p. 1133), prompted me to examine the case records of 489 cases treated with buccal pitocin. There was no incidence of uterine rupture, maternal mortality, or foetal mortality, and there were only three cases of excessive uterine action. All three responded quickly (15–20 minutes) to withdrawal of the tablets from the buccal pouch. Hyperactivity of this nature (contractions at one-minute intervals) has occurred in the control group. Furthermore, in 31 cases where buccal oxytocin failed intravenous Pitocin (oxytocin) was successful in 10 cases only.

No one can deny that a risk of uterine rupture exists—as reported by Dr. P. M. Leney (12 September, p. 689) and Dr. H. O. Nicholson (3 October, p. 876), but spontaneous rupture does also occur and in fact did so in one case of the non-buccal group.

I do not think that obstetricians using buccal oxytocin deny the place, or better control, of intravenous oxytocin. However, oral administration is generally preferred to the parenteral even if there is some slight loss in performance—cf. oral iron and Ferrivenin (saccharated iron oxide).

Thus I cannot agree that buccal pitocin is a retrograde step, and the evaluation of 489 cases treated with the buccal hormone would support Mr. J. A. Chalmers's and Dr. J. L. Ng's (24 October, p. 1070) views.—I am, etc.,

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The Beginning of Ergot

SIR,—Professor J. Chassar Moir states ("The Obstetrician Bids, and the Uterus Contracts," 24 October, p. 1025) that the use of ergot in obstetrics started with Dr. John Stearns in U.S.A. in 1808. But Dr. Stearns, in his letter to his friend, Samuel Akery, published in the *Medical Repository* in New York in 1808, merely relates that the American midwives have been using for a long time, and with considerable success, the "black tea," or, as he calls it, "pulvis parturiens" (*sic*), to produce contractions of the uterus. In fact his letter was nothing more than the title indicates—namely, "Account of the pulvis parturiens, a remedy for quickening childbirth." There were no scientific data or any valuable clinical observations as to how and when to use it. This had to wait till 1823, when he first published his "Observations on the use of secale cornutum, etc., for its use in parturition" (*American Medical Recorder*, 1823).

It is now fashionable to attribute all discoveries to Americans or Russians, but in fact the first publication on ergot, its use, mode of action on the uterus, and its abuse and danger, appeared in London, by O. Prescott: "Dissertations on the natural history and medical effects of secale cornutum, etc." (*Medical and Physical Journal*, 1815).

When Dr. Stearns published his letter he was probably unaware that on the Continent ergot, under various forms, had been used by midwives for at least a century. The first to mention it was Camerarius (*De Ustagline Frumenti*, Tübingen, 1709). Like Dr. Stearns so much later, Camerarius was merely describing observations made by midwives. Ergot was used all over Europe under various names such as "pulvis partum accelerans,"

"poudre obstétrical," etc. The first study of the use of ergot in obstetrics in Europe was also, curiously enough, published as a letter, the famous "Lettre à l'Abbé Pozier relative à l'emploi du seigle ergoté pour accélérer l'accouchement" (*Journal de Physique*, t. 4, Paris, 1774). In it the virtues and dangers of ergot are clearly told.

At the beginning of the 19th century Roche warned: "Like every new drug it (ergot) becomes a panacea and tends to be used with inconceivable boldness, yet its misuse can cause rupture of the uterus." He concludes: "Only time and experience will teach us what is real in the numerous virtues with which this new drug is invested."

Professor Chassar Moir's conclusions about the new uterine contractants, one and half centuries later, give the same wise warning. Indeed, the "wheel completes" a most perfect circle.—I am, etc.,

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S. GALESKI.

Vasopressor Activity of Ergotamine

SIR,—Dr. I. W. Manson's case report ("Post-partum Eclampsia Complicated by the Anterior Tibial Syndrome," 31 October, p. 1117) was of considerable interest to me, but I feel certain that he will forgive me for pointing out what my paper¹ did say, in fact. Regarding the vasopressor activity of ergometrine, administered intravenously, it was stated that the rise in blood-pressure in toxæmic hypertensives was found to be maximal within three minutes.

The duration of the circulatory response is rather difficult to determine. In women delivered by forceps no definite conclusion could be reached in this respect, since the procedure usually terminated while the pressor effect was still in evidence. Moreover, narcotic medication post-operatively, handling of the patient, environmental and bed temperatures, and return of voluntary activity all tended to influence its duration. Greater opportunity for observation of this aspect of the study was afforded by cases of caesarean section reported elsewhere,² in which the response remained in evidence for 30–40 minutes.—I am, etc.,

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THOMAS W. BAILLIE.

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- 1 Baillie, T. W., *Brit. med. J.*, 1963, 1, 585.
- 2 ——— Thesis for Degree of Doctor of Medicine, University of Glasgow.

SIR,—I have read with much interest Professor J. Chassar Moir's lecture "The Obstetrician Bids, and the Uterus Contracts" (24 October, p. 1025) in which he states that "rather surprisingly, recent years have brought occasional reports of unexpected hypertension after the administration of ergometrine." Your readers may be interested in the following case report of a patient who not only developed an unexpected hypertension but also eclamptic convulsions approximately 65 minutes after an injection of ergometrine.

Mrs. A. B., a 17-year-old primigravida who had her L.M.P. on 1 October 1962, was admitted to Mile End Hospital on 9 July 1963 in labour with a blood-pressure of 120/80 mm. Hg and slight ankle oedema. General examination was normal and uterine enlargement was consistent with a 40 weeks' gestation. Her previous history

was non-contributory. The antenatal period was uneventful, the highest blood-pressure recorded in her 11 visits being 130/80 mm. Hg. Labour progressed satisfactorily. Triclorol (triclofos) was given in early labour followed by pethidine on two occasions. The blood-pressure throughout did not exceed 124/80 mm. Hg and urinalysis remained negative. A spontaneous normal delivery of a healthy baby weighing 6 lb. 11 oz. (3 kg.) occurred after a labour of 13 hours 35 minutes. An intramuscular injection of 0.5 mg. ergometrine maleate was administered to the patient as the head crowned. The blood-pressure at the end of the third stage—that is, 12 minutes after the ergometrine injection—rose sharply to 170/115 mm. Hg. Omnopon 20 mg. was injected and the blood-pressure recorded as 130/105 mm. Hg after 30 minutes. Approximately 25 minutes later she developed a severe frontal headache, the blood-pressure rose to 170/110 mm. Hg, and typical generalized eclamptic convulsions ensued. The usual eclamptic regimen including sedation with morphine and paraldehyde controlled the fits. The blood-pressure five hours after delivery and until she was discharged remained below 130/80 mm. Hg. Haematological investigations and blood and urine chemistry were within normal limits. A neurological opinion failed to reveal any cause for the convulsion apart from eclampsia. Screening test for pheochromocytoma was negative. Follow-up examination six months later showed no abnormality.

The vasopressor effects of ergometrine have been reported by Nixon¹ and Baillie² among others in this country, although several studies have appeared in the American journals since the work of Priver *et al.*³ A search of the literature, however, has revealed only two reports of post-partum eclampsia in previously normotensive patients in whom ergometrine was considered a causative factor.^{4,5} Short of eclamptic convulsions, Casady *et al.*⁶ presented evidence for incriminating ergometrine, on extremely rare occasions, in causing dangerous post-partum hypertension together with intracranial haemorrhage. The dosage employed in their patient was 0.2 mg. ergometrine maleate, which is less than half that generally used in this country.—I am, etc.,

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Surgery for Peptic Ulcer

SIR,—In reply to Mr. J. P. Lythgoe and Mr. D. S. Hopton (24 October, p. 1070), our problem with regard to anticholinergic drugs has not been as premedication but as medication before operation. No paper can evaluate the electrical stimulation test without discussing this problem.

Both Mr. Lythgoe and Mr. Hopton postulate that vagal trunks destined for the stomach, and needing division in vagotomy, may lie more dorsal than the trunk giving off the coeliac division, and even "amongst the [muscle] fibres of the right crus." If such trunks do exist they can only arrive at the gastric body and fundus after bilateral selective vagotomy by running a retroperitoneal

course to the duodenum and ascending to the stomach by way of the pylorus. Bilateral selective vagotomy must become the standard surgical operation; it gives the best results in the prevention of post-vagotomy diarrhoea. Our concern is not the possible existence of such hypothetical trunks, it is to find and deal with the main posterior trunk which supplies the coeliac division and to section all trunks, however small, lying on the oesophagus.

I believe the insulin test is of no practical value. I agree with Mr. Lythgoe¹ when he states: "Routine employment of the insulin test post-operatively will not help in the management of the individual patient . . ." and, "Clearly, it would be of great value to have a test which could detect an incomplete vagotomy at the time of operation and would allow the surgeon to make a further search for the remaining branches." This is exactly what the stimulation test does. He goes on: "There is no doubt that the electrical stimulation test can detect persisting vagal branches on the lower oesophagus, and that these account for some cases of incomplete vagotomy."

Clark and Murray² compared the two tests. They found the stimulation test "fallacious in indicating complete vagotomy when the insulin test was later found to be positive" in only one of 100 patients, and in this one case their pressure readings were very low. Clearly there was something wrong with the test in this case. I have used the stimulation test in some 400 patients since 1957; there has been only one recurrent ulcer needing operation. The first 200 were followed in great detail and the results published.³ Only one patient has come to further operation, and he for incomplete section. This, I believe, was due to a technical error in the test in its early history. He has remained well since his incomplete vagotomy was completed.

Without the electrical stimulation test 30% of our own cases would be incomplete according to its standards. Clark *et al.*⁴ studied two series, one with and one without the stimulation test, and wrote: "When the test is successfully applied the number of incomplete nerve sections is negligible. On the other hand, in the absence of the test and despite careful dissection at the lower end of the oesophagus it is estimated that the proportion of inadequate vagotomies is about 10%."—I am, etc.,

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Spontaneous Biliary Peritonitis

SIR,—I was most interested to read Mr. A. K. Dhar's account (7 October, p. 1198) of his three cases of spontaneous biliary peritonitis. I have always been interested in this condition, firstly because the title is a complete misnomer, the condition never being spontaneous, and secondly because the peritonitis, if it exists, is probably not due to bile. Uncomplicated bile in the peritoneal cavity causes minimal disturbance.

In the last four cases of peritoneal biliary effusion that have come my way I have in every one been able to demonstrate perforation of the lower part of the common bile-duct. In the presence of gross oedema and discoloration this is often difficult, and if the perforation is small failure to demonstrate it is probably of no consequence.

Two of these cases were traumatic, one associated with stones and one without obvious cause. May I suggest that in Mr. Dhar's cases it is much more likely that the initial pathology was in the lower part of the common bile-duct and that the pancreatitis was secondary, the result of oedematous obstruction of the closely associated duct of Wirsung?

Some time ago I was just completing an operation for a simple perforated duodenal ulcer when I received an urgent message from the Path. Lab. that the serum amylase, requested without my knowledge, was over 2,000 Somogyi units. I was pleased but not surprised, as it was in line with a view I have held for many years that the common acute oedematous, as opposed to the acute haemorrhagic, pancreatitis is probably always due to oedema from an extra pancreatic cause, the most likely lesion being in the biliary tract. Hence the universally good results no matter what form of treatment, or absence of treatment, is employed.—I am, etc.,

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Management of Respiratory Failure

SIR,—I wholeheartedly agree in principle with Dr. D. C. S. Hutchison and others (7 November, p. 1159) that oxygen therapy should be used in a controlled manner to produce "safe" levels of arterial oxygenation—that is, a P_{O_2} and saturation above those causing dangerous hypoxia but not necessarily in the normal range. I would, however, interpret their findings differently and contend that in fact they point to a simpler regimen than the authors suggest. They perhaps over-estimate the dangers of moderate hypoxaemia and acidemia; I would accept as "safe" an arterial P_{O_2} above 40 mm. Hg, a saturation above 70%, and a pH above 7.22 (H^+ concentration below 60 nanonormal).¹ However, even if their "even safer" criteria (P_{O_2} above 50, pH above 7.25) are applied, their results still point to the following conclusions:

- (1) It is safe to assume that raising (as opposed to lowering) the inspired O_2 concentration to 30% will abolish dangerous hypoxaemia in these patients.
- (2) The pH and P_{O_2} only fell below safe levels when the inspired O_2 concentration had previously been raised above 33% (Cases 2, 3, 7, 9).
- (3) Measurements of pH and/or P_{O_2} were not necessary if the inspired O_2 concentration had not been raised above 33% and the PCO_2 was below 100 mm. Hg.

Our practice is to measure the PCO_2 on admission, and, if it is above 50 mm. Hg, to start O_2 therapy at an inspired concentration of 24½%, thereafter increasing to 28 and (sometimes) to 33% at approximately hourly intervals, provided the patient does not become drowsy or the PCO_2 rise excessively. If the patient does become drowsy, if coughing

becomes ineffectual, and if the PCO_2 rises excessively we employ the other therapeutic measures in the "crescendo" advocated by Dr. Hutchison and his colleagues—while keeping investigations piano. But the therapeutic fortissimo must be achieved within a few hours if severe underventilation, narcosis, and deterioration of the lungs are to be avoided. *We do not begin O_2 therapy with an inspired concentration over 30% and then, when in difficulties, reduce the inspired O_2 ; nor do we wait for clinical evidence of hypercapnia before measuring PCO_2 because, by the time such evidence is clear, the damage is done.*

None of over 50 patients in respiratory failure cared for in this way from admission has developed a PCO_2 over 100 mm. Hg.

If a patient has become unconscious owing to uncontrolled O_2 therapy Dr. Hutchison's results support our experience that the inspired O_2 concentration should not be reduced until steps have been taken to improve ventilation and until the hypercapnia has been brought under control—that is, until the PCO_2 has fallen below 100 mm. Hg. In such cases measurements of P_{O_2} and pH (or bicarbonate) are interesting; measurements of PCO_2 are almost essential.

I have no wish to be reactionary and will welcome the day when all hospitals have facilities for the continuous monitoring of the blood gases immediately available, 24 hours a day, seven days a week. But until that day comes most patients with respiratory failure can be satisfactorily managed with simple facilities already immediately available.—I am, etc.,

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REFERENCE

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Histoplasmosis in Cyprus

SIR,—The occurrence of histoplasmosis in Cyprus reported by Wing Commander D. J. Stoker (26 September, p. 793) is interesting. It is difficult, however, to accept Dr. Stoker's suggestion that the encephalitis in Case 1 was due to histoplasmosis. Although Dr. Stoker does not give the result of a repeat lumbar puncture, he implies in his case history that the patient recovered from the illness spontaneously. This is contrary to the natural history of histoplasmosis of the nervous system, which is invariably progressive in nature unless the patient has received specific antifungal therapy. It is also generally accepted that histoplasmosis of the nervous system only occurs in the disseminated forms of the disease. Dr. Stoker reports no signs of dissemination of the disease except one gland in the cervical chain. If the disease was disseminated, one would expect a palpable spleen or liver, although this is not mandatory.

In any case, may I suggest that the patient be very carefully followed and his cerebrospinal fluid studied at regular intervals. Also that an intensive search for dissemination of the disease (including blood and bone-marrow culture, for instance) be made.—I am, etc.,

Florida, Transvaal,
South Africa.

P. J. CUSINS.