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CEREBRAL BIRTH TRAUMA

IF asked what is the most important form of cerebral birth trauma most practitioners, and probably most obstetricians, would reply "intracranial hæmorrhage." Yet from a practical standpoint—looking at the remote effects of abnormal birth—this is possibly the wrong answer. CRAIG'S instructive study[1] of 126 cases of intracranial hæmorrhage in the new-born brings out not only the many difficulties in diagnosis but also the severity of the effects of bleeding within the skull. Recovery is possible, and he makes a strong plea for "skilled nursing comparable with that ordinarily available to an adult case of pneumonia or typhoid"; but in general the type of lesion that he describes and illustrates seems incompatible with prolongation of life. While supporting his appeal for the better care of such infants, we believe that an even more urgent need is the proper management of another type of birth trauma, summed up in the word "anoxæmia." This is more serious because it is less understood.

In a striking paper[2] SCHREIBER of Detroit, a neurological surgeon, takes the view that asphyxia occulta, as he calls it, is the greatest menace to-day as regards permanent injury to the brain of a new-born baby. Analysing a series of 500 patients seen because of cerebral symptoms persisting or arising after the neonatal period, he has discovered that about 70 per cent. of those whose birth record is available had a history of respiratory difficulty. Seeking for the cause of their failure to breathe properly SCHREIBER finds it in depression of the fœtal respiratory centre by drugs given to relieve or abolish pain during labour. The severity of cerebral symptoms is, he says, in direct relation to the length of the apnœa, and the amount of damage to the brain is in direct relation to the severity of the symptoms. He expounds this thesis of the danger of maternal analgesics in some detail, and quotes the observation of IRVING, BERMAN, and NELSON[3] that when no drugs or anæsthesia were given to the mother only about 2 per cent. of babies failed to breathe; whereas when barbiturates were used the incidence grew rapidly higher, and with Pantopon (papaveretum B.P.C.) and scopolamine (hyoscine) the alarming figure of 67 per cent. was reached. "One of the toxic effects of all drugs used to produce analgesia in labor is depression of the respiratory center" writes SCHREIBER, and he points out that drugs are employed in obstetric practice in doses often much larger than those advised by pharmacologists. For example, a standard pharmacological work states "with ordinary individuals it is not safe to exceed 1/120 grain" of hyoscine, repeating this dose every six to eight hours if necessary; whereas an authoritative obstetric view of the correct dosage is "1/100 grain every half hour for 3 doses and then 1/100 grain every two hours," the average quantity given in a large series of labours being 5/100 grain. By an ingenious diagram he illustrates the phases through which the fœtal respiration centre passes as the lack of oxygen rises from "normal intra-uterine anoxæmia" and "normal birth trauma" to the more dangerous degrees attributable to abnormal conditions, intervention, oxytocics, analgesics, and anæsthetics. The "vital line" ends with asphyxia, continued apnœa, and neonatal death; or (almost worse) with "fixed brain lesions" producing the spastic child, the convulsive child, and the mentally retarded child.

SCHREIBER'S allegations occasion furious thought at this time of increased employment of pain-relieving drugs during labour. If he is right, the risk of respiratory difficulty, and hence of a brain-wrecking lack of oxygen, is by their use multiplied twenty or thirty times. This—and not intracranial hæmorrhage—appears to be the most serious form of cerebral birth trauma, and it arises not from uncontrollable factors but from the administration of drugs in excess of the pharmacological dosage. Here we are faced with another aspect of the problem of "babies who do not breathe," which was discussed in these columns on Sept. 24th.

STEPS TOWARDS INFLUENZA PROPHYLAXIS

IN 1933 it was shown that epidemic influenza is caused by a virus. The work of the next three years brought no suggestion that this virus was of more than one antigenic type: strains were isolated in various parts of the world, and cross-immunity tests in ferrets revealed no significant difference in their make-up. In 1936, however, MAGILL and FRANCIS,[1] using neutralisation tests with sera made in the rabbit, found that two strains, which they had previously considered identical, had definite antigenic differences. Confirmation was soon forthcoming; BURNET[2] in Australia, and STUART-HARRIS, ANDREWES, and SMITH[3] in this country, demonstrated antigenic differences that sometimes went so far as to indicate the existence of separate antigenic types. The bearing of these observations on immunisation against influenza was obvious, and further developments have been anxiously awaited. How many antigenic varieties would be brought to light, and how deep would the differences go? Would it be possible to find what ANDREWES has termed a "master" strain, which had all the dominant antigens of the group and could be generally used for prophylactic

[1] Craig, W. S., *Arch. Dis. Childh.* 1938, **13**, 89.
[2] Schreiber, F., *J. Amer. med. Ass.* Oct. 1st, 1938, p. 1263.
[3] Irving, F. C., Berman, S., and Nelson, H. B., *Surg. Gynec. Obstet.* 1934, **58**, 1.

[1] Magill, T. P., and Francis, T., jun., *Proc. Soc. exp. Biol., N.Y.* 1936, **35**, 463.
[2] Burnet, F. M., *Aust. J. exp. Biol. med. Sci.* 1937, **15**, 369.
[3] Stuart-Harris, C. H., Andrewes, C. H., and Smith, W., *Spec. Rep. Ser. med. Res. Coun., Lond.* No. 228, 1938.