to disagree with this term, but it seems appropriate to us and its meaning has been clearly defined.—We are, etc.,

R. HALL
DAVID EVERED

Department of Medicine,
University of Newcastle upon Tyne

1 Masi, A. T., *American Journal of Public Health*, 1965, **55**, 658.
2 Snyder, P. J., and Utiger, R. D., *Journal of Clinical Investigation*, 1972, **51**, 2077.
3 Ormston, B. J., Alexander, L., Evered, D. C., Clark, F., Bird, T., Appleton, D., and Hall, R., *Clinical Endocrinology*, in press.

I.U.D. and Hydrorrhoea

SIR,—Hydrorrhoea gravidarum is a rare syndrome. I should like to report a case in which the contributory factor was an intrauterine contraceptive device, an association which has not previously been reported.

The patient conceived in spite of a Grafenburg ring which had been inserted two years previously. When some eight weeks pregnant she developed vague lower abdominal pain which resolved slowly over the next three weeks, uterine growth being satisfactory. An ultrasound examination at this time demonstrated an intrauterine pregnancy but failed to show the ring.

At a gestation of 22 weeks she developed a profuse watery vaginal discharge. The size of the uterus was compatible with her dates and the cervical os was tightly closed. The fetal heart was clearly audible and regular. From then on a watery fluid drained intermittently and in great quantity. It was alkaline and glucose free, but contained substantial quantities of protein. No fetal cells could be seen. An abdominal *x*-ray showed a normal fetus with a cephalic presentation. The ring was lying below the fetal head and above the internal os. She remained well until 32 weeks and fetal growth was good, but she then developed some pink staining of her discharge which was shown to be blood. A placental scan showed a grade I anterior placenta praevia.

Uterine growth continued and serial oestriol estimations were satisfactory until 35 weeks gestation when she developed some fresh painless vaginal bleeding, despite which the uterus remained lax and the cervix closed. The fetal heart was audible and regular and this was confirmed by a fetal electrocardiograph which showed no evidence of hypoxia. Bleeding continued throughout the day and so caesarean section was done. On incision of the lower segment a blood clot was seen from which protruded the broken Grafenburg ring. This was lying at the lower edge of the placenta, which had partially separated. The membranes were intact. A live female infant weighing 2·31 kg was delivered. The pelvis was normal apart from some fibrinous adhesions surrounding the right fallopian tube and ovary. Subsequent progress of mother and child was uneventful and there was no undue vaginal discharge.

Moir suggests[1] the hydrorrhoea gravidarum represents a failure of fusion of the decidua vera and the decidua capsularis. One patient is mentioned who had this complaint in three successive term pregnancies. Paalman and Veer[2] reported a series of 41 cases in which 11 (28·6%) had an associated placenta circumvallata and nine had a history of bleeding in early pregnancy. They felt that the fluid probably resulted from partial stripping of the membranes.

These reports and the present case suggest that hydrorrhoea gravidarum is caused by some irritant between the membranes and the decidua, probably blood but possibly a foreign body. In the present case it may be that the collection of fluid was lying just below the placental margin and thus caused slight placental separation—yet one more hazard of the intrauterine contraceptive device.—I am, etc.,

J. T. WRIGHT

Royal Free Hospital,
Liverpool Road Branch,
London N.1

1 Kerr, J. Munro, *Operative Obstetrics*, p. 252 8th edn. by J. Chassar Moir and P. Myerscough. London, Bailliere Tindall and Cassell, 1971.
2 Paalman, R. J., and Veer, C. G. V., der, *American Journal of Obstetrics and Gynecology*, 1953, **65**, 491.

Propellant a Factor in Asthma Deaths?

SIR,—In suggesting causes of the recent epidemic of deaths among asthmatics both your leading article (25 November 1972, p. 443) and Herxheimer[1] have overlooked the strong possibility that the propellant used in aerosol cans for dispensing medication may have been a contributor. Some of these aerosol propellants have been implicated in sudden deaths due to "aerosol sniffing,"[2 3] and one aerosol product (Pertussin) has been banned in the United States because of associated sudden deaths. Various halogenated hydrocarbons (haloalkanes), isobutane, propane, vinyl chloride, and other agents have been used as propellants in aerosol cans.[2 4] Probably not all, but several of them have been used in preparations for inhalation by asthmatics. Some of these agents, notably some of the haloalkanes, have been shown to be associated with cardiac arrhythmia and ventricular fibrillation as well as other effects in animals.[4-12] In one human experiment, premature atrial and ventricular contractions were noted after haloalkane and isoproterenol inhalation.[13] In some of these experiments, it was thought that the agent simply sensitized the heart to anoxia or to exogenous adrenergic agents,[4 12] but subsequent experiments have shown cardiac effects without either anoxia or exogenous adrenergic agents.[6 7 10 11] In addition, "aerosol sniffing" deaths have occurred from aerosols containing no adrenergic agents.[2] Both anoxia and adrenergic agents, however, enhance the cardiotoxicity of the haloalkanes.[4 9 11 12] Stolley focused attention on the high concentration of isoprenaline in some of the preparations for asthmatics, and showed that excess asthma deaths ocurred in those countries that had used the "high concentration" preparations.[14]

Herxheimer pointed out that isoprenaline is not cardiotoxic, and pointed out some discrepancies in the distribution of excess asthma deaths which Stolley's hypothesis failed to explain.[1] He went on to attribute the excess deaths to inadequate medical management as others had done.[15] Unfortunately, this hypothesis is even less adequate than Stolley's in explaining the distribution of excess asthma deaths. It utterly fails to account for the rise in asthma deaths during the time period which accompanied the introduction of the pressurized aerosol asthma preparations in England,[14 15] and the fall in those deaths after an official warning about use of pressurized medication by asthmatics was issued.[17]

Although several investigators have argued that asthmatic aerosol preparations are safe when properly used,[9 15 18 20] it appears likely that in sensitive individuals, a combination of overuse, hypoxia, adrenergic agents and certain aerosol propellants could produce cardiac arrhythmias and ventricular fibrillation. Increases in either hypoxia or concentration of adrenergic agents would probably increase the likelihood of ventricular fibrillation from these preparations.

The associations noted by Stolley[14] may have been with a preparation which accidentally combined high concentrations of adrenergic agent with the most cardiotoxic of the propellants. Other combinations of aerosol propellant and adrenergic agent could readily account for the spotty distribution of excess deaths noted by Herxheimer.—I am, etc.,

VICTOR E. ARCHER

National Institute for Occupational Safety and Health,
U.S. Department of Health, Education and Welfare,
Salt Lake City,
Utah, U.S.A.

1 Herxheimer, H., *British Medical Journal*, 1972, **4**, 795.
2 Bass, M., *Journal of the American Medical Association*, 1970, **212**, 2075.
3 Baselt, R. C., and Cravey, R. H., *Journal of Forensic Sciences*, 1968, **13**, 407.
4 Reinhardt, C. F., Azar, A., Maxfield, M. E., Smith, P. E., and Mullin, L. S., *Archives of Environmental Health*, 1971, **22**, 265.
5 Kilen, S. M., and Harris, W. S., *Journal of Pharmacology and Experimental Therapeutics*, 1972, **183**, 245.
6 Taylor, G. J., Harris, W. S., and Bogdonoff, M. D., *Journal of Clinical Investigation*, 1971, **50**, 1546.
7 Taylor, G. J., and Harris, W. S., *Journal of Laboratory and Clinical Medicine*, 1970, **76**, 857.
8 Taylor, G. J., and Harris, W. S., *Journal of the American Medical Association*, 1970, **214**, 81.
9 Clark, D. G., and Tinston, D. J., *Annals of Allergy*, 1972, **30**, 536.
10 Flowers, N. C., and Horan, L. G., *Journal of the American Medical Association*, 1972, **219**, 33.
11 Mullin, L. S., Azar, A., Reinhardt, C. F., Smith, P. E., and Fabryka, E. F., *American Industrial Hygiene Association Journal*, 1972, **33**, 389.
12 Azar, A., Zapp, J. A., Reinhardt, C. F., and Stopps, G. J., *Journal of American Medical Association*, 1971, **215**, 1501.
13 Brooks, S. M., Mintz, S., and Weiss, E., *American Review of Respiratory Disease*, 1972, **105**, 640.
14 Stolley, P. D., *American Review of Respiratory Disease*, 1972, **105**, 883.
15 Harris, M. C., *Annals of Allergy*, 1971, **29**, 250.
16 Smith, J. M., *Lancet*, 1966, **1**, 1042.
17 Inman, W. H. W., and Adelstein, A. M., *Lancet*, 1969, **2**, 279.
18 Azar, A., Reinhardt, C. F., Maxfield, M. E., Smith, P. E., and Mullin, L. S., *American Industrial Hygiene Journal*, 1972, **33**, 207.
19 McClure, D. A., *Toxicology and Applied Pharmacology*, 1972, **22**, 221.
20 Silverglade, A., *Journal of the American Medical Association*, 1971, **215**, 118.

Hypergastrinaemia in Rheumatoid Arthritis

SIR,—While one may excuse Dr. P. J. Rooney and his nine colleagues (30 June, p. 752) for not knowing of early studies suggesting an increased incidence of achlorhydria in rheumatoid arthritis[1-3] it is more surprising that they fail to note the report of Buchanan *et al.*[4] concerning Sjogren's syndrome. These authors demonstrated achlorhydria in four of six patients with this condition. Since 20% of the patients reported by Dr. Rooney and colleagues have keratoconjunctivitis sicca this is surely of importance in discussing the significance of the demonstrated hypergastrinaemia. As theirs was a preliminary report one wonders whether a further paper is already in preparation describing the results of gastrin assays in patients with Sjogren's syndrome? Should this be the case the authors may care to note that among the many prescient "objectives for further

study" listed by Short *et al.*[3] is that of gastric acidity in patients with Sjogren's syndrome.

Furthermore, they have reviewed the question of gastric acidity in rheumatoid arthritis,[3] particularly with reference to Edstrom's studies.[2] The latter author found achlorhydria among 12·8% of patients with rheumatoid arthritis under 40 and 28·6% of those over 40, a higher incidence than in comparable control groups. Short *et al.* confirmed these findings for the under 40 age group. While it may be questionable whether the data as presented by these workers can be subjected to proper statistical analysis the studies clearly deserve mention. Together with the point about Sjogren's syndrome they imply that studies of acid secretion should be done before implicating other factors—for example, anti-inflammatory drugs—as a cause of hypergastrinaemia.—I am, etc.,

KEVIN J. FRASER

Oxford Regional Rheumatic Diseases Research Centre,
Stoke Mandeville Hospital,
Aylesbury, Bucks

1 Moltke, O., and Ohlsen, A. S., *Lancet*, 1936, **2**, 1034.
2 Edstrom, G., *Acta Medica Scandinavica*, 1939, **99**, 228
3 Short, C. L., Bauer, W., and Reynolds, W. E., *Rheumatoid Arthritis*, p. 361, Cambridge, Mass., Harvard University Press, 1957.
4 Buchanan, W. W., *et al.*, *Gut*, 1966, **7**, 351.

Gastric Secretion over the Years

SIR,—In your leading article (18 August, p. 368) you concluded reasonably that "it is not possible to decide from these relatively limited data whether hypersecretion precedes or follows duodenal ulceration, though the evidence weighs in favour of hypersecretion being an aetiological factor." However, the follow-up studies cited.[1-5] were all of small numbers. There are other, larger, series in which the measurement was of serum pepsinogen and not gastric acid.

Fourteen of 2,031 recruits developed, during their two-year army service, *x*-ray evidence of duodenal (13) or gastric (1) ulcers, and their mean serum pepsinogen level (707 units) was significantly higher than that of the whole population (440 units).[6] Thirteen of the 14 with ulcers had a serum pepsinogen level above the normal mean and 10 above the mean plus one standard deviation. In these two years 15% of men in the top 5% of the serum pepsinogen range developed a duodenal ulcer.[7] A two-year follow-up of a smaller group of 263 craftsmen showed an identical tendency for radiolgical ulcers to appear in those subjects previously free of dyspepsia who originally had high levels of serum pepsinogen.[8] A four-year follow-up of 931 Yale freshmen showed that seven of the eight who developed duodenal ulcer originally had high or high normal serum pepsinogen levels.[9]

In so far as serum pepsinogen is an index of gastric secretory mass, these American studies support the hypothesis[10 11] that hypersecretion precedes and predisposes to duodenal ulceration and that duodenal ulcers develop almost exclusively in those in the upper half of the range of secretion of acid and pepsin.—I am, etc.,

J. H. BARON

Department of Surgery,
Royal Postgraduate Medical School,
London W.12

1 Ryle, J. A., and Bennett, T. A., *Guy's Hospital Reports*, 1937, **87**, 145.
2 Doll, R., Jones, F. A., and Maclagan, N. F., *Lancet*, 1949, **2**, 984.
3 Lander, F. P. L., and Maclagan, N. F., *Lancet*, 1934, **2**, 210.
4 Baron, J. H., *Gut*, 1962, **3**, 158.
5 Novis, B. H., Marks, I. N., Bank, S., and Sloan, A. W., *Gut*, 1973, **14**, 107.
6 Yessler, P. G., Reiser, M. F., and Rioch, D. M., *Journal of the American Medical Association*, 1959, **169**, 451.
7 Mirsky, I. A., *American Journal of Digestive Diseases*, 1958, **3**, 285.
8 Dunn, J. P., and Cobb, S., *Journal of Occupational Medicine*, 1962, **4**, 343.
9 Niederman, J. C., Gilbert, E. C., and Spiro, H. M., *Annals of Internal Medicine*, 1962, **56**, 564.
10 Baron, J. H., *Clinical Science*, 1963, **24**, 357.
11 Koster, K. H., *Scandinavian Journal of Gastroenterology*, 1966, **1**, 199.

Forcible Examination

SIR,—I found that your leading article on the Frau Meinhof case (1 September, p. 466) only added confusion to an already puzzling situation.

I understand that Frau Meinhof's fitness to plead is in question. A prisoner awaiting trial in Scotland or England whose fitness to plead was questioned by the prosecution would be referred for psychiatric examination. If the accused objected to such examination and the psychiatrist believed there was good evidence from the nature of the crime and observations by others that the accused might be suffering from serious mental illness he would persist in attempting a psychiatric examination despite the prisoner's objections and would submit a report to the court. Is the writer of your leading article objecting to this?

To a Scottish psychiatrist the Frau Meinhof case is puzzling as in this country a decision on fitness to plead is based on an opinion of the accused's mental processes—a demonstration of structural brain damage would be essentially irrelevant. An account of the German view of fitness to plead would have been more helpful to puzzled readers like myself than the naive comments of your leading article.—I am, etc.,

ERNEST P. WORRALL

Royal Dundee Liff Hospital,
by Dundee

Childhood Obesity and Carbohydrate Intolerance

SIR,—Having read with interest your leading article (21 July, p. 122) on this subject I read the instructions on the packets of rusks and cereals freely available in this area. I was shocked to see that one manufacturer recommends the addition of ¾ rusk dissolved in boiled milk to each bottle feed at 2 months, increasing to 1¼ rusks at 5-6 months. There is no suggestion that reference should be made to the health visitor or doctor. I wonder how many of us are allowing these products to be sold in our clinics without being aware of the instructions?—I am, etc.,

JILL POOLE

Hertfordshire County Council,
Health Department,
Hemel Hempstead

Published Format of Clinical Trials

SIR,—Dr. G. Nyberg's letter (7 July, p. 47) prompts me to record my agreement with his general criticisms of published trials in angina and to make similar criticisms of trials of antihypertensive therapy.

In the course of recently reviewing the published papers on the actions of the various beta-adrenergic blockers in angina and in hypertension I found 23 acceptable double-blind randomized controlled trials in hypertension, but in only 11 were the basic observations (as opposed to "processed data") included. The reader, therefore, is unable to ascertain for himself whether each patient was in fact hypertensive (some otherwise admirable studies have included patients with diastolic pressures less than 90 mm Hg on placebo!) and whether the hypertension was "controlled." I think most physicians would agree that our aim should be the achievement of a diastolic pressure of 90 mm Hg or less, at least in mild and moderate hypertension (W.H.O. stages 1 and 2), but such data are available only in 50% of published papers.

The score is approximately the same in angina. Of 30 double-blind controlled trials there was adequate definition of patients with individual data on attack rates, glyceryl trinitrate consumption, and exercise tolerance in only 12.

I suggest that much of the blame lies with the editorial policies of journals, many of which insist on "processed data" because of pressure on space (for which reason I do not have the temerity to ask you to publish the 50 or so references related to this letter) and the cost of tabulation. I would like, therefore, to plead with all editors not merely to accept but to demand that the basic variables be presented for publication and, if necessary, restrict statistical comments to the text.—I am, etc.,

LUKE HARRIS

Summit, New Jersey,
U.S.A.

Lavatory Doors

SIR,—A 20-year-old epileptic patient of mine has just died in the lavatory at his home, after having a fit during which he fell against the lavatory door. The door had not been locked but as he was a heavy man and had fallen against the door it took his father half an hour to get through a window to release him. By this time he had died, presumably from asphyxia.

I have known people have heart attacks, pulmonary emboli, hypoglycaemic attacks, fainting after passing melaena stools, fainting after valsalva manoeuvres, miscarriages, and even babies in the lavatory. It is obvious that some of these emergencies are more likely to happen in the lavatory than anywhere else.

The old fashioned smallest room tended to be long and narrow and so there was always a good chance of getting the door open provided it was not locked. With the modern demand on space the lavatory is tending to be increasingly small, with a correspondingly greater chance of an unconscious person lying jammed against the door. I wonder whether architects could be persuaded in these circumstances to hang lavatory doors outwards, and also devise some mechanism rather than a conventional bolt so that they could easily be opened from the outside in an emergency.—I am, etc.,

M. D. BEGLEY

Frome