Histological Examination.—The liver showed a varying picture of acute damage, fibrous proliferation, and regeneration: (1) large areas of autolysed necrotic hepatic parenchyma; (2) islets of surviving epithelial tissue disposed in irregular dislocated columns, and with the peripheral cells often necrotic or autolysed; (3) areas with absorption of dead liver tissue revealing collapsed sinusoids with zones of congestion, haemorrhage, and round-cell infiltration; and (4) groups of small islets of liver parenchyma surrounded by bands of young connective tissue containing many poorly formed bile ducts. Almost the entire right lobe showed acute damage, contrasting with the damage in the left lobe, which was mainly subacute. There was nothing of note in the spleen and kidneys. The pancreas showed minute areas of necrosis in the pancreatic fat, but there was no abnormality in the parenchyma or ducts of the gland.

Discussion

The clinical and pathological findings (apart from the endocarditis) are similar to those that have been described in America in chloronaphthalene poisoning. The patient did not have any chloracne, although this appeared in other workers in the same plant: no correlation has been found between the skin and the liver lesions (Mayers and Silverberg, 1938). Illness occurred in which jaundice eventually developed, but was never severe until the patient was near death. Greenburg, Mayers, and Smith (1939) record similar histories: their 3 fatal cases were treated at home (for catarrhal jaundice) for periods of from 6 weeks to 5 months; one patient continued to work for 5 months from the onset of the jaundice, and the necessity for hospital treatment was not realized until convulsions developed.

The liver findings are consistent with the length of the history; the histological picture is not of extensive liver necrosis of simultaneous production but of recurrent liver damage taking place over a short period. Thus recent necrosis occurs side by side with various stages of repair represented by fibrous proliferation and regeneration. The areas of recent damage are much more extensive than those in which repair is taking place and regeneration is not marked. Two of Greenburg's cases appear to have suffered at least one previous attack of acute hepatitis followed by some improvement before the onset of the fatal attack. In addition to the liver damage Greenburg et al. record more severe pancreatic damage than in the present case, in which it was confined to minute areas of fat necrosis. These changes are thought to result from the liver necrosis and not from the effects of the toxic agent. The retroperitoneal oedema found was probably the result of giving intravenous fluids in large quantities to a subject with cardiac decompensation.

In America no predisposing factor in the affected workers has been discovered. In the present case the endocarditis, with development of breathlessness, and oedema of the feet, which disappeared on rest, suggest cardiac decompensation as a predisposing factor, especially since in the latter the liver undergoes nutritional and vascular changes. It is possible, however, that liver damage initiated the decompensation, which clinically was not severe, while the valvular lesions were of moderate grade. The puffiness around the eyes is more indicative of oedema due to a cause other than cardiac.

Drinker et al. (1937) have shown that animals when exposed to low concentrations of the chloronaphthalenes may incur liver damage without presenting any clinical evidence. Furthermore, when animals of this group that give no evidence of liver damage are exposed to small doses of carbon tetrachloride, well tolerated by controls, they promptly die from acute hepatic necrosis, often with marked jaundice. This raises the questionwhether the chloronaphthalenes may only be one of two factors in the production of liver damage. Accordingly, Greenburg et al. (1939) suggest that neither anyone who at any time in the past has had any liver disease nor anyone with a history of typhoid, malaria, gall-stones, or other diseases known to affect the liver adversely should work with those substances. Likewise, persons receiving hepatotoxic drugs (e.g., arsenobenzol) should not be exposed to chloronaphthalenes, nor should chloroform, trichlorethylene, or avertin anaesthesia be given to chloronaphthalene workers. The present case may suggest that no one with organic heart disease should be allowed to work with the compounds.

It is unfortunate that the serious nature of the patient's illness was not realized earlier—a comment which applies to every

case so far reported. This error would be eradicated if these workers were instructed in the peculiar hazard of their occupation, for not all of them have knowledge of the technical process and the chemicals involved, and probably few medical practitioners know of the chloronaphthalenes as hepatotoxic agents. Since experimentally a certain amount of liver damage may occur without visible sign, it is clear that workers in chloronaphthalenes should receive special attention on the least indication of ill-health apart from the jaundice. Furthermore, since the concentration of chloronaphthalenes in the air of the workshop in the present case was sufficient ultimately to produce fatal hepatic necrosis in one worker who presumably had a special predisposing factor, it is possible that recurrent subclinical liver damage may be occurring in others, leading to cirrhosis.

Summary

A case is described of fatal liver necrosis due to poisoning by chloronaphthalene. This substance is now extensively used in industry for insulating wire and electrical apparatus, and there is risk of its being absorbed by the skin or by inhalation of fumes. There is evidence, from cases described abroad, that the onset of the poisoning may be insidious: slight jaundice may appear unaccompanied by more clamant symptoms which would induce the individual to cease work. In the present instance the subject had been at risk of exposure for 6 months before the onset of any illness, but, though the first symptoms were very mild, death occurred 8 weeks later. The case was complicated by the existence of rheumatic cardiac disease.

We are indebted to Dr. A. N. Currie, H.M. Medical Inspector of Factories, Glasgow, for industrial data, and to Prof. J. Shaw Dunn and Dr. W. R. Snodgrass for advice and permission to publish the case. One of us (N. McL.) is indebted to the Rankin Medical Research Fund, University of Glasgow, for a grant to defray expenses of investigation.

REFERENCES

A COMPARISON OF DYSPEPSIA IN THE ARMY FOR 1940 AND 1941

BY

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It was hoped that a comparison of data of dyspepsia in the Army for 1941 with those for 1940 might throw some further light on the subject; but there are so many variable factors bearing upon the question for which corrections cannot be made that it is impossible to draw many conclusions.

In 1940 8,702 medical cases were seen at the hospital, and of these 1,270 (14.6%) were suffering from dyspepsia. In 1941 5,606 cases were seen-2,000 less than in 1940-and of these 974 (17.4%) were dyspeptic. The drop in the total number of cases is because Dominion hospitals have claimed their own patients. A correction should perhaps be made as 76 cases were admitted to the gastric ward direct from near-by hospitals. These cases, however, were unselected and were in different stages of investigation and treatment, many of them undiagnosed. I have included them in the results. The patients were composed of Army Class 38%, Volunteers 30%, Reservists 16%, and Regulars and Territorials 8% each. These figures differ widely from those for 1940, but they are, I think, within reasonable expectations when one considers the class of troops in the area. There may be a reduction in the number of Reservists affected during 1941, but it must be remembered that many of this class had already been discharged in 1940. Actually only 99 (10%) of the cases in this series developed symptoms for the first time after joining the Army. In 17 of these the trouble was really anxiety neurosis.

The following notes refer only to patients who came directly under my care, either as out-patients or in the gastric ward. There were 390 out-patients and 367 in-patients, a total of 757. The remaining 217 cases were seen by the other specialists.

Table showing Details of Results

	Radiograph			Totals	Totals
to the Co.	+	_	Not Taken	1941	1940
Ulcers: Duodenal Gastric Jejunal Gastro-duodenal	168 72 4	10 3	36 17 —	310 (43%) 214 92 4	391 (55%) 282 103 6
dyspepsia: Pylorospasm Irritable stomach Hyperchlorhydria Chronic gastritis Carcinoma of stomach Achalasia of cardia Reflex dyspepsia: Neurotic dyspepsia Colon Ch. appendix Gall-bladder Various	36 36 —————————————————————————————————	26 11 105 — 60 — 6		218 (30%) 36 36 26 11 105 3 197 (27%) 131 30 18 12 6	254 (36%) 47 27 32 14 132 1 62 (9%) 40 55 9 3 5

In addition, during 1941 there were 32 cases of acute gastritis; but they are not relevant to the subject, and will not be referred to again.

The falling off in numbers during 1941 is probably due to the fact that there were fewer British soldiers in the area, and to the absence of any severe epidemic. Without knowing the average number of troops in the region served by the hospital a comparison of actual totals for the two years is unreliable, and it is necessary to fall back upon percentages.

Increase in Neurotic Dyspepsia

In 1941 there was an increase of 2.8% in the number of dyspepsia cases of all types. This increase was produced entirely in the group of reflex dyspepsia. All the cases in which an anxiety neurosis was the predominant symptom are classed as neurotic dyspepsia; many of them showed pylorospasm or irritable stomach when radiographed; and hyperchlorhydria, often of a high degree, was common. But wherever these findings have been subordinate to the neurosis the cases have been placed in the neurotic dyspepsia group.

Reflex dyspepsia in 1941 made up 27% of the whole, as compared with 9% in 1940. The greatest increase was in neurotic dyspepsia, which rose from 5.6% (40 cases) to 18% (131 cases). This increase in neurosis was very definite, and was not confined to dyspepsia alone. But the curious part about these neuroses is that in all but 17 patients they originated before the war began and before the men were called into the Army. Those people of poor personality who in peacetime are only just able to accommodate themselves to their home environment are no longer able to do so when this is changed on enlistment to the discipline of Army environment. Whether it is pure chance that their neurosis is centred on their digestion it is difficult to say, but in my opinion a constitutional weakness is inherent in them.

Decrease in Organic Dyspepsia

In contrast with neurotic dyspepsia, organic dyspepsia decreased. The percentage of peptic ulceration fell from 55 to 43. However, on looking into this group very little change is observed in the relative frequency of the different types of ulcer or their locality. Duodenal ulcer, with which is included duodenitis, fell by 3%. Gastric ulcer was below that for 1940. In the stomach itself the location of the ulcer was remarkably constant, the ratio being 5 ulcers in the body of the stomach to 3 at the pylorus. Duodenal ulcer is still twice as common as gastric ulcer in the Army. Of the ulcer cases, 33 had perforated in the past, 35 had had a severe haemorrhage, either haematernesis or melaena, and 20 had undergone some remedial operation on the stomach or duodenum. A few of the patients stated that they had had a short-circuit done, but x-ray examination did not reveal any trace of interference.

Gastro-duodenal dyspepsia other than peptic ulceration also decreased in 1941, falling from 36% to 30%. Pylorospasm and irritable or hypertonic stomach, as seen radiographically, is usually only one stage in a larger process. Thus it may be a sign of early hidden ulceration or of healing recent ulcer, or it may be a reflex from some other part of the body. It was common with spastic colon, and also with neurosis. It was a frequent finding, but, wherever possible, it has been included with the larger group, such as peptic ulcer or reflex dyspepsia.

Chronic gastritis cases still comprised a large group, mostly seen as out-patients, investigations being restricted to a barium meal. In civil life the majority had been accustomed to attacks of indigestion from time to time, but had carried on with their work, losing a day here and there, and dieting from time to time. These patients were not fully investigated, as the main object was to keep them at work whenever possible. Only 43 of a total of 607 dyspeptic out-patients were actually admitted for fuller investigation.

To sum up, it may be said that, whereas there has been a slight fall in the frequency of ulceration, there has been a small increase in dyspepsia. This increase is due to neurotic dyspepsia.

Disposal of Dyspeptic Patients

There is still no uniformity in the disposal of dyspeptic patients. Should they be sent to a convalescent home or depot, or back to duty with their units? It has been a working rule at this hospital to put up all cases of peptic ulcer, haemorrhage, or operation for boarding out of the Army. To these are added a few chronic dyspeptics and those neurotic dyspeptics who are deemed by the psychiatrist to be useless as soldiers. The remainder are recommended for home service. There are, of course, exceptions in both directions. Each case has to be judged not only on the condition in the patient's stomach but also on his working conditions. Occasionally the Army conditions are better than the civilian ones would be. Only a few—the more satisfactory cases—are sent to a convalescent depot. The remainder are returned to their units.

Of the dyspepsia cases I saw at a convalescent depot, only 50% had been able to get on with Army food. They had had a few days' mild indigestion to begin with, but had settled down and were free of symptoms again. 25% had ulceration, haemorrhage, or operation, and should have been boarded out of the Army. The remaining 25% consisted of chronic cases, always in and out of hospital, and neurotics. None of this last group could ever become fit men, and therefore should not have been sent to a convalescent depot.

In the case of the neurotic, it is essential that he should be got out of hospital as early as possible after the completion of his investigation, and, furthermore, he should not be allowed back into hospital again. It is extremely bad for him to be allowed to drift from hospital to hospital, collecting radiographs and test meals at each. For this is his greatest delight, and all too frequently he is encouraged in it.

Summary

The figures of dyspepsia at a "static" hospital are compared for 1940 and 1941.

An increase for dyspepsia of all types of 2.8% was found. Neurotic dyspepsia was responsible for this increase, having risen by 12.4%. Peptic ulceration fell by 12%, and gastroduodenal dyspepsia by 6%. Duodenal ulcer was twice as common as gastric ulcer, and ulcer in the body of the stomach was in the ratio of 5:3 to pyloric ulcer.

The disposal of patients is discussed.

G. S. Barrett, C. H. Rammelkamp, and J. Worcester (Amer. J. Dis. Child., 1942, 63, 41) record two cases of meningitis due to B. coli, in a girl aged 6 years and a boy aged 2 weeks, who recovered under treatment with sulphanilamide and its derivatives. This form of meningitis is commonest in infants under 3 months of age, and without chemotherapy is fatal in about 80%. The portal of entry may be obscure, but associated infections of the middle ear, urinary tract, and umbilicus may be present. Bacteriaemia has been found in a number of cases. The course may be protracted, with relapses and the occurrence of hydrocephalus. Whole-blood transfusions in infants under 2 years may aid in the treatment.