

Gastritis, Aspirin, and Alcohol

SIR,—I was interested in Dr. D. N. Croft's excellent article on gastritis (21 October, p. 164), and welcome the prominence that he gives to the role of aspirin and alcohol in provoking gastritis and haematemesis. He shows that gastritis is usually self-induced. He does not, however, mention the considerable risk of gastroduodenal haemorrhage when alcohol and aspirin are taken together, the latter often at the end of a binge in an endeavour to prevent a hangover.

Some years ago I reported to the Association of Physicians of Region No. 1 the results of a small survey of every emergency medical admission with haematemesis and melaena to this hospital in 1958 and in 1960 (unpublished). In 108 cases we found that 72 had taken aspirin in some form, as far as could be ascertained, within 24 hours of the onset of bleeding. In the year 1958, of 31 male admissions 23 had taken aspirin and 16 had taken alcohol within 24 hours of the onset, and 10 had taken both. We thought that alcohol considerably increased the risk of bleeding due to aspirin, and it was significant that male admissions with haematemesis were more frequent at week-ends and during public holidays.

Incidentally, we found that at first patients often denied aspirin ingestion, until they were carefully interrogated using several of the more popular trade names. We now hand each patient a printed list of the 321 aspirin-containing preparations they would do well to avoid in future. The most confusing was undoubtedly Alka-seltzer, none of its victims being aware of the aspirin content of this well-advertised preparation, and many thinking that it was purely an alkali and therefore taking it for gastric discomfort or pain.

Further inquiry was made into the drinking habits of the ulcer patients. There were 75 males with gastroduodenal bleeding, and 48 of these were considerable drinkers, nearly always bitter beer, and consumption varied from a mere one gallon (4.5 l.) of beer per week to as much as seven gallons per week. Of these 48 individuals 32 were considered to have a duodenal ulcer, mostly confirmed or strongly suspected. These and other figures lead one to reflect that in this district regular beer drinking, especially bitter beer, over a long number of years eventually leads to duodenal ulcer, and one speculates about whether this may explain why it is that duodenal ulcer is so much more common in men than women. If this is correct the incidence of duodenal ulcer in total abstainers should be low. It seems to me that the role of alcohol in relation to gastroduodenal troubles has hitherto been underrated, and that here is a problem which merits wider attention and further study. Also the public should be warned not to take aspirin after drinking.—I am, etc.,

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Body Building on Drugs

SIR,—Your leading article on the use of anabolic steroids in athletic training (11 November, p. 310) in reply to Dr. D. E. Pearson's letter (11 November, p. 353) is both timely and of importance. It may, however, be of interest to you to know that this subject has already been considered at some

length by the Medical Advisory Committee to the British Olympic Association, and that they utterly condemned the use of anabolic steroids in athletic training and pointed out the possible dangers involved in taking them. A copy of this recommendation has been sent to all the governing bodies of sport in this country.

In addition to this the International Olympic Committee has issued the following statement to the National Olympic Committees of 123 countries: "The International Olympic Committee considers the use of anabolic steroids (except for medical purposes) constitutes 'doping' from the Olympic viewpoint."—I am, etc.,

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Coagulation of Rubber Latex in the Stomach

SIR,—The common poisons used by agricultural workers in Ceylon attempting suicide are insecticides, which are easily available to them. Rubber latex is not considered to be a poisonous substance, and we report here a case of attempted suicide by ingestion of rubber latex. The only symptoms produced by the ingestion of rubber latex were due to the mechanical effects on the stomach of the coagulated rubber latex.^{1,2} This is an unusual foreign body in the stomach about which there have been no earlier reports in the literature.

A 49-year-old woman who works as a rubber tapper on a rubber estate was admitted to the medical ward with a history of having swallowed a cup full of fresh rubber latex. She had confessed to her daughter that she had taken the rubber latex with the intention of killing herself. She had ingested the rubber latex four hours prior to her admission to hospital. Although alert and conscious at the time she was brought to the hospital outpatients department she refused to answer any questions. Apart from slight abdominal pain, she had no other symptoms, and seemed reasonably comfortable. On examination the pulse was 90 a minute, regular, and blood pressure was 110/90 mm. Hg. An elongated sausage-shaped lump was palpated in the epigastrium. It was firm, but could be pitted on pressure. A plain x-ray of the abdomen did not reveal any abnormality.

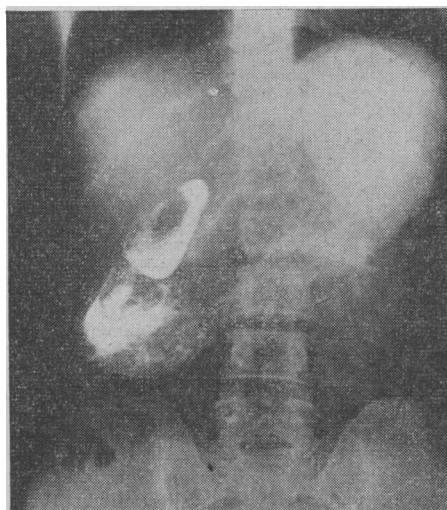


FIG. 1.—X-ray of the abdomen taken after barium showing the filling defects in the stomach and in the second part of the duodenum.

The x-ray of the abdomen taken after giving a dilute solution of barium to swallow showed two filling defects; one corresponding with the lump felt in the abdomen in the stomach and the other due to the presence of solid latex in the second part of the duodenum (Fig. 1).

At operation the solid latex, moulded to the shape of the stomach, was removed and the gastrostomy wound closed. This solid rubber was of creamy yellow colour with the impressions of the rugae of the stomach on it (Fig. 2).

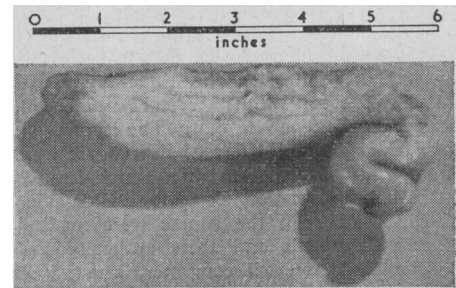


FIG. 2.—The specimen of solid rubber removed from the stomach.

When thrown on the floor it bounced like any other piece of rubber. The other latex lump was identified in the second part of the duodenum. Duodenostomy was done and the lump removed. She made an uneventful recovery. She was later seen by the psychiatrist.

Rubber latex is a milky fluid obtained by cutting the bark of the rubber tree. This latex, when coagulated, forms the solid rubber. In the rubber factories of Ceylon coagulation of rubber is obtained by adding certain chemical substances to the latex. Eight ounces (200 ml.) of 8% sodium bisulphite and 5 oz. (125 ml.) of 6% formic acid are used for 40 gallons (182 l.) of latex for it to coagulate. In this particular case the acid present in the stomach must have in some way contributed to the coagulation of the latex in the stomach.

We would like to thank Mr. M. de S. Jayasinghe, of the Photographic Department, General Hospital, Colombo, for his help with the photographs in this case.

—We are, etc.,

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- 1 Aird, I., *A Companion in Surgical Studies*, 1958, 2nd ed. Edinburgh.
- 2 Souttar, H., *Textbook of British Surgery*, 1956, London.

Prevention of Rh-haemolytic Disease

SIR,—My colleagues and I were interested in Dr. Sheilagh Murray's letter (4 November, p. 296), and the following are the replies to her queries:

The treated and control women in the Liverpool group who had subsequently Rh-positive babies were tested for antibodies immediately after delivery of the second baby. The babies were in all cases Coombs-negative, normal, and liveborn. In the clinical trials elsewhere, all we know is that the babies were also Rh-positive, normal, and liveborn, and that no treated women have made antibody, but we do not know the precise time at which the tests were carried out.

As far as we know, the 110 women remaining in the treated group have not become pregnant

again, nor is there any record of either stillbirths or abortions, except for a single case of abortion in the controls. However, complete ascertainment is extremely difficult and there may well have been a few cases which have not been reported. Our routine procedure is to give the mother a card when she is tested for antibodies six months after the birth of her first baby, and she is told to send it to us when she becomes pregnant again. As a precaution the case sheets of the mothers are also examined at intervals in case there is any indication that the woman has become pregnant.

The number of cases in the various trials has increased considerably since I gave my lecture, and the data have been sent to Dr. Murray. Our general experience in Liverpool of the time interval between first and second pregnancies does not suggest that a particularly small percentage of the women in the trial have had second babies.

The anti-Kell experiment gave inconclusive results because of difficulties with the anti-Kell antibody.

Dr. Murray speculates that suppression of D-immunization by anti-D gammaglobulin may be accompanied by some impairment of general immunity to infection. Not only have we followed up both our volunteers and trial women and found nothing to suggest this, but the gammaglobulin we inject differs from preparations used for other purposes only in that 1/500th part is anti-D, and I cannot see that its effect on general immunity can be different from that of the more usual preparations.

We have now tested for anti-Gm antibodies 62 treated women and have found positives in four, giving an incidence of 6.3%. The controls are still being studied, but we do not think that the formation of anti-Gm antibodies is likely to be damaging, for, if it were, it would make the giving of blood transfusions a most dangerous procedure, since incompatible Gm antigens must often be injected in large quantities in transfusions of whole blood.

Although in our first experiments, in about 1960, there was enhancement of immunization by IgM, which has never been satisfactorily explained, yet since that time we had not used IgM but always IgG both in our experiments and the clinical trials.

We do not understand the relevance of Dr. Murray's questions about the "ultimate relationship of anti-Gm in rheumatoid arthritis" to our work. The four women with the anti-Gm are entirely well and there is no suggestion that they have or are developing rheumatoid arthritis. Furthermore, there is no evidence that Gm antibodies, either natural or induced by immunization, predispose to rheumatoid arthritis, and the distribution of the Gm groups is normal in patients with this disease.

While agreeing that more research is needed, the most important point to us seems to be to find the smallest effective dose, and an M.R.C. working party is organizing trials to determine this. There is some very recent evidence on the subject from New York.¹ Four groups of 10 Rh-negative men were given 10 ml. of group O Rh-positive whole blood intravenously at monthly intervals for a total of three injections. Twenty-four hours following the administration of blood three of the four groups were given 1.0 ml., 0.5 ml., or 0.25 ml. respectively of an ortho preparation containing 1,200 µg.

Group	Dose of Ortho Anti-D Gamma-globulin	Number With Anti-Rh at 9 Months	% With Antibody
I	0	6 of 10 men	60
II	1,200 µg	0 " 10 "	0
III	600 "	0 " 10 "	0
IV	300 "	0 " 10 "	0

Pollack et al. (1967).¹

of anti-Rh (D) antibody per ml. Blood samples were obtained thereafter at monthly intervals for nine months and examined for the presence of anti-Rh. The results are summarized in the Table.

It therefore looks as though 300 µg. of ortho anti-D (approximately the amount being used in our current 1-ml. trial) is effective in protecting against a fairly large volume of injected Rh-positive blood.

Whether the treatment is "unquestionably right for national use" is a decision for the Ministry of Health, which has set up a sub-committee of its Standing Medical Advisory Committee to consider the whole matter.—I am, etc.,

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- Pollack, W., Singher, H. O., Gorman, J. G., and Freda, V. J., Scientific Exhibit, American Association of Blood Banks Meeting, October 1967, New York.

Allergic Alveolitis

SIR,—In your leading article (16 September, p. 691) the term "extrinsic allergic alveolitis" was suggested as the appropriate general description for a group of conditions resulting from the inhalation of antigenic material and occurring in farmers, mushroom-pickers, bird-fanciers, pituitary snuff-takers, bagasse handlers, etc.

We would accept this term as appropriate if the pathology were confined to inflammatory changes in the interalveolar septa with or without an exudate into alveolar spaces, but this is not so, because at one stage a salient feature is the presence of numerous "sarcoid-like granulomata" which induced Dickie and Rankin,¹ who first described the histology in farmer's lung, to use the term "acute granulomatous interstitial pneumonitis." Another frequent feature, as indeed is pointed out in the leading article, is a bronchiolitis, the exudate of which may become organized. Further, as is to be expected in a pulmonary Arthus or Type III reaction, vasculitis occurs. Organization of the inflammatory damage often occurs in this group of diseases, leading to a chronic stage characterized by pulmonary fibrosis.

The term "extrinsic alveolitis" invites the suggestion that this fibrosis is to be compared with diffuse idiopathic fibrosing alveolitis, now apparently the term replacing idiopathic diffuse interstitial pulmonary fibrosis or "chronic Hamman-Rich disease," where the end result produces a fine interstitial fibrosis with a respiratory function profile of transfer factor defect.

In the group of diseases now under consideration, however, this is only one of the possible end-results. In many patients there is much fibrosis involving terminal conducting airways—for example, an obstructive airways disease profile is seen in about one-third of sufferers from chronic farmer's lung.² Reporting on lung-function studies in bagassosis, Weill and others³ concluded that some dyspnoeic patients in the chronic stage had obstructive airways disease which they considered was causally related to bagasse exposure.

We therefore feel that "extrinsic allergic pneumonia" is less specific and more appropriate than "extrinsic allergic alveolitis."

One of the difficulties in interpreting the published work on farmer's lung is that authors seldom make it clear whether they

are discussing the acute potentially reversible stage or the chronic stage of irreversible fibrosis. In order to obviate this situation it is suggested that the acute stage be referred to as "acute extrinsic allergic pneumonia," and the fibrotic end-result as "chronic extrinsic allergic pneumonia." The term used should describe clearly the whole clinico-pathological entity.—We are, etc.,

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Demonstration Aerosol Inhalers

SIR,—Dr. A. Herxheimer (28 October, p. 236) suggested that manufacturers of pressurized aerosol preparations should provide "demonstration" inhalers so that prescribing doctors could instruct their patients more effectively.

It has been our practice to provide doctors with inert aerosol inhalers from time to time, through our representatives. Our object has been to stress the desirability of correct administration, and encourage doctors to supplement the instructions in the patient's leaflet with a practical demonstration.

As part of our current efforts to avoid misuse and abuse of pressurized aerosols, our representatives are now offering inert inhalers to all doctors on whom they call. The co-operation of all prescribers is being sought in ensuring that patients are using the right technique, and thereby obtaining maximum response from minimum dosage.—I am, etc.,

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Leicestershire.

H. E. LEWIS,
Medical Adviser,
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Purpura in a Patient Taking Chlordiazepoxide

SIR,—Non-thrombocytopenic purpura occurring following chlordiazepoxide (Librium) administration has been seen recently in this hospital.

The patient, a 65-year-old well-nourished Jewish woman, was investigated for a generalized purpuric rash. She had been receiving protamine zinc insulin for many years, and for at least 12 months had been receiving irregular courses of chlordiazepoxide (10 mg. twice daily) at approximately monthly intervals, each course lasting approximately a week. On admission the chlordiazepoxide was discontinued. The routine blood investigations were all normal.

The tourniquet test was positive, clot retraction was 45% (normal 48–64%), and the platelet count varied between 114,000 to 245,000/cu. mm. A peripheral blood film showed normal platelet morphology with perhaps occasional large forms. No lupus erythematosus cells were seen. The urine contained 130 mg./100 ml. protein, sugar at times, and an occasional granular cast. Serum proteins were 7.6 g./100 ml. (albumin