1927).
12. P. Manson-Bahr, *Dysenteric Disorders* (Baltimore: Williams and Wilkins, 1940).
13. J. L. Greene and F. J. Scully, "Diet in the Treatment of *Balantidium coli* infection," *J.A.M.A.*, LXXXI (July 28, 1923), 291.

carbolic acid in pills, extract of male fern, methylene blue, ipecacuanha, emetine, yatren, oil of chenopodium, Stovarsol, Santonin, and Carbarsone. Medicated enemas include: iodine solution 1-10,000, tannin solution 1-1,000 with 10 to 15 drops of tincture of opium, enemas of quinine suspension, methylene blue, oil of chenopodium in olive oil, and carobinase (a watery extract of *Jacoranda decurrens*, a South American plant) 25 gm. in 500 cc. of hot water. Injection therapy has been limited to emetine hydrochloride subcutaneously in doses of 0.05 gm. for fifteen to twenty injections.

The great majority of these drugs have fallen into disuse with the exception of two or three that have proved of definite value, such as Stovarsol and Carbarsone. In 1925 Aguilar reported forty cases of balantidial infection treated successfully with acetarsone (Stovarsol). Commenting on that report, Cort [14] stated that before Aguilar's work the only suggestions available were to treat as for amoebic dysentery; mortality was given at 30 percent. Aguilar regarded a course of 0.25 gm. of Stovarsol after each meal for seven days as specific, especially if the excess of carbohydrates in the diet was replaced by proteins and green vegetables. Since Aguilar's cases, various others have been reported as successfully treated with Stovarsol in doses of four grains, two or three times a day for eight to ten days. One of the cases reported here, a child, was rid of the parasites after very small doses of the drug; another case was treated in the University Hospital with the same good results.

In 1939 Young[15] reported seven cases of balantidiasis among inmates of the South Carolina State Hospital. The cases were doubly interesting because there was no history of contact with pigs and the possibility arises, as Young believes, of contraction of the disease from human sources. Two of these cases were treated effectively with Carbarsone in doses of 0.25 gm., twice daily for ten days.

Treatment with medicated enemas has been losing popularity. Both Carbarsone and Stovarsol can be used and have been used in enemas, but there seems to be no particular advantage over the oral method. In obstinate cases, simultaneous use of both methods may prove helpful.

The use of oil of chenopodium by rectum is worth mentioning. This drug is of particular interest because of its frequent use and

14. E. C. Cort, "Infection with *Balantidium coli:* Twelve Cases Treated with Oil of Chenopodium," *J.A.M.A.*, XC (1928), 1430.
15. M. D. Young, *op. cit.*

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because this method caused the death of a child in Puerto Rico several years ago—a reminder of its potency and danger. In 1928 Cort[16] reported from Siam a series of twelve cases of Balantidiasis treated with oil of chenopodium in retention enemas. Fifteen cc. of the drug were dissolved in 150 cc. of olive oil and the mixture allowed to run in slowly. The patients were instructed to try and hold the enemas for two hours, if possible, at the end of which time a soap-sud cleansing was given. The patients were rid of the parasites after twenty-four to forty-eight hours and remained free of them during the follow-up period, in one case for almost two years. No toxic symptoms were observed in eleven cases. The twelfth case received two treatments within twenty-four hours by mistake and was seriously ill, but recovered eventually.

Following Cort's method, one of our colleagues[17] treated a six-year-old child in 1931, using 4 cc. of the oil of chenopodium in 25 cc. of olive oil. The procedure resulted in death of the patient several hours after treatment. The method is doubtless effective, as is shown by Cort's cases, but it is a heroic and dangerous measure and, in our present knowledge, an unnecessary risk to the patient.

The diet treatment, as suggested by Greene and Scully in 1928, should be given serious consideration, if not as an independent therapeutic measure, at least as a valuable adjunct to drug treatment. Nelson's[18] experiments on feeding reactions of *Balantidium coli* from the chimpanzee and pig have brought forth evidence that the selection in feeding exercised by the parasite depends not on the size but on the quality of the particles; there was a definite preference for starchy ones. Four cases treated by Greene and Scully under dietary regime alone were managed as follows: the patients were fed two and a half quarts of whole milk daily in moderate amounts. After several days, soft-boiled eggs were added to the diet. No drug was used except bismuth, if the stools were too frequent or watery, and stewed fruits, if the bowels became sluggish. A gradual return to a full diet was allowed according to progress. Stools became negative in eight to twenty days after beginning treatment.

16. E. C. Cort, *op. cit.*
17. A. Serra, "Balantidial dysentery in Child, Death Following Rectal Administration of Oil of Chenopodium," *P.R.J.Pub.Health & Trop.Med.*, VI (1931), 443.
18. E. C. Nelson, "Feeding Reactions of *Balantidium coli* from Chimpanzee and Pig," *Am.J. Hyg.*, XVIII (1933), 185.

CASE REPORTS

In the series studied, the four cases ranged in age from six to nine years. Three of them (two boys and a girl) were siblings; the fourth was a cousin and next-door neighbor. In all instances the history was the same, except in Case 1 where there had been no blood in the stools. A point of interest was that these patients had been in close association with pigs on several occasions for months during their early childhood.

Only two of these cases are reported here. The other two were omitted because after one or two positive stool examinations, they became negative without treatment and have remained so for over a year.

Cases were under observation for a period of about six weeks on a full diet. During this period they received treatment for other incidental intestinal parasites. On the sixth week Case 1 was started on a routine Stovarsol treatment for congenital lues (Table 1) and

TABLE 1

Case I[a]

Week	*Number of Stools Examined per Week*	*Number of Positive Stools per Week*	*Number of Negative Stools per Week*	*Percentage of Positive Stools per Week*
1	6	6	0	100
2	7	1	6	14
3	5	2	3	40
4	7	3	4	57
5	6	3	3	50
6	4	2	2	50
7	5	2	3	40
8	6	4	2	66
9	7	1	6	14
10	5	0	5	0
11	6	0	6	0
12	6	0	6	0

[a] Data compiled and tabulated in weekly periods. Computation of data obtained from the examination for *Balantidium coli* of all stools passed during a three-month period. Antiluetic treatment with Stovarsol started on the sixth week.

the effect of the drug on the *Balantidium* observed. Case 2 was treated with Chiniofon, both orally and by medicated enemas (Table 2).

Case 2 failed to respond to treatment with Chiniofon and was

TABLE 2

Case II (First admission)[a]

Week	Number of Stools Examined per Week	Number of Positive Stools per Week	Number of Negative Stools per Week	Percentage of Positive Stools per Week
1	4	2	2	50
2	5	3	2	60
3	5	0	5	0
4	7	3	4	41
5	6	4	2	66
6	5	2	3	40
7	7	3	4	41
8	6	0	6	0
9	3	2	1	66
10				
11				
12				

[a] Data compiled and tabulated in weekly periods. Computation of data obtained from the examination for *Balantidium coli* of all stools passed during a nine-week period. Chiniofon enemas started on fifth week. Chiniofon by mouth started on sixth week. All medication discontinued on seventh week because of intolerance for the drug.

TABLE 3

Case II (Second admission)[a]

Week	Number of Stools Examined per Week	Number of Positive Stools per Week	Number of Negative Stools per Week	Percentage of Positive Stools per Week
1	10	4	6	40
2	6	2	4	33
3	6	4	2	66
4	6	2	4	33
5	5	2	3	40
6	6	6	0	100
7	5	1	4	20
8	4	3	1	75
9	7	3	4	43
10	6	0	6	0
11	7	0	7	0
12	6	0	6	0

[a] Data compiled and tabulated in weekly periods. Computation of data obtained from the examination for *Balantidium coli* of all stools passed during a three-month period. Patient on a high protein diet for nine weeks without medication. Stovarsol treatment started on the tenth week.

readmitted several months later, at which time he received Stovarsol with good results (Table 3).

All stools passed during hospitalization periods were examined carefully. Proctoscopic examination performed in all cases failed to reveal significant findings.

CASE 1. *J.M.*, a light mulatto boy of nine years admitted to the hospital on January 13, 1941, with the following history: anorexia, diarrhea, and generalized abdominal pain off and on for four years previous. Diarrheal periods prolonged and frequent, child passing an average of five to six stools a day and occasionally one or two at night. Stools were semisolid to liquid in character and sometimes contained mucus, but no blood. At times defecation was attended by tenesmus. Patient suffered from frequent colds, often complained of weakness and failed to put on weight. Past history revealed patient had had measles and chickenpox in early childhood, and pneumonia a year previous to admission. Patient's father was known to have had syphilis and had died of pulmonary tuberculosis in middle age. Patient's mother was still living but mentally deranged. Child was cared for by maternal grandmother.

Physical examination. A poorly nourished and poorly developed nine-year-old, apparently in no discomfort. Skin negative except for low-grade chronic, superficial ulcerations on legs, presumably of traumatic origin. Cervical and inguinal lymph glands slightly enlarged. On admission, patient was suffering from acute upper respiratory infection; lungs and heart negative on physical and X-ray examinations. Liver and spleen not enlarged; no tenderness on palpation of the abdomen. There were none of the common stigmata of congenital lues.

Laboratory Findings

Hemoglobin	73%
R.B.C.	3,480,000
W.B.C.	19,000

Differential Count

Polys	19%
Lymphocytes	69%
Eosinophils	12%

Blood Kahn	4.4.4
Urine	Essentially negative
Feces	*B. coli;* uncinaria; *G. lamblia*

Treatment. Patient was placed on an optimum diet, receiving treatment for intestinal parasites other than *Balantidium coli.* Iron therapy also in-

stituted. Stovarsol started on sixth week as given routinely in Antiluetic Clinic. Small doses of 50 mg. given daily the first week; 100 mg. daily the second week, thus gradually increasing dosage until 750 mg. daily were given by the eighth week.

Results. Improvement noticed from the start with rest and nourishing food. Response to the drug not evident until third week, when dosage had been brought up to 150 mg. daily. From then on stools became consistently negative and remained so for over a year. Incidentally, serology also remained negative.

CASE 2. *P.J.*, a nine-year-old white boy (cousin to Case 1), admitted the same day as preceding patient and giving very similar history: anorexia, intermittent diarrhea (occasionally bloody), accompanied by tenesmus and generalized abdominal pain; duration, four years; no fever, vomiting, respiratory or, genitourinary symptoms. Child active and happy but failed to put on weight. Boy had had measles in early childhood; no other illnesses except occasional colds. Father and mother living, both middle-aged but apparently well, father having received antiluetic treatment. Two siblings living and well; one sister suffering from pulmonary tuberculosis.

Physical examination. A well developed, somewhat undernourished nine-year-old, apparently well. Skin and appendages negative; head and neck revealing no important findings except for hypertrophied tonsils and dental caries. Lungs clear throughout and negative to X-ray; heart also negative. Liver and spleen not enlarged; external genitalia and extremities normal.

Proctoscopic examination revealed no ulcerations of rectal mucosa.

Laboratory Findings

Hemoglobin	72%
R.B.C.	3,470,000
W.B.C.	10,400

Differential Count

Polys	52%
Lymphocytes	34%
Eosinophils	10%
Monocytes	4%
Blood Kahn	3.3.3
Urine	Essentially negative
Feces	*B. coli;* ascaris eggs; hookworm eggs; whipworm

Treatment. Patient observed for six weeks and then treated with Chiniofon medicated enemas, supplemented after a week with Chiniofon by mouth in doses of 0.25 gm., t.i.d. After six days of combined treatment, patient developed epigastric pain, frequent vomiting and anorexia, passing one or two loose blood stools. Withdrawal of drug resulted in prompt recovery. Patient discharged on a regular diet and followed up in O.P. Department. Periodic stool examinations consistently positive for *B. coli.* Readmitted about three months later, placed on high protein diet for six weeks and then administered Stovarsol in doses of 0.25 gm., t.i.d. Stools negative after forty-eight hours (Table 3). Drug discontinued after a week and antiluetic treatment continued with mercury and bismuth preparations for some time before arsenicals were again administered.

Results. Stools have remained consistently negative for *B. coli* for over a year.

SUMMARY

1. A brief summary of the recent literature on *Balantidium coli* has been presented.

2. Two cases of balantidial dysentery are reported with data to demonstrate the curative effect of Stovarsol.

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