presence of tympanic injection, otitis media, and abnormal physical signs in the chest (notably rhonchi and/or crepitations). Table IV shows the incidence of these complications, which are few in number, and no significant differences emerge.

TABLE IV.—*Incidence of "Complications" as Observed at the First Visit, on the Fourth Day Thereafter, or on Both Days*

	No. of Patients								
	First Visit			Second Visit (Fourth Day)			Both Visits		
	O.M.	T.I.	R.S.	O.M.	T.I.	R.S.	O.M.	T.I.	R.S.
Penicillin	1	10	7	0	4	8	0	5	3
Virugon	0	8	7	1	9	5	3	1	2

O.M.=Otitis media. T.I.=Tympanic injection. R.S.=Respiratory signs.

Conclusions

There are no differences between the two groups on the basis of the comparisons noted above, which confirmed the clinical impressions gained. The number of patients with complications is very small, and it might be that a study of a much larger number of cases would reveal subtle differences. The College of General Practitioners (1957) has suggested that the use of antibiotics produces a small reduction in the incidence of complications but that their use was not justified.

No significant differences were found between virugon and penicillin V in the treatment of 200 cases of measles.

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Preliminary Communications

Definitive Diagnosis of Amoebic Liver Abscess: Value of Liver Biopsy

[WITH SPECIAL PLATE]

In the diagnosis of a parasitic infection it is clearly desirable to recover and identify the causative agent. Necropsy studies of amoebic liver abscesses have shown that the amoebae are to be found in the wall of the abscess rather than in its contents or in the surrounding liver tissue (Craig, 1944). Amoebae have also been demonstrated in three out of five biopsy specimens of the liver which inadvertently included the wall of an abscess (Kean, 1955). Accordingly an attempt has been made to demonstrate amoebae by deliberate biopsy of the wall of the abscess.

MATERIAL AND METHODS

Ten consecutive cases of liver abscess were studied. The patients' ages ranged from 22 to 70 years. The duration of symptoms referable to the abscess varied from three days to four months. The criteria for diagnosis were similar to those of Lamont and Pooler (1958). The response to emetine hydrochloride and chloroquine in nine cases suggested the likelihood of an amoebic aetiology. In none of the cases were amoebae detected in the stools.

Initially a pleural aspiration needle was introduced at the most tender spot, or directly into an obvious mass if present; the approach was subcostal in four instances and via the intercostal route in seven. The position of the abscess and its depth from the surface were established by the withdrawal of pus. Biopsy of the abscess wall, using a Vim–Silverman needle, was then performed. Latterly the abscess has been located with the biopsy needle. This outer needle has been withdrawn until pus could no longer be aspirated; the split inner needle has then been introduced and a biopsy taken in the usual way. Failure to obtain a specimen occurred only once in 12 attempts.

When a biopsy core was received in the laboratory it was fixed in formol-mercury, processed, and embedded in paraffin wax. Three sections were cut at each of three different levels, at a thickness of 5 μ. One section of each level was stained with haematoxylin and eosin and one by the McManus periodic-acid–Schiff method.

When minute fragments were received they were transferred to a conical centrifuge tube and the entire processing was carried out manually. When the dehydrating and clearing fluids were decanted the small fragments were tightly packed at the bottom of the container. The specimen was centrifuged before each change. After clearing, hot filtered wax was poured into the tube, which was then placed in an incubator at 56°–58° C. for four to five hours. The tube was then removed and the wax allowed to solidify while standing upright. With care the wax mass containing the fragments was removed, sectioned, and stained as described above.

RESULTS

In seven cases amoebae were seen. They were always found in necrotic liver tissue, which in addition contained inflammatory cells and red blood corpuscles (Special Plate, Figs. 1 and 2). In four cases fibrous tissue was present (Special Plate, Fig. 3) and in three cases hepatic parenchyma was seen. In these three cases the findings in the liver tissue were non-specific and consisted in mild to moderate lymphocytic infiltration of the portal tracts and some increased portal fibrosis.

In one patient in whom the abscess was probably pyogenic a remittent fever subsided on antibiotic treatment before 5 ml. of thick yellow pus was aspirated. Amoebae were not demonstrable in this case.

COMMENT

Amoebae are seldom found in the contents of a presumed amoebic abscess (Sodeman and Lewis, 1945). The demonstration of amoebae by Elsdon-Dew and his co-workers in pus from 84% of 75 liver abscesses (Maddison *et al.*, 1959) is quite exceptional. Such impressive results demand time and facilities not available to a routine laboratory. The macroscopic appearance of the contents may be purulent or sanguineo-purulent rather than "anchovy-sauce" (Lamont and Pooler, 1958). In the differential diagnosis of a liver abscess the importance of recognizing the solitary pyogenic abscess has been emphasized both in areas where amoebiasis is endemic (McFadzean *et al.*, 1953) and those where it is sporadic (Stokes, 1960).

Despite the theoretical possibilities of contamination of pleura or peritoneum, immediate or delayed untoward effects were not observed. Though the series is admittedly limited, the risk would seem to be no greater

than that of conventional diagnostic or therapeutic needle aspiration. It is, however, emphasized that the method is recommended only to those who are thoroughly familiar with these procedures.

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Medical Memoranda

Glandular Fever Encephalitis

Involvement of the nervous system in glandular fever was first described by Johansen in 1931. Only about 1% of cases develop this complication, but frequent reports have shown that a wide variety of neurological lesions may occur (Bergin, 1960). Serous meningitis and polyneuritis are two of the commoner manifestations, while permanent paralyses and death from respiratory failure are on record (Erwin *et al.*, 1959). About 60 cases of encephalitis have been reported, many of which also showed a meningeal reaction. The mortality rate in cases with neurological involvement is estimated at between 15 and 40% (Erwin *et al.*, 1959), and in one series 43% of deaths from glandular fever were due to this cause (Lawrence, 1951). Corticosteroid therapy has been used with apparent benefit in these serious cases (Fiese *et al.*, 1953; Hughes and Paulley, 1960; Stillerman and Peltz, 1960). Below is described a case of severe encephalitis in which the patient recovered suddenly without such treatment.

CASE REPORT

On April 27, 1961, a 16-year-old girl had an uncomplicated dental extraction under local anaesthesia. At school a week later she was more than usually talkative, and spoke oddly. Next evening she complained of headache, nausea, and pain round the costal margins. By morning she was febrile—temperature 100.8° F. (38.2° C.)—drowsy, and unable to remember the date of her birthday. She vomited twice and was admitted to hospital on the same evening with a diagnosis of meningitis. By then she was irritable and uncooperative, with a temperature of 103° F. (39.4° C), photophobia, and slight neck stiffness.

Investigations.—Haemoglobin 86%; W.B.C. 6,000/c.mm. (polymorphs 60%, lymphocytes 36%, monocytes 4%); blood sugar 92 mg./100 ml. C.S.F.: protein 60 mg./100 ml.; sugar 80 mg./100 ml.; chloride 690 mg./100 ml.; erythrocytes 100/c.mm.; leucocytes less than 1/c.mm. Urine: no albumin or glucose. Chest *x*-ray examination revealed nothing abnormal.

During the night she was restless, but next morning her unresponsiveness almost indicated hysteria. However, her temperature was 100° F. (37.8° C.), her arm movements were at times athetoid, and twitching of her facial muscles was seen. An hour later she gave an epileptic cry and had a major convulsion. Generalized fits continued throughout the day in spite of large doses of phenobarbitone sodium and paraldehyde. In the evening she was comatose and began to overbreathe so vigorously that carpal spasm was continuous. As signs of pneumonia and ketosis were absent, a diagnosis of aspirin poisoning was considered. The serum electrolyte and blood-urea levels were, however, normal. Her temperature rose to 106.8° F. (41.6° C.) and pulse rate to 196, and tepid sponging was started. During the next few hours the hyperpyrexia and hyperventilation subsided. By morning her temperature was 99° F. (37.2° C.) and respiration rate 25. She was unrousable, although the corneal reflex was present. Her arms were rigidly flexed across her body as if she was decerebrate; her legs were extended but lacked tone, and only the right plantar reflex was extensor. Next morning, although very drowsy, she was conscious, afebrile, and able to drink and talk intelligibly. For the next few days she was weak and euphoric, but recovered completely and left hospital on May 23.

Stool virus cultures and the mumps complement-fixation test were negative, but on May 12 atypical mononuclear cells of the glandular fever type were seen in a routine blood count (W.B.C. 8,000/c.mm. (neutrophils 32%, eosinophils 2%, lymphocytes 55%, monocytes 11%)). As a result of this observation a Paul–Bunnell test was carried out on May 15. This gave a positive result for glandular fever: agglutination to a titre of 1 in 256, reduced to 1 in 128 after absorption with guinea-pig kidney and to zero after absorption with ox red cells. The test was negative when repeated on May 23. No rash, pharyngitis, or glandular or splenic enlargement was detected during the illness.

Comment.—The progression of this alarming ailment confirmed an initially insecure diagnosis of encephalitis. Coxsackie infection was at first suspected in view of the chest pain, and the possibility of glandular fever was not considered. Personality change in the early stages has, however, been a feature of previous cases (Ream and Hessing, 1954; Bergin, 1960), and a few patients (three of them Italian) have presented with psychoses (Raymond and Williams, 1948; Bellinato, 1960). The hyperpyrexia and overbreathing presumably resulted from damage to the hypothalamus, since "central" hyperventilation sometimes follows trauma in this region (L. Walshe, 1961, personal communication). Ream and Hessing (1954) noted Cheyne–Stokes breathing in their patient, and decerebrate rigidity preceded death in the case described by Bergin (1960), which was very similar to the present one.

DISCUSSION

The neurological complications of glandular fever often develop in the second or third week of illness (Leibowitz, 1953). However, they may precede the usual signs, which are often minimal or, as in this case, absent (Erwin *et al.*, 1959). As the Paul–Bunnell test is usually positive in cases with complications (Evans, 1960), it should be performed routinely in patients with meningoencephalitis of doubtful origin. The test is not

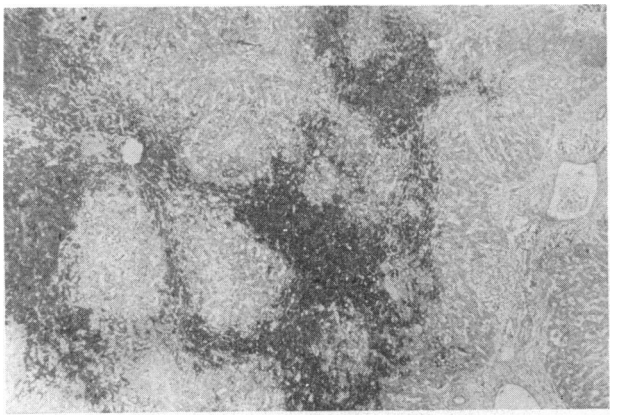

FIG. 1.—Section of liver showing intense centrilobular congestion. (H. and E. ×22.)

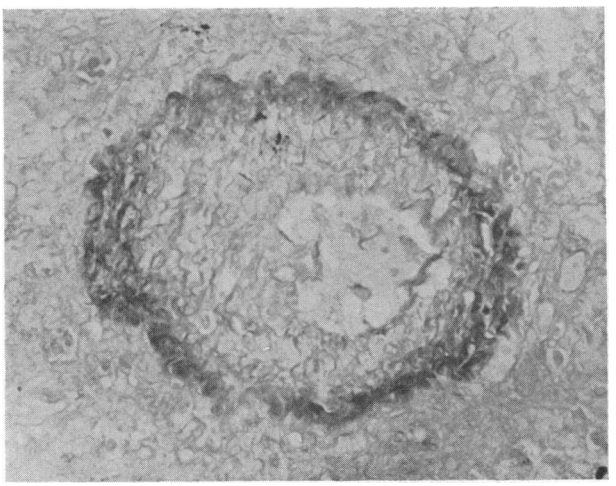

FIG. 2.—High-power view of a central vein showing fibrous endophlebitis. (Elastic van Gieson. ×200.)

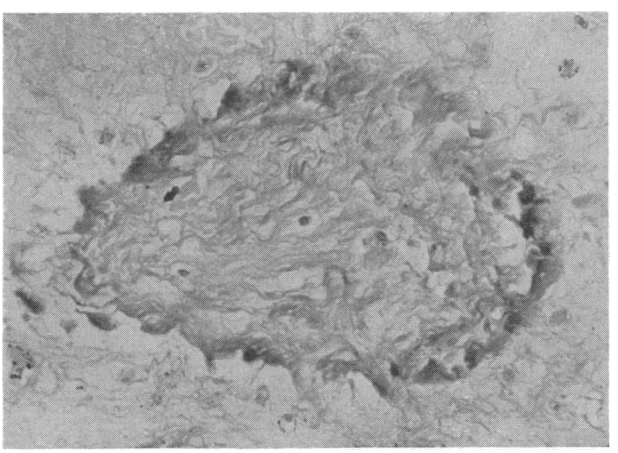

FIG. 3.—Central vein almost completely occluded. (Elastic van Gieson. ×288.)

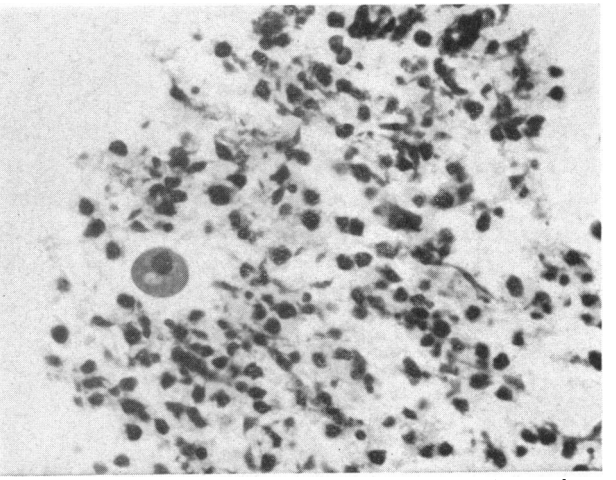

FIG. 1.—Section of necrotic tissue showing an amoeba, nuclear remains, lymphocytes, and occasional polymorphonuclear leucocytes. (H. and E. ×495.)

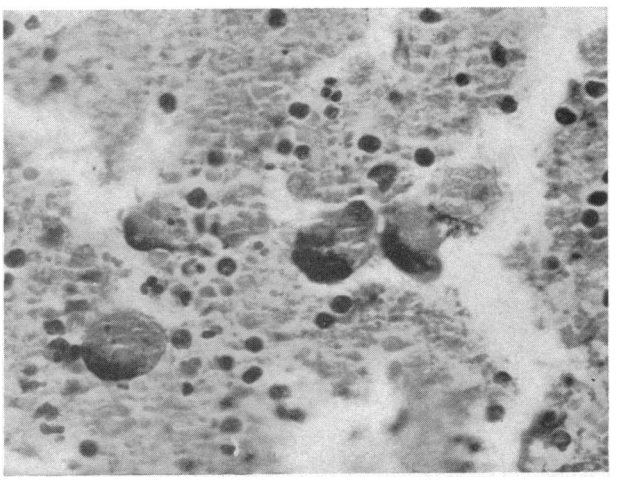

FIG. 2.—Section of necrotic liver tissue showing several vegetative forms of *Entamoeba histolytica*. A few contain phagocytosed red blood corpuscles. (P.A.S. ×495.)

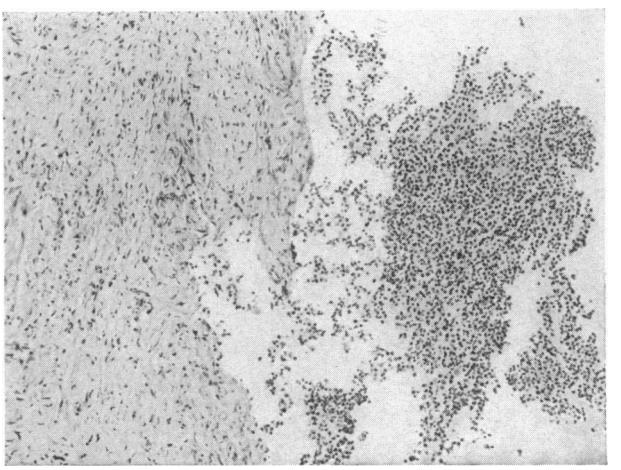

FIG. 3.—Low-power view of biopsy specimen showing fibrous tissue on left and necrotic tissue on right. (H. and E. ×75.)