

was based on a significant rise in the titres of complement-fixation and metabolism-inhibiting antibodies to the organism. Cold agglutinins were present in all cases. In two of them agglutination also occurred in blood samples kept at room temperature. All the patients had high serum levels of IgM (330-500 mg/100 ml), while the concentrations of IgG and IgA were normal. Such a selective increase in IgM is known to occur in infections caused by mycoplasmas.^{3,4} All the patients had raised serum and urine concentrations of amylase. Analysis of isoenzymes of amylase in serum suggested that the increase in this enzyme in the serum originated from the pancreas. None of the patients had a history of gall stone disease and none took alcohol. No virus was isolated from throat washings and faeces and serological tests for coxsackie B, cytomegalo, and mumps virus were also negative.

Three of the four patients had a severely depressed respiratory exchange and two of them had to be treated with a respirator. One patient had E.C.G. changes of the type seen in pericarditis. One of them developed a hyperosmolar diabetic coma one week before she died. She had a high titre of cold agglutinins and multiple thromboses were found at necropsy. The three patients who survived had an increased concentration of serum amylase for several weeks after the clinical symptoms of pancreatitis had disappeared. Two of them had a transient rise in the blood glucose level.

A relationship between infectious pancreatitis, such as in mumps⁵ and coxsackie B infections,^{6,8} and diabetes mellitus has been suggested. In this respect pancreatitis in *M. pneumoniae* infections requires further investigation.—We are, etc.,

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- 1 Sterner, G., and Biberfeld, G., *Scandinavian Journal of Infectious Diseases*, 1969, 1, 203.
- 2 Grayston, J. T., Foy, M. M., and Kenny, G. E., in *The Mycoplasmales and the L-phase of Bacteria*, ed. H. Hayflick, p. 651. Amsterdam, North-Holland Publishing Company, 1969.
- 3 Feizi, T., *Annals of New York Academy of Sciences*, 1967, 143, 801.
- 4 Mårdh, P.-A., *Acta Pathologica et Microbiologica Scandinavica*, 1970, 78b, 726.
- 5 Melin, K., and Ursing, B., *Nordisk Medicin*, 1958, 63, 1715.
- 6 Gamble, D. R., Kinsley, M. L., Fritzsche, M. G., Bolton, R., and Taylor, K. W., *British Medical Journal*, 1969, 3, 627.
- 7 Gamble, D. R., and Taylor, K. W., *British Medical Journal*, 1969, 3, 631.
- 8 Gamble, D. R., and Taylor, K. W., *British Medical Journal*, 1973, 1, 289.

Effect of Respiration on Parkinsonian Tremor

SIR,—A tremor artefact on the electrocardiogram is a now well-recognized feature of Parkinson's disease. The accompanying figure shows such an artefact varying with the phases of resting respiration—an observation which to my knowledge has not been recorded previously.

In this case quiet inspiration (centre of record) was associated with a marked reduction of the gross tremor, which returned during the period of expiration. Although there was no obvious change in the heart

rate during the phases of resting respiration, I wonder if the decrease in vagal tone which is believed to occur during inspiration contributed its own anticholinergic effect which reduced the tremor? Since inspiration requires the positive contraction of the respiratory muscles it is possible that this effort alone could have been sufficient to reduce the tremor, especially since resting expiration is mainly the result of elastic forces. However, the presence of a greater tremor artefact during the period of forced expiration at the end of the E.C.G. record would appear to contradict this latter suggestion.—I am, etc.,

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Radioimmunoassay Follow-up of Hydatidiform Mole

SIR,—The letters of Sir John Stallworthy (3 March, p. 550) and Professor W. S. Tow (7 April, p. 49) seem to be answered by the data in my letter (17 February, p. 414). Repetition is justified only to counter the tendency to rate data from Singapore as more relevant to patients in the United Kingdom than data collected here. Professor Tow refers to a Singapore "malignancy rate" of 36.6% in women aged 40 or more, whereas in our series of 280 patients followed up after hydatidiform mole the "malignancy rate" for women aged 15 to 54 was 5.7% and for those aged 32 or more it was 0%. Singapore and the U.K. might differ in their disease patterns, or in their criteria of malignancy, or in both these factors. Extrapolation from one to the other has its limitations.

I see no objection to primary hysterectomy for the elderly or multiparous woman provided the follow-up be as careful as without hysterectomy. The suggestion that I am rash to claim that hysterectomy would not reduce the mortality rate is intriguing, since the rate was zero without routine hysterectomy in the series under discussion. However, if hysterectomy is life-saving in Singapore, why is it not advocated routinely for young and old alike? Either a life-saving procedure is withheld so that the survivors preserve their reproductive function or its benefits are judged to be marginal. What is the current rate of exchange between saving life and saving reproductive function and who fixes it?

Unfortunately, the place of hysterectomy is unlikely to be defined by the "scientifically designed prospective study" advocated by Sir John. It is a first principle to compare like with like. Our data showed that comparing hysterectomized elderly patients with non-hysterectomized younger patients, as he suggests, would be fruitless. Instead, patients would have to be defined by age and parity and allocated at random to hysterectomy and non-hysterectomy groups. Since we have seen choriocarcinoma presenting up to 17 years after primary hysterectomy for mole, the follow-up would need to be protracted. Such a trial might be difficult to organize, but the recently introduced registration scheme for patients with hydatidiform mole should go some ways to providing more ex-

tensive data than have been available hitherto.—I am, etc.,

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Anaesthetic Contamination

SIR,—I read with interest your leading article on anaesthetic contamination of operating theatres (24 March, p. 693) and the article by Dr. R. S. Vaughan and others on its prevention (p. 727). While I congratulate Dr. Vaughan and his colleagues on their valuable contribution to this subject I would like to point out that the solution offered by them solves only part of this problem. The activated charcoal filter will remove only the vapours of volatile anaesthetics, still leaving anaesthetic gases such as nitrous oxide and cyclopropane to pollute the operating theatre environment.

Nitrous oxide is a major cause of pollution in operating theatres, perhaps because it is used in high flow and concentration. In one study nitrous oxide has been measured in end-tidal samples and found to average 20 p.p.m.¹ and another study showed that the level of nitrous oxide could be as high as 7,000 p.p.m. in the inhalation zone of anaesthetists using non-rebreathing systems.² Chronic exposure to nitrous oxide is known to cause bone marrow depression.³ Bruce *et al.*,⁴ in their survey of causes of death in anaesthetists over the period 1947-66, concluded that the incidence of lymphoid and reticuloendothelial malignancy was two to three times greater than expected, amounting to almost 25% of all deaths due to malignant causes. Bruce Johnson⁵ suggests that nitrous oxide may be the causative agent of these malignant conditions and possibly of other problems associated with the operating room. In a recent study my associates and I have shown that nitrous oxide and cyclopropane in clinical concentrations inhibit the growth of respiratory pathogens and that this inhibition is related to the time of exposure rather than to the concentrations of anaesthetic gases (unpublished observations). The antimetabolic effect of nitrous oxide has been demonstrated in studies on cultures of mouse myoblasts and on the growth of cells in monolayer culture.^{7,8} It may be that the anaesthetic gases do cause abnormalities of cell division, and nitrous oxide may be responsible for congenital abnormality, spontaneous abortion, and involuntary infertility.

In a study now in progress Dr. W. J. Cole and I have found a high concentration of isopropyl alcohol vapour along with the anaesthetic agents causing contamination of the environment. In all air samples taken from the operating theatre this consistently produced a most intense peak on the gas chromatograph trace which was observed after the halothane peak. A 70% solution of isopropyl alcohol with chlorhexidine is commonly used for the preparation of skin before surgery. With the help of an Askrog exhaust valve we have been able to eliminate effectively the anaesthetic gases and vapours from the operating theatres. In the air samples from the theatre where the system is in use the concentration of halothane vapour has been found to be less than 0.1 p.p.m. We consider that venting of anaesthetic gases and vapours from the theatre is a simple, effective, and inexpensive method

