exertion such as digging or mowing a rough lawn with an old lawn-mower. The abdominal wall felt icy to the touch, and after such exertion I would have an acute attack as described. It could not be put down to icy water as we had no refrigerator and usually the water remained tepid after boiling. Stools were negative.

Apart from these acute attacks I normally had three or four loose stools daily during the hot weather. I eventually found that a "belly pad" consisting of about a square foot of woollen vest, lined on the inside with an old piece of "lock-knit" cotton material, tied round the neck and waist, was almost specific. The Chinese (Hakka) people dress their small children in a "belly pad" in the hot weather.—I am, etc.,

Leysburn, Yorks.

ALAN A. CROOK.

Acid Drinks and Sulphonamide Therapy

SIR,—Dr. Petronella Potter (May 10, p. 654) raises a theoretical point on the effect of fruit-juice drinks on the urinary pH, but I don't think she can have taken the precaution of testing it in practice. The same point occurred to me when working with heavy doses of sulphadiazine in a country whose climate required a large fluid intake to maintain adequate urinary output. The intake was largely of fresh orange- and lemon-juice drinks. To test their effect on urinary pH, plain water was substituted for them in one group of patients. There was no significant difference in the urinary pH of the two groups as tested by universal indicator. There therefore seemed no practical justification for withholding the fruit drinks—a welcome conclusion, since the patients' general condition suffered on the water regime, which they did not enjoy taking and would avoid if possible.

As citric and tartaric acids and their salts are organic and liable to catabolism in the body, even Dr. Potter's theoretical arguments are in doubt.—I am, etc.,

Oxford.

C. W. M. WHITTY.

Post-operative Pulmonary Atelectasis

SIR,—Dr. M. H. Armstrong Davison (May 17, p. 695) says that I am tilting at illusory windmills in emphasizing the importance of the routine use of simple postural coughing in the avoidance and treatment of post-operative atelectasis. I was not trying to instruct Dr. Davison himself; I think that would indeed be tilting at an illusory windmill because clearly we are in general agreement; but I am certain that we can all do a most important service by emphasizing the principles of simple prophylaxis and treatment which are not yet sufficiently known.

Bronchoscopic aspiration has many advantages in proper circumstances. The object of my letter was to protest against the fostering of the belief that when confronted with a post-operative atelectasis one should at once perform or ask for a bronchoscopy. It is true that anaesthetists should be able to perform bronchoscopic aspiration, but it is going to be a long time before there are enough anaesthetists up and down the country skilled enough to do so. In the meantime post-operative sputum retention is a very common occurrence in all hospitals, great and small. Its ill-effects can be avoided or overcome in the majority of cases by a simple drill available at once in every one. I find that there is very widespread ignorance of this fact and I think that all teachers have a duty to emphasize to house officers and nurses that an efficient remedy lies in their own powers and should be used. Bronchoscopic aspiration should be reserved for the small proportion of cases that fail to respond at once.—I am, etc.,

London, W.1.

R. C. BROCK.

Use of Drugs in Asphyxia

SIR,—For the correct use of drugs in asphyxia it is at least desirable to know something of the physiology of respiration. Prof. Barcroft's recent researches into foetal respiration have shown us a whole new mechanism of respiration which is hardly hinted at in the normal physiology textbooks. They serve to emphasize the essentially reflex nature of respiration. So far as the brain is concerned normal ordered respiration involves a number of centres at various levels in and above the medulla—*asphyxia*, "blackouts" (in flying, etc.)—these centre from above downwards; and in cases where the onset of respiration

is postponed, the possibilities lie in the establishment of a reflex gasp involving some cutaneous or other peripheral stimuli acting at the lowest level—i.e., low down in the medulla. When once respiration is properly established and the brain fully oxygenated, the respiration is thenceforward regulated by the well-known mechanism described by physiologists. Incidentally Prof. Barcroft shows that in the foetal sheep the earliest respiratory movements are elicited by touching the snout area alone, and this is, of course, the area on which the lamb normally drops when it is born, thus providing the maximum cutaneous stimulus to the most sensitive area.

In view of Barcroft's researches, the most effective way to combat asphyxia neonatorum would seem to be to apply a cutaneous stimulus—as in the traditional slapping or by directing a vigorous jet of oxygen on to the nose or into the mouth—or, if that failed, to give some drug which would enhance the sensitiveness of the nervous system so that it will respond to stimuli to which it otherwise would not—in other words, to give something in the nature of a convulsant. This justifies the old-fashioned recommendation of giving as much as 1/400 gr. (0.16 mg.) of strychnine (now, I think, never used, though I refer to this again later) and the more modern use, which I have described several times, of cardiazol-ephedrine. Cardiazol is a well-known convulsant, and in cases *in extremis* I have used it successfully in doses which, in a fully oxygenated baby, are certainly convulsant. (The largest dose I have used, and with complete success, was for a baby who failed to make any response for 15 minutes, but finally did so after receiving 0.5 ml. intramuscularly and then two doses of 0.5 ml. intracardially.)

This theoretical discussion, with its slight experimental demonstration, clears the way for a consideration of the possible bearing of Barcroft's work on the drug treatment of asphyxia in later life. We all know that asphyxia or high dosage of carbon dioxide depresses the sensitivity of the respiratory centre in the medulla, so that finally respiration ceases whatever the concentration of carbon dioxide may become. The question then becomes: Does the original basal or protopathic mechanism of respiration—i.e., responding to cutaneous stimuli instead of to asphyxia—still persist, or is it also abolished with the more normal pattern? I have already mentioned that Barcroft found that at a late stage in foetal life the cutaneous response persists in the foetus when the oxygen tension in the cerebral circulation is almost inconceivably low—i.e., when the response to asphyxia is practically abolished. There are various clinical observations which suggest that in adult life an early developed mechanism may be capable of functioning even though the normal later one is not—e.g., in insensibility due to cerebral haemorrhage, etc., the involuntary basal functioning of certain organs (bladder, etc.) shows itself even though it may have been suppressed in man for more than half a century, while in some types of heart disease the ventricle may reassume its own fundamental rhythm even though for 70 years or more it has followed quite a different one impressed by the auricle. If this is so, the logical way to treat a case of asphyxia would be to try to utilize the original but dormant cutaneous method of stimulation until the re-oxygenated brain reasserted its normal method of control.

I have described in the *B.M.J.* and repeated at the A.M.C. the case of a boy who was immersed in the river for about 15 minutes and who was responding hardly, if at all, to efficient artificial respiration. I injected 1.0 ml. of cardiazol-ephedrine and 1/30 gr. (2.1 mg.) strychnine sulphate, turned him over and gave him a few vigorous slaps, with the result that within a minute he was crying lustily and continued to do so for an hour or so. He finally made a complete recovery in spite of a raging pneumonia. This is only one case, but it does suggest that the injection of convulsants in cases of asphyxia may prove a most valuable and rapid method of treatment. I stress the importance of rapidity, for though cases have recovered after prolonged artificial respiration, all the evidence we have shows that the brain is extremely liable to damage from complete lack of oxygen, and if we can reduce the period of asphyxia we can—if it is not tautology to say so—hope for a much greater percentage of complete recoveries.

It is worth mentioning in this connexion that our fathers constantly used injections of strychnine as a respiratory stimulant, but this practice has fallen into desuetude since the physiologists proved that in the laboratory strychnine has no such