ferric ions, are added the colour disappears. The chemical nature of the colour complex between ferric ions and the phenolic group is unknown.

REFERENCES
[1] Gibbs, N. K., and Woolf, L. I., *Brit. med. J.*, 1959, 2, 532.
[2] Nellhaus, G., *J. Amer. med. Ass.*, 1959, 170, 1052.

E.S.R. in Pregnancy

Q.—*In the later months of pregnancy the E.S.R. is raised. What are the normal limits? Would 80 mm. Westergren after 1 hour be pathological?*

A.—The association of pregnancy and a raised E.S.R. was first noted by Fahraeus[1] in 1918, while seeking an early test for pregnancy. We now know that the sedimentation rate begins to accelerate from about the tenth week and does so until the later months of pregnancy. This increase is quite variable, and a figure of 80 mm. Westergren after one hour would not necessarily be pathological. Values as high as 100 mm. have been recorded in normal pregnancy. There is a slow return to normality in the puerperium which is reached at about the fourth week post-partum.

REFERENCE
[1] Fahraeus, S., *Acta med. scand.*, 1921, 1, 55.

Ectodermal Dysplasia

Q.—*What is known of the pathology of congenital ectodermal dysplasia and what is the treatment of the offensive smell from the nose it can cause?*

A.—Congenital ectodermal dysplasia is a genetically determined developmental abnormality due to either a dominant mutant gene or to a sex-linked recessive gene in which the female carriers are unaffected. The offensive smell from the nose is due to an atrophic rhinitis. No satisfactory treatment is known for this apart from ordinary hygienic measures. Possibly the use of an antibiotic nasal cream might be of some help.

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[1] Jespersen, H. G., *Acta paediat. (Uppsala)*, 1962, 1, 712.
[2] Moynahan, E. J., in *Congenital Abnormalities in Infancy*, 1963, edited by A. P. Norman. Blackwell, Oxford.

Aluminium from the Pot

Q.—*Is there any danger in leaving cooked food in an aluminium pot overnight, and what is the danger?*

A.—Almost every disease to which the flesh is heir, from diarrhoea to constipation, seems to have been attributed to the use of aluminium pots and pans for cooking. However, these claims have not been substantiated and the use of aluminium utensils is not considered in any way injurious to health. This is because it is scarcely absorbed from the gut and has negligible oral toxicity.

Aluminium occurs naturally in many edible plants. Food cooked in aluminium ware will naturally take up some of the metal from the pan. The amount taken up will depend on such factors as *p*H and duration of contact—i.e., it will be increased by overnight contact. Even if the level is increased by overnight contact it will not be dangerous, since it will not be absorbed. These aspects of the toxicology of aluminium have been usefully summarized by Browning.[1]

It is rather amazing that this question about the safety of aluminium kitchen ware should recur so regularly when there are so many real hazards to worry about. For instance, one of the main dangers of leaving food exposed overnight is the possibility of bacterial contamination. It is interesting that while it is very easy to get people agitated about imaginary dangers, such as the use of aluminium pans, it is often very difficult to persuade them to take a rational course of action to deal with dangers for which there is good evidence.

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Browning, E., *Toxicity of Industrial Metals*, 1961. Butterworths, London.

Notes and Comments

Sulphonamides and Kernicterus.—Dr. P. TURNER (Dunn Laboratories, St. Bartholomew's Hospital, E.C.1) writes: With reference to your expert's reply to this question ("Any Questions?" 3 October, p. 866) there is considerable evidence that sulphonamide drugs may produce kernicterus, particularly in the premature baby. The sulphonamide may compete with bilirubin for binding sites on serum albumin, thus causing a rise in free cerebro-toxic bilirubin. This was first noticed clinically with sulphafurazole,[1] and sulphadiazine and sulphamethoxypyridazine have also been incriminated.[2] Odell[3] and Black[4] have drawn attention to the dangers of giving sulphonamides to the mother just before or during delivery, as these drugs cross the placental barrier.

Dr. BERYL CORNER (Bristol 8) writes: The answer to the question on the relationship of sulphonamides in pregnancy to kernicterus in the newborn baby is somewhat misleading, as it is incomplete. Kernicterus has occurred in newborn premature infants who received sulphafurazole, and at peak bilirubin levels of only 15 mg./100 ml.[1 5] Animal experimental work confirms that sulphonamide appears to lower the permeability of the blood-brain barrier.[6] The probable mechanism for this is that sulphonamides, salicylates, and possibly other substances compete with bilirubin for plasma-albumin-binding and thus increase the availability of diffusible bilirubin.[3] This is of particular importance in the premature infant, where plasma albumin levels are already low.

During pregnancy sulphonamide and bilirubin diffuse across the placenta, and both are normally eliminated satisfactorily by the mother, but if sulphonamide is administered shortly before birth some will inevitably remain in the tissues of the newborn infant and thus temporarily reduce the level of albumin available for bilirubin-binding. This could increase the risk of kernicterus if bilirubin-conjugating mechanisms are inadequate, as in premature babies. Sound advice would therefore seem to be that it is unwise to administer sulphonamide at the end of pregnancy or in labour, particularly if this is premature.

Dr. P. ROBSON (Farnborough Hospital, Farnborough, Kent) writes: I should like to add to the answer to the question on sulphonamides and kernicterus that furan derivatives are known to produce hyperbilirubinaemia in infants. Sulphafurazole is rightly classed as a sulphonamide and is used for urinary-tract infections, but it has a furan group and is known to produce jaundice in the newborn,[1] as is vitamin K. So far only the bones have been picked from the red-herring known as physiological jaundice, and it might be safer to restrict the use of all icterogenic drugs, especially at a time when more babies are being discharged home 48 hours after birth.

Dr. I. ABRAHAMS (West Middlesex Hospital, Isleworth, Middlesex) writes: The answer to the question on sulphonamides and kernicterus states the belief of only some authorities. There are others who are convinced that sulphonamides can cause hyperbilirubinaemia and thus kernicterus by competing with bilirubin for glucuronic acid[7] or plasma albumin.[8] Consequently they discontinue the use of these drugs in the latter part of pregnancy. This is obviously of great importance in the case of a premature baby. It could also be added that drugs other than vitamin K have been incriminated in this respect—e.g., salicylates.

Dr. PAMELA A. DAVIES (The Radcliffe Infirmary, Oxford) writes: Sulphonamides displace bilirubin from the protein-binding sites, and kernicterus has been reported in newborn premature infants given sulphafurazole.[1] When a long-acting sulphonamide such as sulphamethoxypyridazine is given to the mother before delivery very little is found in the amniotic fluid, but the drug passes readily across the placenta and is excreted only slowly by the newborn infant, appreciable serum levels being present so long as four days after birth.[9] Dunn,[10] describing hyperbilirubinaemia in nine infants whose mothers were treated with this drug, suggests a probable association. The work of Kantor *et al.*[11] leads them to imply that sulphonamides should be avoided in labour if there is a previous history of marked neonatal jaundice or any likelihood of rhesus incompatibility. This seems reasonable, as does Dunn's plea that the long-acting sulphonamides be avoided altogether until more information is available.

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[1] Silverman, W. A., Andersen, D. H., Blanc, W. A., and Crozier, D. N., *Pediatrics*, 1956, 18, 614.
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[3] Odell, G. B., *J. Pediat.*, 1959, 55, 268.
[4] Black, J. A., *Practitioner*, 1962, 189, 99.
[5] Bowman, J. M., Rapmund, G., and Harris, R. C., *Amer. J. Dis. Child.*, 1957, 93, 75.
[6] Johnson, L., and Day, R., *ibid.*, 1957, 94, 441.
[7] Lewis, T. L. T., *Progress in Clinical Obstetrics and Gynaecology*, 1964, 2nd edition. J. and A. Churchill, London.
[8] Donald, I., *Practical Obstetric Problems*, 1964, 3rd edition. Lloyd-Luke, London.
[9] Sparr, R. A., and Pritchard, J. A., *Obstet. and Gynec.*, 1958, 12, 131.
[10] Dunn, P. M., *J. Obstet. Gynaec. Brit. Cwlth*, 1964, 71, 128.
[11] Kantor, H. I., Sutherland, D. A., Leonard, J. T., Kamholz, J. H., Fry, N. D., and White, W. L., *Obstet. and Gynec.*, 1961, 17, 494.

Conditioned Inhibition.—Dr. M. TREISMAN (Institute of Experimental Psychology, University of Oxford) writes: This term was introduced not by Wolpe but by Hull, who defines it in his book *Principles of Behaviour*.[1]

OUR EXPERT replies: Dr. Treisman is quite right when he says that C. L. Hull used this term before Joseph Wolpe. He may like to know, however, that conditioned inhibition was in use before Hull's book was published in 1943. Hull himself writes in an article[2]: "Several other forms of negative habits have been elaborated by the reflexologists, such as differential inhibition and conditioned inhibition. . . ."

REFERENCES
[1] Hull, C. L., *Principles of Behaviour*, 1943. D. Appleton-Century Co., Inc., New York.
[2] —— *J. abnorm. soc. Psychol.*, 1930, 25, 200.

Correction.—We regret a printer's error in our "To-day's Drugs" article on "Treatment of Acute Poisoning" in the *B.M.J.* of 17 October. In the paragraph headed "Barbiturates" on p. 995 the phrase "there does not seem to be a place for the more modern stimulant drugs such as bemegride" should have read "there does seem to be a place for the more modern stimulant drugs such as bemegride."