facility as the alcohol became absorbed. In each case the reaction time was taken as the mean of at least seven determinations made at random intervals.

Results

Nystagmus appeared in 4 out of 12 subjects within 15 minutes of taking alcohol. There was variation in the dose needed to produce the physical sign; the majority required from 0.7 to 1 ml. of absolute alcohol per kg. body weight, while a few did not exhibit nystagmus until they had received 1.4 ml. per kg. The reaction time was measured at the first indication of nystagmus or nystagmoid jerks (Howells, 1952). It will be seen from the Table that in

Table of Results

Subject No.		Reaction Time in Seconds	Standard Deviation	No. of Observations	Increase in Reaction Time
1	Normal	0.198	0.017	10	
	After 1 hour	0.228	0.031	10	15%
2	Normal	0.262	0.043	9	
	After 10 mins.	0.298	0.044	9	13.7%
	" 20 "	0.3	0.021	7	14.5%
3	Normal	0.232	0.024	10	
	After 18 mins.	0.31	0.044	9	33.6%
	" 28 "	0.326	0.03	9	40.5%
	" 50 "	0.288	0.036	7	24.1%
4	Normal	0.24	0.035	10	
	After 15 mins.	0.26	0.035	7	8.3%
5	Normal	0.156	0.036	10	
	After 30 mins. approx.	0.212	0.036	10	35.9%
6	Normal	0.138	0.037	23	
	After 40 mins.	0.161	0.02	12	16.7%
7	Normal	0.125	0.023	24	
	After 20 mins.	0.16	0.024	14	28%
	" 50 "	0.167	0.029	11	33.6%
8	Normal	0.148	0.043	28	
	After 15 mins.	0.167	0.048	22	12.8%
9	Normal	0.163	0.033	20	
	After 15 mins.	0.17	0.034	18	4.3%
	" 45 "	0.22	0.039	32	34.9%
10	Normal	0.167	0.026	44	
	After 45 mins.	0.183	0.026	31	9.6%
11	Normal	0.185	0.034	32	
	After 45 mins.	0.222	0.058	29	20%
12	Normal	0.21	0.034	25	
	After 18 mins	0.22	0.036	36	4.8%
	" 45 "	0.23	0.044	25	9.5%

every case the appearance of nystagmus was accompanied by an increase in reaction time. The increase at the onset of the physical sign varied from 4.3% to 35.9%. In all those cases where further estimations were made it was evident that the increase was not maximal at the onset of nystagmus, and in one subject it subsequently rose as high as 40.5%. In none of the subjects examined was any abnormality of the pupil found. Alcohol when given in approximately the same quantities in the form of whisky or gin produced the same effects.

Discussion

This experimental evidence suggests that the nystagmus found in the alcoholic motorist was due to the taking of alcohol and can be reproduced in experimental subjects by giving ethyl alcohol, whisky, or gin. Its appearance after taking alcohol suggests that a subject of average weight has received a minimum of 50 ml. of absolute alcohol or its equivalent. The increase in reaction time which was evident during the period of nystagmus is an indication of impaired function of the central nervous system. The fact that no other eye sign was found indicates that nystagmus is the earliest eye sign produced by alcohol and is not dependent on any abnormality of the pupil size or reaction.

In a large proportion of subjects it was noted, when determining reaction time after alcohol, that the first response to the stimulus was greatly delayed. This occurred in spite of the fact that the subject was well aware that the test was about to take place. Similar delay was not found in the first test of the control series before alcohol. This point is being further investigated.

Summary

The ingestion of a known volume of alcohol in volunteer subjects has been studied. In all cases nystagmus was produced after a variable period of time. On the appearance of this physical sign the reaction time to a light or sound stimulus was prolonged.

I am grateful to Professor G. L. Brown for providing laboratory facilities, and also thank Dr. M. G. Eggleton for her help and suggestions.

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EFFECT OF POLYAMINOSTYRENE ON GASTRIC ACIDITY

BY

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Polyaminostyrene ("styrion") is an anionic exchange resin, having the following chemical structure:

It is a brownish material insoluble in water; a weak base which will give salts with strong acids. These salts are also insoluble.

After ingestion, neutralization of gastric acidity converts the resin into the chloride form, which on reaching the alkaline medium of the small intestine changes back again into the original substance, with the formation of water, CO₂, and NaCl. The unchanged resin is excreted in the faeces.

There is no release of gas in the stomach, and there is no loss of electrolyte from the body. Neither is there a risk of alkalosis occurring during therapy. It would appear that polyaminostyrene constitutes the almost perfect antacid, but little attention has been paid to the substance in Great Britain. For this reason a small trial was made in order to see whether the results merited a further and longer trial. Five patients, four with duodenal and one with gastric ulceration, were investigated. All had high gastric acidity, as measured by a fractional test meal.

Method and Results

A gruel fractional test meal examination was made and the presence of hyperacidity demonstrated. Forty-eight

FIG. 1.—Superimposed fractional test meal curves of five patients with peptic ulcer.

FIG. 2.—Fractional test meal of same five patients as in Fig. 1 after ingestion of 0.8 g. of resin at point indicated.

hours later a similar examination was performed, but after the third specimen had been withdrawn two tablets (0.8 g.) of resin were given. The subjects were asked to chew the tablets to a fine powder, and then swallow them. The curves of free and total acid (average of the five patients) are shown (Fig. 1). The second graph (Fig. 2) is of the same five, except that polyaminostyrene has been given at the point indicated. It can be seen that the free acid is eliminated and the total acid greatly reduced. This effect has been observed to last for an average of two hours after the tablets are taken. No unpleasant side-effects were observed; and it is perhaps significant that, of the five patients, three considered that their ulcer pain was relieved within ten minutes of taking the resin.

Discussion

Since this was merely a pilot investigation to determine whether in fact a prolonged clinical trial would be worth while, no attempt was made to observe the effect of the resin upon healing of the ulcer, etc. Blood electrolyte studies were not made, in view of the very small dosage given, but in a larger series, when the resin would be given over a prolonged period of time, this would become necessary. However, the effects of anionic exchange resins as antacids have been studied by Segal *et al.* (1945) and by Wirts *et al.* (1950). The results obtained show that the resins have an action similar to that of aluminium hydroxide. Both substances will cause a rise in the pH of the gastric contents, reducing the free acid and depressing pepsin activity. Anionic resins in these cases did not cause electrolyte imbalance. Hjort (1953) comments on the double action of polyaminostyrene in reducing peptic activity. This worker demonstrated that not only was pepsin inactivated by the rise in gastric pH, but that the resin was capable of absorbing pepsin and removing it from the stomach. Despite favourable reports from Scandinavia and the U.S.A., there has been a relative neglect of anionic resins in this country, and, as Jordan (1951) states, these substances would seem to merit a trial. Since the sheet anchor of ulcer therapy is still antacids, any simple and safe method of producing neutralization of gastric acidity would be advantageous.

It is difficult for ambulant patients to carry bottles of medicine about with them, and it is important that such patients should have easy access to their drugs throughout

the day. For this reason, medication in tablet form is the vogue, and there are many preparations of this nature available. Of these, perhaps the most reliable is "nulacin" (Douthwaite and Batty Shaw, 1952). This tablet, while sucked in the mouth, provides an alkaline milk drip for the active patient, and has been shown capable of neutralizing gastric hyperacidity. Nevertheless, the tablets are too large for some, and if taken too often may cause alkalosis. Polyaminostyrene, however, has also been shown to be effective, safe, and free from side-effects. Apart from its effect on the pH, the ability of the resin to absorb pepsin makes it attractive to the clinician, and results elsewhere, as stated, have been favourable. Thus Tomenius (1955) sums up by saying that the resin "... of our antacids is one of the best, possibly the best so far available, in the treatment of peptic ulcers." Because of these results, and in the light of personal experience, it is hoped to make a trial of polyaminostyrene in the long-term treatment of peptic ulcer on a much larger group of patients. It is hoped that these findings will be published at a later date.

I would like to thank Dr. J. C. Hawksley, on whose patients these investigations were made.

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Medical Memorandum

Association of Jejunal Diverticulosis with Macrocytic Anaemia

The association of macrocytic anaemia and jejunal diverticulosis has seldom been recorded. The first case we have been able to trace is that of Taylor (1930). Other cases have been recorded by Montuschi (1949), Badenoch and Bedford (1954), Paulley (1954), and Dick (1955). This last is very similar to our own, and Dick noted the beneficial effect of chlortetracycline (aureomycin) on the intestinal symptoms. Liver preparations had been given previously, and it is not possible to judge what part, if any, the chlortetracycline played in the haematological control. It is therefore worth while reporting a case in which a satisfactory response was obtained with chlortetracycline alone.

CASE REPORT

A married woman aged 70 was referred to the out-patient department in May, 1954, with abdominal pain; she had suffered from such attacks for 40 years and had had her appendix removed in 1926. After an attack of bronchitis in December, 1953, her health declined, with repeated attacks of abdominal pain and vomiting, and loss in weight. She had never had diarrhoea. For the past two or three years her tongue had periodically been sore. She was not regarded as anaemic. Barium-meal examination showed a large diverticulum at the duodeno-jejunal flexure and many fluid levels in the bowel. At operation the large diverticulum was found to be fixed to the inferior mesenteric vein, and as it was thought to be capable of obstructing the bowel (rather in the manner in which a pharyngeal pouch produces dysphagia) it was removed. Beyond this there were 80 or more fair-sized jejunal diverticula (Fig. 1).

After her discharge she suffered from diarrhoea as well as from periodic abdominal colic and vomiting. Her stools