per c.mm. If this fails to produce complete regression of lymph nodes an additional course of treatment is given six weeks later. To prevent relapses a supplementary (prophylactic) course of injections of shorter duration after an interval of two to three months has been found useful.

Treatment with chloroethylamines should be given not only in the advanced stage of the disease when x-ray therapy has proved unsuccessful, but also in the early stages.

With such treatment, provided it is given in the early stages and according to a rational method, positive remote results—that is, preservation of life and working capacity for more than five years from the beginning of treatment—may be obtained in 50% of cases.

The immediate and late results of chloroethylamine treatment of early cases of Hodgkin's disease are at least as good as those of x-ray therapy.

The above two treatments are applicable in combination, the following two methods having been found useful in our hands: (1) an initial course of chemotherapy is given, and this is followed after an interval of six to eight weeks by x-irradiation of nodes which have not completely regressed; or (2) the two methods of treatment are applied alternately in subsequent relapses.

A new drug—2:6-dioxy-4-methyl-5-(2-chloroethyl) aminopyrimidine (dopan)—has been developed which, as it can be administered orally and has only a slightly toxic action on the gastro-intestinal tract, renders the chemotherapy of Hodgkin's disease more convenient for the patient, one tablet being given twice weekly for three to five weeks.

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The Minister of Health, Mr. R. H. TURTON, has written to hospitals to ask them to re-examine any remaining restrictions on the daily visiting of children in hospital. His letter is based on advice given by the Standing Medical Advisory Committee and endorsed by the Central Health Services Council on a number of difficulties shown by a review in 1954 to be the main obstacles to the universal acceptance of daily visiting. One of the main problems concerns infectious diseases hospitals. The Minister is advised that subject to certain safeguards frequent visiting can be allowed and, indeed, is already allowed in a number of such hospitals. Safeguards suggested include the presence of sufficient nursing staff to ensure close supervision of the conduct of visiting parents, the wearing of protective clothing by visitors, and the application to them of the routine measures for the prevention of the spread of infection. It is suggested that if visitors are to wear masks and gowns they should, where possible, be put on in the child's sight. Some hospitals which do not allow daily visiting give as the reason the undesirability of admitting visitors on operating days. The Minister is advised, however, that there is no general reason why all visiting on these days should be prohibited.

EFFECTS OF ALCOHOL ON GASTRIC MUCOSA

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The excessive consumption of drinks containing ethyl alcohol is generally believed to induce acute or chronic gastritis, the latter often being described as "atrophic"; moreover, many clinicians attribute the dyspeptic manifestations of alcoholism to such inflammation. Theoretically, gastritis is a reasonable expectation, and there is reliable evidence that it does occur in alcoholic subjects. However, in a recent personal histological study of gastric mucosa from such subjects inflammatory lesions were notably slight or entirely absent, even in some inveterate spirit-drinkers. This observation, together with the paucity of histological data referring to the effects of alcohol on the gastric mucosa, suggested the need of a re-evaluation of current opinion. Impressions based only on clinical findings, including gastroscopic and radiological observations, are not always accurate. It is only by the histological examination of fresh gastric mucosa that a true assessment of structural appearances can be made. The problem is clearly of practical importance when it is realized that over £800,000,000 is spent annually in Great Britain and Northern Ireland on alcoholic drinks.

In an attempt to obtain information on the effect of alcohol on the stomach mucosa, a series of biopsy, gastrectomy, and necropsy specimens from alcoholic subjects were examined. In addition, a detailed study was made of the effects of alcohol on the guinea-pig stomach. These investigations and the results obtained are now described.

Human Gastric Mucosa after Alcoholic Excess

An examination was made of gastric mucosa from 25 adults with a history of alcoholic excess. Sixteen of these patients were admitted to hospital for full investigation of vague symptoms apparently related to alcoholism, and gastric biopsy was performed on them, using a modified Australian flexible tube (Coghill and Wynn Williams, 1955). In addition, the portion of stomach from three patients who underwent partial gastrectomy for chronic gastric or duodenal ulcer and six stomach specimens obtained at necropsy were examined; the latter were fixed with formol-saline within one hour of death.

All the tissues were fixed in 10% formol-saline, and paraffin sections were stained routinely with haematoxylin and eosin. Where additional information was required, sections were stained with Southgate's mucicarmine, Motteram's trichrome stain for gastric mucosa, and Masson's trichrome stain.

The pathological changes seen included one or more of the following: loss of glandular elements; "intestinal metaplasia"; replacement of body glands by glands of pyloric type; hyperplasia and nuclear hyperchromatism of epithelium at the mucosal surface; excessive numbers of small round cells, plasma cells, or polymorphonuclear leucocytes in the stroma; oedema; hyperaemia; haemorrhage; fibrosis; erosion; ulceration. The commonest of these were chronic inflammatory changes without specific characters and often mild in degree.

Biopsy Specimens

Two specimens of mucosa were removed from the body of the stomach in each of 16 patients. One patient was a

woman of 45; the others were men between 20 and 68, and averaging 46 years of age.

In seven patients the microscopical appearances of the mucosa were normal. Examples:

Man aged 20 with intermittent epigastric discomfort for eight months and a history of heavy drinking for six months. Radiographic appearances and gastric juice normal. Gastric biopsy: normal mucosa.

Woman aged 45 with delirium tremens and a history of heavy drinking in the past 15 years. Liver and spleen enlarged. Gastric biopsy: normal.

Man aged 48 with long history of alcoholic excess and enlargement of liver. Gastric biopsy: normal.

In five patients the stomach mucosa presented the histological features of chronic non-specific inflammation, mostly mild in degree and without glandular atrophy. Examples:

A bar-tender aged 35 drank an average of 70 bottles of beer and two bottles of gin a week and complained of morning sickness and occasional vomiting for eight months. Gastric biopsy: an excess of chronic inflammatory small round cells was present in the superficial part of the mucosa; in addition, the surface epithelium was flatter than normal, suggestive of regeneration following superficial erosion.

Man aged 49 with alcoholic cirrhosis and peripheral neuritis. Gastric biopsy: mild chronic inflammatory changes only.

In the remaining four patients the histological findings were those of chronic atrophic gastritis:

Man aged 42 with dyspepsia for 14 months and demonstrable gastric ulcer. Drinks up to 8 pints (4.5 litres) of beer each evening, sometimes wine as well. Gastric biopsy: chronic atrophic gastritis. No intestinal metaplasia.

Man aged 55 with alcoholic cirrhosis, histamine-fast achlorhydria and diabetes mellitus. Gastric biopsy: chronic gastritis with some intestinal metaplasia.

Chronic alcoholic aged 59 with peripheral neuritis and proved gastric ulcer. Free HCl present in gastric juice. Gastric biopsy: chronic atrophic gastritis with marked intestinal metaplasia.

Chronic alcoholic aged 60 with marked anorexia, vomiting, and weakness, some diarrhoea and hypochromic anaemia. Gastric biopsy: chronic atrophic gastritis with some intestinal metaplasia.

Partial Gastrectomy Specimens

Partial gastrectomy was performed in the following three cases.

Man aged 35 with a history of fairly severe alcoholic excess and upper abdominal discomfort for four years. Partial gastrectomy performed for duodenal ulceration. A brother also had a duodenal ulcer. Stomach specimen: normal mucosa.

A salesman aged 48 with an eight-year-old history of gastric ulcer drank spirits to excess. His mother was suspected of having a peptic ulcer. Stomach specimen: a large chronic benign ulcer was present on the lesser curve 4 cm. from the pyloric ring and there were two small acute superficial erosions on the anterior wall of the body. Mild chronic gastritis was present in the mucosa generally, but there was no intestinal metaplasia; more conspicuous was widespread hyperplasia of surface epithelium; some disappearance of body glands was also seen.

A slaughterman aged 51 with a history of severe alcoholic excess had also had epigastric pain for 12 years. Perforation of a duodenal ulcer occurred two years previously. Partial gastrectomy performed for chronic duodenal ulcer. Stomach specimen: mild chronic gastritis was present in the body mucosa and moderately severe chronic gastritis in the distal mucosa, with some intestinal metaplasia in the pyloric canal.

Necropsy Specimens

In the following six cases stomach specimens were obtained at necropsy.

A ship's steward aged 33, who had been drinking heavily, including five bottles of rum a week, for four weeks and died with severe weakness, cyanosis, burning epigastric discomfort, and haematemesis of two days' duration. Necropsy findings were pulmonary sarcoidosis and fatty liver, kidneys, and heart. Stomach: mucosa normal except for multiple small haemorrhages and haemorrhagic erosions, also a healing pyloric erosion.

Man aged 37 who died of carcinoma of the pancreas with obstructive jaundice and terminal haematemesis. There was a

history of severe chronic alcoholism. Stomach: several minute haemorrhagic erosions in the mucosa of the distal part, also mild to moderately severe chronic gastritis.

Man aged 49 admitted with delirium tremens and died suddenly two days later. There was a history of being constantly drunk. The only notable necropsy finding was considerable fatty change in the liver. Stomach: normal.

Man aged 66, a confirmed spirit-drinker, who died with carcinoma of bronchus and congestive cardiac failure. Stomach: two large shallow acute haemorrhagic ulcers present in the pylorus and scattered haemorrhagic erosions elsewhere, also mild chronic gastritis.

Man aged 67 chronically addicted to whisky, died with carcinoma of pancreas, obstructive jaundice, and terminal haematemesis. Stomach: mucosa normal except for a few small haemorrhages in the body and antrum.

Man aged 74 with history of alcoholic excess until about four years ago. Died with portal cirrhosis, primary carcinoma of liver, obstructive jaundice, and pulmonary oedema. A small subacute duodenal ulcer also seen at necropsy. Stomach: normal except for multiple small mucosal haemorrhages and haemorrhagic erosions.

Results of Examination of Specimens

All 25 cases of alcoholism were examined. Normal gastric mucosa was present in nine patients, whose ages were between 20 and 68 and averaged 38.5 years; mild to moderately severe chronic inflammatory changes, mostly mild, were present in seven patients, whose ages were between 29 and 66 and averaged 46 years; and chronic atrophic gastritis was present in six patients, whose ages were between 48 and 60 and averaged 52.5 years. Mucosal haemorrhages were found in five stomachs and erosions in six. Peptic ulceration was present in six patients, three of whom had a chronic gastric ulcer and one had two acute gastric ulcers; of the remaining two, a chronic duodenal ulcer was found in one and a subacute duodenal ulcer in the other.

Among the six patients with chronic atrophic gastritis, three had a chronic gastric ulcer and one a chronic duodenal ulcer. These lesions, and also the mucosal haemorrhages and erosions which were observed, may have been caused, directly or indirectly, by excess of alcohol, but proof is lacking; equally the pathogenesis might be essentially unrelated to alcohol: in the cases of obstructive jaundice, for instance, the mucosal haemorrhages could have been the result of prothrombin deficiency. Finally, in assessing the significance of alcohol in relation to inflammatory changes in the gastric mucosa, the general trend towards an increasing incidence and severity of chronic gastritis with increasing age (Wynn Williams, 1950; Joske *et al.*, 1955) must not be ignored.

Animal Experiments

The effect of graded concentrations and volumes of ethyl alcohol on the stomach wall was studied in young adult guinea-pigs, 300 to 400 g. in weight. A length of "polythene tubing," with a bore 0.86 mm. in diameter and a wall 0.57 mm. thick, was passed into the stomach through the mouth and alcoholic solutions were administered after attaching a 10- or 20-ml. Record syringe. At the completion of the experiment the animals, which had usually been fasted overnight, were killed by chloroform anaesthesia. Immediately after death the abdomen was opened and ligatures were placed at the cardia and pylorus; then 20 ml. of 10% formol-saline was injected into the stomach cavity. Afterwards the stomach was removed and immersed in 10% formol-saline for at least 24 hours. When fixation was complete the stomach was opened and the mucosa washed under a gentle stream of running water. This technique permits examination of a well-fixed mucosa, free of excessive folds (Wynn Williams, 1951). Finally, representative blocks of stomach wall were removed and paraffin sections of these were stained routinely with haematoxylin and eosin.

Concentration of Alcohol

The effect of variations in concentration on the gastric mucosa was studied by administering alcohol to six groups of non-fasting guinea-pigs, each group containing three

animals. The dose (5 ml.) was constant, but the concentration (volume for volume) increased in arithmetical progression from 10% in the first group to 60% in the last. The animals were killed 24 hours later. No notable changes were seen in animals given 10%, 20%, or 30% alcohol; but those given 40% or more had multiple haemorrhages and erosions and ulcers in the stomach, and also showed some loss of weight and fatty change in the liver, especially in the periphery of the lobules. The gastric lesions were of variable size and shape and often widely dispersed, but were more conspicuous in the upper two-thirds of the stomach. Erosions and ulcers were often deep and frequently haemorrhagic; most were of irregular shape; some were oval, round, or linear. No perforations were found; this was surprising, because microscopy often revealed almost complete penetration of the stomach wall in many lesions.

Of six guinea-pigs given the same volume and concentration of alcoholic solution but allowed to survive longer than 24 hours, three died within 48 hours with comparable lesions in the stomach. Similar haemorrhagic erosions and ulcers were noted in guinea-pigs given 5 ml. of reputable brands of "neat" Scotch and Irish whisky or gin.

Volume of Alcoholic Solutions

The effect of increasing the volume of the lower concentrations of alcohol was studied in non-fasting animals. To each of six guinea-pigs fasted for 24 hours 10 ml. of 10% alcohol was administered; the same volume of 20% alcohol was given to a second group of six animals and 10 ml. of 30% alcohol to a third group of six. Twenty-four hours later a few small mucosal erosions and ulcers were observed in the stomachs of animals that had been given 20% alcohol. More conspicuous erosions and ulcers were seen in animals given 30% alcohol, and these animals also showed slight weight loss and fatty change in the liver; moreover, the stomachs contained copious quantities of watery and often bile-stained fluid, containing little or no free HCl and less pepsin than normal; on culture of gastric contents Gram-negative intestinal coliforms and heat-resistant streptococci were constantly found and *Clostridium sporogenes* was isolated from three animals.

This experiment was repeated, using double the volume of alcoholic solution but concentrations of 10% and 20% only. After 24 hours the appearances of the mucosa were essentially the same as those seen after the administration of the smaller volumes of these concentrations.

Influence of Fasting

The effect of fasting was studied in three groups of guinea-pigs, each group containing six animals. Animals were each given 10 ml. of alcohol; those in the first group received 10% alcoholic solution, those in the second 20%, and those in the third 30%. The animals were allowed water *ad lib.*, but were deprived of food for 24 hours by being kept in stocks, this being necessary because hungry guinea-pigs eat their faeces. They were killed 24 hours after the administration of the alcohol. In the animals given 20% and 30% alcohol the appearances of the gastric mucosa resembled those seen in non-fasting animals given the same volume and concentration of alcoholic solution, but the lesions were rather more severe.

Early Mucosal Changes following Alcohol Administration

Six guinea-pigs, starved overnight, were each given 5 ml. of 60% alcohol and killed at intervals of half, one, two, four, six, and eight hours. After half an hour the mucosa was hyperaemic and there was some desquamation of the surface epithelium. After one hour numerous necrotic foci were observed in the mucosa; these varied considerably in depth and breadth. In animals killed two to eight hours after the alcohol administration necrotic foci were more conspicuous, sometimes haemorrhagic, and invariably associated with some degree of polymorph leucocytosis. There were also frank erosions and ulcers, often extensive in area and depth, in these animals.

Effect of Alcohol on the Healing of Experimental Gastric Ulcers

An ulcer 4 mm. in diameter was produced in the middle of the posterior wall of the body of the stomach in 30 guinea-pigs. Previous experiments had shown that such a lesion heals completely in 17 days in healthy guinea-pigs (Wynn Williams, 1953). Each animal was given 4 ml. of 60% alcohol daily by polythene tube. Only eight animals survived 17 days; in four the ulcer had healed completely; in two healing was complete except for a minute area in the centre; in the remaining two there was a larger unhealed area, approximately 1.5 to 2 mm. in diameter, and many parts of the adjacent mucosa were reddened and partially eroded. In all the stomachs there were multiple alcohol-induced erosions and ulcers; knowledge of this fact had made it imperative to mark the site of the experimental ulcer at operation by "tattooing" the stomach wall at four points around the ulcer site; this made subsequent identification of the healed or unhealed lesion easy.

Discussion

Beaumont's published studies (1833) on the appearances of erythema, simple erosions ("apthous patches"), and a cloudy viscid secretion in the stomach of Alexis St. Martin following alcoholic sprees remain prominent in the history of gastro-enterology. It is perhaps unfortunate that more was assumed from such dramatic findings than the evidence warrants, so that the conviction that alcoholism inevitably leads to acute or chronic gastritis became established in the minds of most people, including many doctors. Faber (1935) suggested that minor degrees of recurring damage to gastric mucosa—for example, in chronic alcoholics—result in chronic atrophic gastritis; and Gray (1943) stated that about 50% of chronic alcoholics have chronic gastritis on gastroscopy. The observations of Hirsch (1916), who fixed the stomachs of 21 people immediately after death from acute alcoholism, have been largely overlooked. He found that, except for mucosal haemorrhages, the macroscopic and microscopical appearances of the stomach were unaltered.

Palmer (1954) made gastroscopic and biopsy studies on 34 young men within six hours of severe alcoholic drinking. None of the subjects could be classified as a chronic alcoholic. Acute gastritis was diagnosed gastroscopically in 30 of the men and was manifest as mucosal hyperaemia, erosions, petechiae, and purulent exudate. The gastric mucosa was reported as normal in the remaining four. Biopsy from the body region of the stomach showed necrobiosis in the region of the necks of the glands in all specimens, also polymorph leucocytosis and oedema; very small superficial erosions were a feature in the mucosa of 24 of the 34 specimens; in eight specimens there was a mild infiltration of the stroma with plasma cells and lymphocytes. The surface epithelium appeared normal in all the specimens. In addition to this investigation, Palmer also examined mucosal biopsies from the body of the stomach in 200 random patients; 112 had taken alcohol in variable amounts and times, and 35 were described as having been "dead drunk" at least once a month during the year prior to the investigation. The histological findings were normal in all these patients. Palmer states emphatically that chronic gastritis is not of alcoholic aetiology.

Berry (1941) and Gray and Schindler (1941) examined gastroscopically 200 patients with a history of severe alcoholism. In some patients, 4–6 pints (2.3–3.4 litres) of 20 to 90% alcohol had been consumed daily for as long as 40 years, yet the authors found no more change than was ordinarily encountered in non-alcoholic dyspeptics. A high incidence of gastritis was described by Villaret *et al.* (1936), who gastroscopied 50 alcoholics with liver disease; and Schindler (1947), another gastroscopist, noted that the incidence of gastritis—about 45%—was much greater than expected for non-alcoholic dyspeptics chosen at random; but, nevertheless, he expressed the opinion that chronic alcoholism was not, as many believed, the chief aetiological factor in chronic gastritis.

In gastric biopsies of 20 chronic alcoholics with signs of malnutrition Motteram (1951) found that in 19 patients the stomach mucosa appeared normal or was only slightly different from normal; in biopsies of three patients immediately after a phase of heavy drinking there was a superficial gastritis with necrotic cells in the adherent mucus. Doig and Wood (1952), however, found in gastric biopsy material that 12 out of 48 patients with superficial gastritis and 7 out of 64 patients with chronic atrophic gastritis were chronic alcoholics. They were inclined to believe that chronic alcoholism is associated with gastritis through the related malnutrition rather than through the direct effect of alcohol on the mucosa. Cox (1943) did not believe that alcoholism caused gastritis, but Joske *et al.* (1955) recorded a high incidence of gastritis in alcoholic subjects: they described such changes in 50 of 95 biopsies—39 with superficial gastritis and 11 with atrophic changes. They attributed these to a direct toxic effect of alcohol.

It is apparent that the medical literature relating to alcoholic excess in humans contains a wide variety of observations and opinions. Have published reports of animal experiments increased our understanding of the effects of alcohol on the stomach? The impression gained from such reports is that serious damage to the gastric mucosa is rare or absent; however, because the number of publications is small and the experimental conditions are so variable, final deductions cannot be drawn with confidence. Ivy *et al.* (1950) mention that Quinke, in 1875, reported the local application of alcohol to the gastric mucosa as one method of producing acute ulceration in experimental animals. Grant (1945) also described acute superficial erosion following the application of 50% alcohol to the gastric mucosa of cats; she was impressed by the rapidity of healing, which was found to be faster than that seen in the skin.

Sternberg (1907, 1908) observed gastric ulcers in guinea-pigs following the introduction of 96% alcohol into the stomach, but Greggio (1916-17) reported an absence of ulceration in 26 rabbits given various amounts of 16% alcohol or Marsala wine by stomach tube for periods up to 50 days; and Friedenwald (1905), who administered alcohol to 120 rabbits in doses of 5 to 8 ml. for several months to over four years, found chronic gastric ulcers in only two cases and scars of healed ulcers in four others. Payne and Carey (1943) gave 75 ml. of 20% ethyl alcohol daily by stomach tube to four dogs for periods of 336, 394, 799, and 906 days respectively, and found no haemorrhages, erosions, or ulcerations at necropsy. Finally, Ivy *et al.* (1950) refer to the unpublished work of Wigodsky in their laboratory: he gave 12 dogs cheap bonded whisky for up to one year in amounts which would correspond to a litre daily for a 70-kg. man and found no gastric erosions or ulcers.

The results of the animal experiments described in the present report indicate that haemorrhages, erosions, and ulcers are found in the stomach following the administration of alcoholic solutions by stomach tube in concentrations of 20% or more. The greater the concentration of alcohol the more severe are the lesions. Furthermore, increasing the volume of alcohol also tends to increase the severity of the gastric lesions, as also does fasting. Spirits like whisky and gin have an alcohol concentration of about 40%; fortified wines such as port or sherry a concentration of about 20%, while beer and cider have a concentration of 5-10%. Although the amounts of alcohol administered to the guinea-pigs were relatively very large compared with those consumed by the majority of human subjects, it may be reasonable to assume that the consumption of large quantities of the more concentrated alcoholic drinks may lead to haemorrhages, erosions, or ulcers in humans, especially if the drinks are taken on an empty stomach. Moreover, it would seem probable that drinks, such as beer, which contain small concentrations of alcohol might be well tolerated even when taken in excess for many years.

The interpretation of the pathogenesis of so-called chronic atrophic gastritis has remained an enigma, but I have been

impressed by the similarity of the histological appearances in this condition to those seen in completely healed deep erosions or shallow ulcers of the stomach. As Grant (1945) observed, the rate of healing of gastric erosions produced by alcohol is very rapid. It is possible, therefore, that in a proportion of stomachs with chronic atrophic gastritis the latter may be attributable, in part at least, to chronic alcoholic excess. That this lesion is not seen more often in chronic alcoholics may be due to many factors associated with drinking and eating habits.

It is customary to prohibit alcoholic drinks in the treatment of peptic ulcers because they are believed to impede healing by stimulating gastric secretion and also, possibly, by direct irritation of the ulcer. Ivy *et al.* (1950), however, state that there is no proof to support the clinical impression of impaired healing. They add, moreover, that there is no proof that alcohol is important in the pathogenesis of peptic ulceration. It was found in the animal experiments described here that a gastric ulcer produced by burning the mucosa *could* heal normally despite the daily administration of 4 ml. of 60% alcohol in 50% of the animals; in all the animals there were numerous erosions and ulcers dispersed in the gastric mucosa at post-mortem examination, but only in half the number of animals had these lesions interfered with the healing of the ulcer produced by burning. These results may have some relationship to human pathology, and suggest that strong alcoholic drinks are probably best avoided in peptic ulcer patients.

Summary

An account is given of the appearances of the stomach mucosa in 25 alcoholic subjects and also in guinea-pigs given alcohol by stomach tube. A normal histological appearance is not infrequently found in alcoholics, and the relationship of alcoholism to chronic gastritis is uncertain. In concentrations comparable to those of common spirits, such as gin or whisky, alcohol may produce severe haemorrhagic and ulcerative lesions in the animal stomach. Although there is no proof that alcohol produces peptic ulcers in humans, or interferes with the healing of such ulcers, it would seem prudent to continue the current practice of avoiding strong alcoholic drinks in patients with peptic ulcer.

I am indebted to Professor J. S. Young for his enthusiastic help and to Dr. N. F. Coghill for providing the gastric biopsies, also to the several technicians who helped me.

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