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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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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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