due to post-maturity. From this distance it appears that its primary cause was a poorly developed placenta.

Later in this article, the penultimate paragraph of which I do not understand, he seems to say that most of the difficulties attributed to post-maturity are in fact due to cephalo-pelvic disproportion—badly diagnosed or badly managed. This is a much better line of talk, and is parallel to the strong common sense of A. J. Wrigley's paper (*Proc. roy. Soc. Med.*, 1946, **39**, 569).

In your annotation on this subject (p. 1376), the urbane editorial manner of which converts any criticism of mine into impertinence, you fail in my view to point the moral of these figures from Oxford. The message which I would like you to have proclaimed, undiluted and unequivocal, is that of 171 cases of pregnancy prolonged more than 14 days, no death was due to post-maturity.

I am indebted to Professor Andrew M. Claye for permission to give details of the case I have quoted.

—I am, etc.,

Leeds.

A. AMBERY SMITH.

SIR,—In a paper on post-maturity in your issue of June 16 (p. 1364) I find myself reported as saying that I advise induction of labour whenever a woman goes three days beyond her expected date of delivery. Really I am too old an obstetrician to be caught with so dogmatic a statement. What I did say in the text of the paper to which Dr. D. Latto refers was the moment full term is passed the foetal environment begins to deteriorate. The paper was an attempt to define from the data furnished by cases of true post-maturity what is the sequence of events leading to the onset of normal labour at term, and its main thesis was that this sequence is initiated by the only biological timekeeper present in the system, the foetus itself, a heresy which I have held and taught for twenty years.

In the discussion of the paper the clinicians there, perhaps not without a little kindly malice, posed the unanswerable question of how long should one wait, a question to which the only answer is the standard retort that it all depends, but, believing as I do that post-maturity is an important cause of hard labour and stillbirth, I did express the view that once a woman is three to five days overdue the risks it carries must be carefully assessed and termination considered.

Which view is right, that the intrauterine environment deteriorates in respect of the needs of the mature foetal brain, or Dr. Latto's view that the problem is one solely of disproportion between the head and the pelvis, cannot well be argued here; what, however, is important is that the admitted risks should not be glossed over. Techniques are now safe enough to deal with post-maturity safely.—I am, etc.,

Liverpool, 1.

PERCY MALPAS.

SIR,—Dr. Douglas Latto is to be congratulated on his account (June 16, p. 1364) of 171 cases of prolonged pregnancy with a loss of little over 6% babies. The danger of his article lies in the support which it appears to give to the policy of non-intervention in cases of post-maturity, although he must be given credit for not making an explicit statement to that effect.

It has been shown by the late Sir Joseph Barcroft that foetal asphyxia must of necessity become more pronounced the longer the foetus is retained *in utero*, and W. J. Hamilton and J. D. Boyd (*Proc. roy. Soc. Med.*, 1951, **44**, 489) have recently referred to the alterations in permeability of the placental membrane, which is most permeable to radioactive sodium at the 36th week, with rapid decline in the subsequent weeks. It seems logical to conclude that in all cases of prolonged pregnancy there is a gradually increasing risk of foetal asphyxia. This may be responsible for intrauterine death only in extreme circumstances, but it surely increases the foetal hazard from other dangers, and is particularly prone to cause cerebral haemorrhage with trauma that would not otherwise prove fatal. When S. G. Clayton (*J. Obstet.*

Gynaec. Brit. Emp., 1941, **48**, 450) found no stillbirths that were certainly due to post-maturity, he neglected to include the deaths from cerebral haemorrhage that might not have occurred in the absence of foetal asphyxia. In a similar way Dr. Latto discounts foetal death following administration of "pitocin" and quinine, measures that may prove innocent of danger to hundreds of babies that are not already somewhat asphyxiated.

It would be interesting to have further details of measures that may have been responsible for the low foetal loss in the recorded series. We are not told how many times labour was induced at the 42nd week to avoid further post-maturity, nor how many of the 24.5% of forceps deliveries were indicated on account of foetal distress or were used to anticipate foetal danger. The article has proved the value of expert obstetric care in a world-famous unit, but it would be a pity if it encouraged practitioners to underrate the hazard of foetal asphyxia in post-maturity.—I am, etc.,

Birmingham.

W. G. MILLS.

Non-specific Cervicitis

SIR,—We have been interested in the remarks of your correspondents regarding so-called non-specific cervicitis. Jelinek (March 17, p. 585) mentions the finding of pleuropneumonia-like forms by Durel in a small number of cases. In 40 cases so far investigated we have been able to isolate from all a small pleomorphic coccobacillus which may belong to the corynebacterium group of organisms. Although we have as yet no proof of an aetiological relationship between this organism and the lesion it is interesting to note that as the condition of the patient improves this organism disappears from the discharge.

With regard to treatment, we have previously had most success with gentian violet, but this mode of therapy is disagreeable to the patient and on occasion increases the irritation. More recently we have tried other substances belonging to the same chemical group and have finally chosen the sodium salt of diamino disulphomethyl fuchsonium sulphionate. By itself this compound has only a slight effect, and we have tried the effect of using it with other compounds according to the method of Bigger (*Lancet*, 1950, **2**, 46). In combination with sodium bichlorate the fuchsonium compound has a very satisfactory effect. We have used the mixture in the form of a cream for local application. Seventy patients complaining of vaginal discharge and pruritus have so far been treated, the average duration of the complaint being two years. The cream was applied to the cervix and vagina every second day. After the first or second application all irritation disappeared, and in the average case the discharge ceased after five applications.

The preparation appears to be equally effective in vaginal thrush, but is of little use in trichomonas infestation.—We are, etc.,

Glasgow.

D. MCKAY HART.

C. HUTTON BROWN.

Albumin Antibodies for Rh Grouping

SIR,—Dr. I. M. Tuck has very rightly drawn attention (June 30, p. 1490) to the scarcity of "saline acting" anti-Rh agglutinins for Rh grouping. His description of a modified Chown capillary technique must be applauded as an effort to overcome this difficulty. In the same issue your correspondent ("Any Questions?" June 30, p. 1523), in a most informative answer to a question on rapid Rh grouping, claims that "rapid" methods are liable to give false negative results. If this statement were confined to the Chown capillary tube method I would heartily agree, for in these laboratories we have found the method too unreliable. Nevertheless, I have used a rapid slide test for many years with complete satisfaction. The test used has advantages over the Diamond slide method, perhaps the most important being the use of albumin or incomplete anti-D (titre 1/32) diluted with albumin solution (1 part serum/3 parts 20% flake egg albumin in distilled water). For the test a thick drop of patient's cells suspended in their own serum is

mixed with one drop of the albumin-diluted incomplete anti-D on a glass slide. The tests are incubated in a moist chamber to prevent drying at 37° C. for 30 minutes and read with the naked eye by tilting the slide.

The moist chambers which we use are flat "perspex" boxes 12 by 7 by 1 in. (30 by 18 by 2.5 cm.) with loose-fitting lids, and each contains a piece of wet blotting-paper. These boxes accommodate ten 7-in. (18-cm.) slides each carrying ten tests. Some form of moist chamber is essential for the test. The positive results are discernible in a very few minutes, but are left for 30 minutes to develop complete agglutination.

I regret that space does not permit of a full description of the technique, but, should your correspondents be interested, I will be happy to lend them a colour film describing the method in detail.—I am, etc.,

Queen Charlotte's Maternity Hospital,
London, W.6.

JOHN MURRAY.

Prickly Heat and Heat Exhaustion

SIR,—We were interested in Dr. W. S. S. Ladell's article in your issue of June 16 (p. 1358), in which he suggested that the apparent failure of acclimatization in a subject who had had prickly heat was the consequence of Type II heat exhaustion (Ladell, Waterlow, and Hudson, 1944) and that this variety of heat exhaustion could be considered a disease of adaptation. It is in no carping spirit that we ask what the evidence was for failure of adaptation. The subject had been doing his normal work without symptoms for months, and was apparently able to take strenuous exercise in a most trying environment (effective temperature 94° F.—34.4° C.) for two hours and to such an extent that at the end of this period his rectal temperature was nearly 103° F. (39.4° C.). If the idea of a disease of adaptation is to mean anything, surely the stress to which the victim fails to adapt completely must be of not more than average or "normal" severity. It is difficult to conceive that Dr. Ladell's subject was in an "exhaustion stage" with a markedly diminished heat tolerance at the time of his second test if the whole man is considered and not just the changes in sweat chloride and sweating rate. In any case the interpretation of these laboratory data as indicating a failure of acclimatization depends on the assumption that the subject was normal at the time when he was "first tested soon after being badly sunburned."

Observations of ours (in the press) show that prickly heat by itself does not produce a rise in sweat chloride. The data were obtained by repeated testing of a group of initially normal subjects from the beginning of the hot season at Karachi in 1947. There was no change in sweat chloride in those individuals who developed prickly heat only. A marked rise occurred in the minority whose prickly heat was followed by the skin change first described by Allen and O'Brien (1944) and Wolkin, Goodman, and Kelley (1944) and called by us "mammillaria" (Horne and Mole, 1951).

Not all subjects with mammillaria had symptoms of heat intolerance, but those that had symptoms presented a clinical picture we called "anhidrotic heat exhaustion" (Horne and Mole, 1950), because there was a gross and clinically obvious deficiency in sweating and because this deficiency seemed the immediate cause of the heat intolerance. We were not sure if this syndrome was identical with Type II heat exhaustion, for in its original description there was some doubt whether there was a real sweating deficiency. Prickly heat was an almost invariable precursor in both groups of patients, and the descriptions agreed in stating that it was healed or healing at the time when symptoms of heat intolerance first appeared. Dr. Ladell considered his present case to be a typical, though mild, example of Type II heat exhaustion, although he had active prickly heat at the height of his symptoms and is also described as sweating freely. Symptoms appear to have subsided as the prickly heat improved. In these respects the clinical picture is not very close to the original description of Type II heat exhaustion, and we would be tempted to label the case simple prickly heat.

Subjects with prickly heat are by no means always free from general symptoms, especially after insomnia. There is a good deal of confusion still about the clinical picture of the different varieties of heat effects. How would Dr. Ladell distinguish clinically between prickly heat and Type II heat exhaustion?—We are, etc.,

G. O. HORNE.
R. H. MOLE.

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Ladell, W. S. S., Waterlow, J. C., and Hudson, M. F. (1944). *Lancet*, **2**, 491, 527.
Horne, G. O., and Mole, R. H. (1950). *Trans. roy. Soc. trop. Med. Hyg.*, **44**, 193.
— (1951). *Ibid.*, **44**, 465.
Wolkin, J., Goodman, J. I., and Kelley, W. E. (1944). *J. Amer. med. Ass.*, **124**, 478.

SIR,—I was interested in the article by Dr. W. S. S. Ladell "Changes in Sweating after Prickly Heat" in the *Journal* (June 16, p. 1358). The paper by E. C. Smith (*Trans. roy. Soc. trop. Med. Hyg.*, 1927, **20**, 344) giving the cause of prickly heat as a fungus infection has never been accepted by modern students: the quoted thesis of J. P. O'Brien (*Brit. J. Derm. Syph.*, 1947, **59**, 125) that the disorder is aggravated by deficiency of fat in the skin (not, probably, "on the skin" as quoted) is more in keeping with everyday observations. During the fiercest heat of the summer (June, July, August) in Fields (Masjid-i-Suleiman) cases of severe prickly heat were rare among Europeans, but during the same period in Abadan every European had prickly heat in degrees varying from slight to severe. The severe widespread variety of the disorder is associated with inelastic crusting of the skin in which fungal spores abound, and even the relatively mild manifestations are associated with waterlogging of the skin, which becomes thick and sodden by imbibition of sweat. Sweat contains, in addition to sodium chloride, some glucose and traces of urea, which encourage the growth of microbial organisms in addition to fungi. The natural grease of the skin appears to be deficient in all cases, but it is doubtful whether the application of oily preparations to the surface of the sodden skin will help matters. Prickly heat in the stages short of advanced crusting can be abolished in a few minutes by placing the patient in a cooled room, and so long as the patient remains cooled the eruption will not reappear. Replace the patient in his hot environment, and the eruption appears again in a few hours. It is abolished in a few minutes by cooling, but takes several hours to reappear on heating again. The British private soldier wears a topee, abbreviated shorts, socks, and shoes or boots. He sunbathes in every spare minute until his whole body is a beautiful brown. He becomes immune to attacks of prickly heat. Severe prickly heat is a very unpleasant disorder: the patient cannot sleep and sweating is painful. The most useful remedy is the prickly-heat powder introduced by Dr. D. C. Rennie in 1932; it is cheap and effective and non-irritating. Preparations such as salicylic acid and perchloride of mercury in spirit, "afridol" soap, etc., are most undesirable. As for the "disease of adaptation," C. A. Mills in 1933 described exhaustion of the adrenal glands in conditions such as heat exhaustion. (References in my article in the *British Encyclopaedia of Medical Practice*, 1937, **6**, 400.)

Cooled ambulances, mobile cooled first-aid posts, cooled hospitals, and cooled casualty clearing stations, together with cooled rooms in ships, are absolute necessities for any active operations undertaken in the Persian Gulf littoral. After all, if ammunition can be kept cool, so can human beings.—I am, etc.,

Epping, Essex.

FRANK MARSH.

Spread of Tuberculosis in Scotland

SIR,—Your annotation on tuberculosis (June 2, p. 1251) states, "The difference in the trend of respiratory tuberculosis in Scotland and in England and Wales during recent years is not easily explained." Perhaps this may help.