spontaneously antevert in the early weeks with or without the aid of posture. When seen in the early weeks patients should be instructed to sleep in Sims's position or, if they prefer, in the full prone position. If anteversion has not occurred by the 12th week, then more active measures should be adopted to avoid the possibility of incarceration with its accompanying pain, retention, and possible super-added urinary infection. Preferably, the patient should be admitted to hospital, a large rubber watch-spring pessary inserted into the vagina, as advocated by Sinclair (1900), and posture continued under supervision. The postural treatment may be elaborated to include knee-chest position and prone lying for short periods during the day, and instructions should be given that any difficulty in micturition is to be promptly reported. Such difficulty may be due to too large a pessary, and a full bladder will tend to prevent anteversion. The regime should be tried for a few days and, if unsuccessful, manual reposition without or with an anaesthetic should be attempted. Once the uterus is anteverted, a watch-spring pessary is inserted to help retain the anteverted position and is worn until the fundus approaches the umbilicus at about the 20th week.

There will be very few cases where one or other of the above procedures is unsuccessful. Laparotomy is mentioned by many authorities for such cases, dense adhesions being blamed for failure of the more conservative methods of treatment. There was one case in the series under review in which the patient had a right tubal pregnancy treated by operation in 1928. In 1929 she was diagnosed as having a fixed retroversion. She was next seen in 1932, when 8 weeks pregnant with a retrodisplaced uterus which appeared fixed. She was treated by posture: spontaneous anteversion occurred and the pregnancy continued to a successful conclusion. It seems probable that there were adhesions, which did not, however, prevent anteversion occurring as pregnancy advanced. On the other hand, if adhesions are extensive it is probable that the patient will be sterile.

If incarceration occurs then it is the bladder that must receive attention in the first instance. Continuous catheterization is preferable to intermittent catheterization, and should be given along with postural treatment. Slow decompression of the bladder is needed if the retention has been of long standing. If this treatment is unsuccessful in 24 to 48 hours, a watch-spring pessary is used for 48 hours as outlined above. If such procedures fail manual reposition will be needed. Urinary antiseptics should be given in all cases.

Summary

A series of 66 patients with a retrodisplaced gravid uterus, who were admitted to the wards, is analysed.

A plea is made for conservative treatment of the sterile woman with a retrodisplaced uterus.

It is suggested that operative correction of a retrodisplaced uterus for a previous abortion is unwarranted.

Incarceration may occur between the 13th and 17th weeks of pregnancy, and two cases of sacculation of the incarcerated gravid uterus are briefly described. The cardinal symptom of incarceration is sudden onset of retention of urine with lower abdominal pain.

Treatment of the condition, which is primarily instituted to avoid incarceration, is outlined.

I wish to express my gratitude to my late chief, Prof. F. J. Browne, and to Prof. W. C. W. Nixon, for permission to make use of the case records of the Obstetric Unit, University College Hospital.

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LIVER DAMAGE IN AMOEBIASIS

BY

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Despite the obviously close connexion of amoebic colitis with liver disease there have been surprisingly few attempts to investigate the condition of the liver in amoebiasis other than by clinical methods. Much of the symptomatology of amoebic dysentery suggests that there is some degree of liver damage; the toxic appearance, anorexia, emaciation, sallow complexion, and chronic ill-health are such symptoms and signs, and to cases presenting these Manson-Bahr (1943) has applied the term "amoebic cachexia." In addition, he says (Manson-Bahr, 1940) that during the course of an attack of amoebic dysentery, and for many months afterwards, the possible supervention of amoebic hepatitis must be borne in mind, and the "condition of the liver must receive the most careful attention"; but this careful attention is presumably dependent entirely upon the clinical acumen of the physician.

The *Quarterly Cumulative Index Medicus* for the years 1931 to 1944 and the *Tropical Diseases Bulletin* from 1934 to 1944 list only four attempts to apply laboratory methods to assessments of liver damage in amoebiasis—namely, Heilig and Visveswar (1944), Gminder (1939), Barbagallo (1936), and Cordaro (1938). In the case of the first three investigations the tests were the hippuric acid synthesis, the Takata-Ara and the Weltmann coagulation reaction. The paper by Cordaro was not available.

When an investigation of the condition of the liver in amoebiasis was contemplated consideration had to be given to the limited time, equipment, and technical assistance available in the laboratory of a large naval auxiliary hospital, and a method chosen which, it was hoped, not only would give reliable results but could also be applied rapidly to a reasonable number of cases. Consideration of the liver-function tests and those empirical procedures which are stated to assess the degree of liver parenchyma damage led to the choice of Hanger's cephalin-cholesterol flocculation test (C.C.F.T.) as the most promising to apply to this problem.

The C.C.F.T. was first introduced by Hanger in 1938, and since that time has been studied and used considerably in North America and, to a smaller extent, in Britain. The results and conclusions have been published by Hanger (1939), Pohle and Stewart (1941), Hanger and Patek (1941), Gutman and Hanger (1941), Rosenberg (1941), Lippman and Bakst (1942), Mateer, Baltz, Marion, and MacMillan (1943), Mirsky and von Brecht (1943), Bruger (1943), Kirschner and Glickman (1943), Kopp and Solomon (1943),

Yardumian and Weisband (1943), and Dick (1945). The consensus of opinion is that it is a sensitive means of demonstrating disease of the liver parenchyma cells.

Material, Technical Procedure, and Results

The patients were selected in that they were all Europeans. Asiatics (Ceylonese) were excluded in view of the high incidence of chronic malaria and helminth infestations to be found in them. This was of considerable importance, as the sera of malaria patients will cause flocculation with cephalin-cholesterol emulsions (Kopp and Solomon, 1943), while Karunaratne (1940) reported five cases of histologically perfect hepatic cirrhosis in Asiatic children with heavy hookworm infestations. Chopra has estimated that 90% of the natives of Ceylon harbour the *Ankylostoma duodenale*, whereas among the European Service personnel dealt with over a period of a year (during which time 450 faecal specimens were examined monthly) only one case of hookworm disease was diagnosed.

The diagnosis of amoebiasis was established in every case by the demonstration of the trophozoites or the cysts of *Entamoeba histolytica* in the faeces or in the material obtained by sigmoidoscopy or proctoscopy.

The C.C.F.T. was done on each patient's serum as soon as the diagnosis was made, approximately half-way through the course of anti-amoebic treatment, and on completion of the course. As the treatment for amoebiasis is both exhausting and unpleasant, the patients were always granted fourteen days' sick leave, which they usually spent in a hill station. Thereafter they returned to the hospital for the test of cure. This necessitated a stay of about a week in hospital, so the opportunity was taken to carry out a final C.C.F.T. before discharge to duty. Owing to the exigencies of the Service, this final test had to be omitted in some cases.

The scheme of treatment in all cases was based on that advised at the Liverpool School of Tropical Medicine, and is as follows:

Emetine hydrochloride was given subcutaneously at night in 1-gr. doses to a maximum of 10 gr. Many of the patients had less than the maximum dosage, as the sterilizing course was started as soon as the acute symptoms had been controlled.

The *sterilizing course* lasted 21 days, the days being numbered 1, 2, 3, 4, etc., to 21. Day 1 is the day following the last emetine injection.

Odd Days: 7 a.m., 11 a.m., 3 p.m., and 7 p.m., 1 gr. (65 mg.) of "auremetine" in a capsule; 9 a.m., 1 p.m., and 5 p.m., 1 dr. (4 g.) bismuth subnitrate by mouth in a quarter-pint (142 ml.) of milk.

Even Days: 7 a.m., 12 noon, and 5 p.m., 4 gr. (0.25 g.) of "stovarsol" by mouth; 8 a.m., 2% sodium bicarbonate lower bowel wash-out; 9 a.m. 2-4% "quinoxyl" retention enema of 1 pint (568 ml.), to be retained 8-10 hours.

Sick leave was spent in a hill station.

The *test of cure* was 7 days in hospital for faeces examination and in some cases sigmoidoscopy.

Hanger's test was done: (1) before the first dose of emetine hydrochloride, (2) between day 9 and day 11 of the sterilizing course, (3) within two days of the completion of the sterilizing course, and (4) on return from sick leave for the test of cure.

The cases were divided, on clinical grounds, into four groups, as follows: (1) acute amoebic dysentery, (2) chronic amoebic colitis, (3) amoebiasis presenting with unusual symptoms, (4) amoebic hepatitis.

Although this work was intended primarily as an investigation of the liver in cases of intestinal amoebiasis, the opportunity was taken of applying the C.C.F.T. to a small number of patients suffering from amoebic hepatitis. They were selected with considerable care, so there was no reasonable doubt of the correctness of the diagnosis. It is

well known that *E. histolytica* is frequently not found in the faeces of patients with amoebic hepatic disease (55% according to Manson-Bahr, 1940), but in all the cases shown in Table IV the cysts of *E. histolytica* were demonstrated, and this fact, combined with the symptoms, physical signs, radiological evidence, and white blood cell counts, made the diagnosis almost certain. The response to emetine therapy clinched the diagnosis in every case.

Cephalin was extracted from sheep brain and made up with cholesterol in ether, as originally described by Hanger (1939). His technique for the test was also followed, except that the final readings for the degree of flocculation (i.e., those shown in Tables I-IV) were made after a 24-hour interval and not after 48 hours. Dick (1945) states that a considerable proportion of normal sera will cause some flocculation after 48 hours, and this observation was amply confirmed in the present work.

Normal controls were included as a routine with every batch of sera from amoebiasis cases which were put up for the C.C.F.T. The numbers in the batches varied from 10 to 26, and with each batch at least two normals and one abnormal were included. The normal controls were sera from members of the medical, nursing, and sick-berth staffs of the hospital. Results were as follows:

Total No. tested	47
No. with negative C.C.F.T.	45 (95.7%)
" " positive	2

The two positives were obtained with the sera of male laboratory technicians: one gave a history of severe and frequent attacks of migraine, while the other had had attacks of catarrhal jaundice 5, 21, and 31 months before the first C.C.F.T. The results of the test in these two men varied from $\pm$ to $++$ over a period of about a year. The positive control sera were obtained from cases of malaria and from patients suffering from obvious liver disease, chiefly catarrhal jaundice. Among the jaundice cases the results varied from $++$ to $++++$, depending upon the severity and stage of the disease. Of 17 cases of malaria 8 were benign tertian and 9 malignant tertian. All gave $++++$ reactions, with the exception of two of the benign and one of the malignant tertian which gave only $+++$ degree of flocculation.

Discussion

Heilig and Visveswar (1944) investigated the functional efficiency of the liver in 15 cases of acute or subacute amoebic dysentery using the hippuric acid synthesis test (oral technique). They concluded that over 46% had definite impairment of liver function. If the cases shown in Tables I and II are considered together, approximately 49% of the patients gave evidence of parenchymal liver damage. There is thus fair agreement in the results obtained, using entirely different laboratory techniques. With regard to the response to treatment, it is impossible to draw an exact parallel between the work of the above-named authors and that recorded in this paper, as the patients investigated with the aid of the C.C.F.T. were given the minimum quantity of emetine hydrochloride required to control any acute symptoms, followed by a complicated schedule with a multiplicity of drugs. On the other hand, all of Heilig and Visveswar's patients received a routine course of 12 gr. (0.8 g.) of emetine.

Gminder (1939) applied the Takata-Ara test to the sera of 23 patients with amoebic dysentery (9 acute and 14 chronic). He concluded that there was no evidence of liver damage in any of the acute cases, while in 10 (over 71%) of the chronic cases there was some evidence of parenchymatous liver change. Barbagallo (1936) used the Weltmann coagulation test in connexion with a series of patients suffering from amoebic and non-amoebic colitis

TABLE I.—*Acute Amoebic Dysentery*

Case No.	Age	Presenting Symptoms	C.C.F.T.			
			1	2	3	4
1	28	22 days' acute diarrhoea ..	+	—	—	—
2	32	28 " diarrhoea and occasional vomiting	+++	+	—	—
3	21	17 days' diarrhoea and colicky upper abdominal pain	—	—	—	—
4	19	3 days' acute diarrhoea ..	—	—	—	—
5	25	3 " mild " ..	++	++	±	—
6	26	5 " " " ..	++	++	—	—
7	19	5 " diarrhoea and abdominal discomfort	+++	++	—	—
8	23	24 hours' hyperacute diarrhoea and abdominal pain	++	+	±	—
9	22	9 days' acute diarrhoea ..	++	—	—	—
10	30	3 " diarrhoea and steady epigastric pain	+++	+	—	—
11	31	3 days' diarrhoea. Also invalidated from S.E.A.C. area with fibroid phthisis	—	—	—	—
12	19	Slight bowel looseness and colicky epigastric and hypogastric pain	++	±	—	—
13	27	3 days' diarrhoea ..	+++	—	—	—
14	19	6 " severe diarrhoea and vomiting	+++	+	—	—
15	20	3 days' mild diarrhoea ..	±	±	—	—
16	33	Acute diarrhoea immediately after "Liverpool" course	±	±	—	—
17	22	2 days' diarrhoea and gripping abdominal pain	+	+	+	±
18	38	2 days' mild diarrhoea ..	++	+	—	—
19	20	24 hours' diarrhoea ..	±	—	±	—
20	20	36 " hyperacute diarrhoea ..	±	—	—	—
21	20	24 " mild diarrhoea ..	±	—	—	—
22	23	6 days' diarrhoea ..	—	—	—	—
23	24	5 " acute diarrhoea ..	++	±	±	—
24	21	3 " severe diarrhoea and gripping abdominal pain	—	—	—	—
25	23	7 days' mild diarrhoea ..	++	—	±	—
26	22	4 " severe diarrhoea ..	—	—	—	—
27	20	4 " mild diarrhoea and slight generalized hypogastric pain	+++	+	—	—
28	21	8 days' acute diarrhoea ..	++	+	—	—
29	24	11 " severe diarrhoea ..	—	—	—	—
30	19	7 " acute diarrhoea ..	+++	+	—	—

C.C.F.T. readings: 50% gave ++ or +++; 23.3% gave +++; 26.6% gave ++.

and found an increase of the coagulation band in all cases. The last two authors' results therefore do not agree with those of Heilig and Visveswar or with those obtained by using the C.C.F.T.

With regard to amoebic hepatitis, Heilig and Visveswar found that in 11 cases there was little evidence of impairment of function, whereas in all the 5 cases shown in Table IV the C.C.F.T. was positive. The literature gives conflicting reports on the value of liver-function tests in amoebic hepatitis. Hurst (1941) maintained that the laevulose-tolerance test always demonstrated hepatic deficiency in this condition, but Manson-Bahr (1940) and Strong (1942) both state that liver-function tests are disappointing. Brown and Hodgson (1938), using the bromsulphthalein retention test, found a reduction of the excretion of the dye in 8 of 13 cases of amoebic liver abscess.

In view of these conflicting reports and opinions it is impossible to draw any final conclusion on the value of any type of liver test in amoebic hepatic disease. It seems probable that any liver disease demonstrable in cases of intestinal amoebiasis is due primarily to secondary bacterial invaders and their toxins. This view is strengthened by the work of Hargreaves (1944-5) and Willmore (1944-5). They found that in chronic relapsing cases of amoebic dysentery, when repeated courses of anti-amoebic treatment had failed, an initial attack on the bacteria with penicillin and sulphasuxidine often led to dramatic symptomatic improvement. Hargreaves (1945) also points out that among the bacteria normally present in the bowel lumen are numerous penicillin-sensitive strains of staphylococci and streptococci, and that there is no evidence that either penicillin or sulphasuxidine is amoebicidal.

Consideration must now be given to the morbid histological picture to be expected in the liver in the absence

of frank abscess formation. Bartlett (1916-17), while investigating the dysentery epidemic among the Allied European troops in Gallipoli in 1915, conducted necropsies on the bodies of 30 individuals. Three may be excluded here, as liver abscesses were found. In the remaining 27 the following histological features were demonstrated in the livers: (1) Degenerative changes—i.e., cloudy swelling with a little fatty degeneration of the parenchyma of the central zones of the lobules; (2) necrotic areas infiltrated with a few neutrophil leucocytes; and (3) infiltration of the portal tracts associated with degenerative or necrotic lesions. Of these 27 cases, 17 were of amoebic aetiology and 10 had suffered from bacillary dysentery. In the amoebic cases the liver showed necrosis and portal infiltration in 2, degeneration and portal infiltration in 4, degenerative changes in 9, atrophy in 1, and normality in 1. The livers of the bacillary dysentery cases had necrosis and portal infiltration in 3, degenerative changes and portal infiltration in 5, and degenerative changes only in 2. The similarity of the microscopical picture in the liver in the two diseases is obvious, and this would appear to be additional evidence of the major part played by bacteria in damaging the liver in amoebiasis.

Bartlett's findings accord reasonably well with those of Palmer (1938), who conducted post-mortem examinations on the bodies of 19 patients in whose large bowels active amoebic lesions were demonstrated. Macroscopically the liver was larger than normal in 12 cases, normal in size and weight in 4, smaller than normal in 2, while in the nineteenth case no record was kept. Microscopically, 18 cases had increased portal connective tissue ranging from marked diffuse to moderate irregular or spotty increase. In 9 of the livers there was pronounced fibrosis and definite bile-duct proliferation, and in 9 there were fatty changes. Palmer's final conclusions were that there is "definite hepatitis associated with active amoebic lesions of the colon, and this is indicated by the generalized increase of portal connective tissue, by lymphocytic and mononuclear

TABLE II.—*Chronic Amoebic Colitis*

Case No.	Age	Presenting Symptoms	C.C.F.T.			
			1	2	3	4
1	36	Diarrhoea "off and on" for 2 years	—	—	—	—
2	30	Diarrhoea "off and on" for 2 months	—	±	—	—
3	21	2½ months' mild diarrhoea ..	—	—	—	—
4	27	18 days' slight bowel looseness	+++	+	—	—
5	25	Diarrhoea occasionally for 2 years; loss of weight and appetite	++	±	—	—
6	41	Occasional attacks of diarrhoea for a year	—	—	—	—
7	26	Bowel looseness for more than a year. Had inadequate anti-amoebic course a year before	++	—	—	—
8	28	Short attack of diarrhoea once a month for a year; loss of 14 lb. weight	++	—	—	—
9	24	Diarrhoea occasionally for over 2 years	—	—	—	—
10	22	Very slight bowel looseness for about 5 months	++	±	—	—
11	25	10 weeks' mild diarrhoea ..	—	—	—	—
12	21	Diarrhoea "off and on" for 3 months; some hypogastric pain	±	±	—	—
13	28	24 days' bowel looseness and tenesmus	++++	+	—	—
14	24	4 months' diarrhoea "off and on"	+	—	—	—
15	22	2 weeks' diarrhoea. Bowels very "unreliable and irregular" since treatment for amoebic dysentery 3 months before. ? relapse	+++	+++	—	—
16	23	6 months' occasional diarrhoea	++	+	±	—
17	21	7 " irregular diarrhoea	—	—	—	—
18	26	10 " early morning bowel looseness	—	—	—	—
19	23	2½ months' mild bowel looseness	+++	+	—	—

C.C.F.T. readings: 47.4% gave ++, +++, or ++++; 21.1% gave +++ or ++++; 26.3% gave ++.

infiltration, and by constant degenerative changes—for example, parenchymatous degeneration, fatty changes, haemosiderosis, and hepatic cell lysis."

Summary and Conclusions

In a series of 73 cases of amoebiasis (excluding those proved to have hepatic involvement) 37 (50.6%) gave evidence of damage to the liver parenchyma—that is, ++, +++, or ++++ flocculation in Hanger's C.C.F.T. The cases were divided on clinical grounds into three groups: (1) acute amoebic dysentery, (2) chronic amoebic colitis, and (3) amoebiasis presenting with unusual symptoms.

The 37 cases giving evidence of liver parenchyma damage were fairly evenly distributed among the three groups, and there was no great difference in the incidence of liver involvement between the groups.

The symptomatology and physical signs did not give any indication of the cases in which hepatic damage could be anticipated.

The institution of suitable anti-amoebic therapy produced a rapid recovery of the liver (as indicated by the C.C.F.T.).

A very small series of hepatic amoebiasis was investigated, but it was not considered justifiable to attempt to draw any conclusions from the results obtained.

The actual pathological changes present in the liver which can be assessed by the C.C.F.T. are probably generalized increase of portal connective tissue, lymphocytic infiltration, and degenerative changes—for example, parenchymatous degeneration, fatty changes, and hepatic cell lysis.

TABLE III.—*Amoebiasis Presenting with Unusual Symptoms*

Case No.	Age	Presenting Symptoms	C.C.F.T.			
			1	2	3	4
1	21	Upper abdominal pain with occasional vomiting after meals; suggestive of peptic ulceration	++	+	—	—
2	23	3 days' nausea and epigastric pain	—	—	—	—
3	19	6 days' epigastric pain	—	—	—	—
4	20	Appendix abscess treated by surgical draining	++++	+	±	—
5	29	Loss of weight, appetite, and energy. Cachexia case	±	+	±	—
6	32	Symptom-free. Cysts found in faeces 6 weeks after full "Liverpool" course	+	±	+	—
7	24	Treated as amoebic dysentery 4 weeks previously. Admitted with vomiting and right hypochondriac pain	+++	++	—	—
8	32	Upper abdominal pain. Diarrhoea 6 and 7 weeks before	—	—	—	—
9	25	Left chest and left shoulder pain	+++	±	±	—
10	21	Pain in left lower chest. No bowel symptoms at any time	—	—	—	—
11	22	Loss of weight and energy. Cachexia case	++	+	±	—
12	20	Weak and "worn-out" feeling. Cachexia case	++	±	±	—
13	21	Weak and "worn-out" feeling. Odd aches and pains	+++	±	+	—
14	19	Anorexia, nausea, and abdominal discomfort	+	+	—	—
15	22	Loss of weight, energy, and appetite. Cachexia case	±	+	±	—
16	18	Subacute appendicitis with vomiting, R.I.F. pain and tenderness	+	++	+	—
17	19	Gripping lower abdominal pain	++	±	+	—
18	24	Admitted on surgical side with anal fissure. Attack of diarrhoea 6 weeks before	+++	—	—	—
19	22	Vague dyspepsia and abdominal discomfort	++	++	—	—
20	23	Lack of energy and anorexia. One attack of diarrhoea 10 months before. Cachexia case	+++	+	—	—
21	24	Hypogastric pain. No history of bowel trouble	—	—	—	—
22	20	Upper abdominal pain after meals, suggestive of peptic ulceration	—	—	—	—
23	29	R.I.F. pain and tenderness and abdominal discomfort; nausea	++++	+	±	—
24	30	Symptoms suggestive of a mild attack of malaria; bowel looseness a year before	++++	++	—	—

C.C.F.T. readings: 54.1% gave ++, +++, or ++++; 33.3% gave +++ or ++++; 20.8% gave ++.

TABLE IV.—*Amoebic Hepatitis*

Case No.	Age	Symptoms and Physical Signs	C.C.F.T.			
			1	2	3	4
1	20	Right-sided thoracic pain. No history of diarrhoea. Deficient air entry at right lung base. Liver not palpable. Irregular fever to 101° F. (38.3° C.). Leucocytosis to 11,600/c.mm. Radiograph showed right dome of diaphragm fixed and raised	++	—	—	±
2	26	Admitted with diagnosis of "clinical malaria." No history of diarrhoea. Fever to 103.2° F. (39.55° C.), with rigors and sweats. Liver palpable 2 in. (5 cm.) below right costal margin. Leucocytosis to 13,400/c.mm. Radiograph showed no abnormality	+++	—	±	—
3	24	Admitted with vomiting and right hypochondriac pain. Bout of diarrhoea 4 weeks before. Liver palpable 1 in. (2.5 cm.) below right costal margin. Leucocytosis to 9,900/c.mm. Low fever to 99–100° F. (37.2–37.8° C.). Radiograph showed right cupola of diaphragm fixed and raised	++++	++	—	—
4	20	? Malaria. Shivering, sweating, anorexia, and abdominal discomfort. Fever to 103° F. (39.4° C.). Liver palpable 1 in. (1.25 cm.) below right costal margin. Leucocytosis absent. Radiograph showed partial collapse of base of right lung	+	±	±	—
5	37	10 days' diarrhoea and pain in right chest. Tender in right hypochondrium. Liver palpable 1½ in. (3.75 cm.) below right costal margin. Fever to 102.4° F. (39.1° C.). Leucocytosis to 19,800/c.mm. Radiograph showed right cupola of diaphragm fixed. Possibly an element of alcoholic cirrhosis	++++	++++	+++	++

It is suggested that secondary bacterial invaders and their toxins are primarily responsible for these changes in the liver in cases of amoebiasis.

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