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Correspondence

Letters to the Editor should not exceed 500 words.

Does the Community Care?

SIR,—Now that you have perpetrated in print (17 September, p. 655) a gibe which has been doing a round in psychiatric circles for some years past (Does the Community Care ?), is it not time that the overworked phrase received a precise definition ? It is not clear why a long-stay unrecovered psychotic who is excellently cared for in a good large psychiatric hospital should not be considered as in the care of the community, whereas a similar type of patient who is cared for in no better fashion in a small local-authority hostel should be considered " in community care." In either case, directly or indirectly, the financial burden falls on the community.

A far more important criterion is whether individual members of the community take a personal interest and help the patients in some practical manner. Here the progressive psychiatric hospital can claim a higher level of " community care." A flourishing League of Friends, a good liaison with other voluntary organizations such as the Women's Voluntary Service and the Women's Institutes, enables individual members of the community to give active help and participate in the care of a long-stay patient. It is doubtful whether small hostels receive such attention. Moreover, these hostels seem to be somewhat in the nature of white elephants, being almost half unoccupied. Would it not be wiser to spend the limited money available in increasing the inadequate numbers of mental welfare officers and social workers rather than on bricks and mortar, so that strictly genuine community care would be further encouraged —namely, to enable more harmless unrecovered psychotics to live in a family situation at home, be it with relatives, with friends, or with enthusiastically minded strangers as at Gheel ? This would give the phrase community care an authentic significance, and it would cease to be the target of gibes.—I am, etc.,

Weybridge, Surrey. I. ATKIN.

Management of Third Stage

SIR,—While I agree in general with an active policy of management for the third stage of labour, combining the administration of Syntometrine and umbilical cord traction, as advocated by Drs. J. R. Fliegner and B. M. Hibbard (10 September, p. 622), it is worth recording that serious accidents can occur. Active management of the third stage of labour has been practised routinely in the obstetric unit at Salisbury since December 1959. In the past 16 months we have had three cases of acute inversion of the uterus.

The first one occurred in May 1965 in a 2-gravida, aged 22. The uterus became completely inverted with the placenta still adherent, and after detaching the placenta the inversion was reduced by O'Sullivan's hydrostatic method. This patient suffered severe post-partum headaches which were thought to be the result of over-transfusion. She subsequently showed signs of super-involution and developed secondary amenorrhoea.

The second case, in February 1966, occurred in a primigravida aged 21 who required an outlet forceps extraction under local anaesthesia. The Brandt-Andrews technique was employed for delivering the placenta, and after the placenta had been delivered a small round mass was found in the vagina, which was identified as the fundus of a partially inverted uterus. This was replaced under general anaesthesia without difficulty and the patient made a rapid and complete recovery.

The third case, in April 1966, occurred in a cottage hospital twenty miles distant from the main unit. The patient was a primigravida aged 18 whose labour had been uncomplicated until delivery of the baby. She was given 0.5 mg. of ergometrine intramuscularly with delivery of the anterior shoulder, following which the placenta was delivered, apparently without cord traction. The patient then became shocked and pulseless, so the Flying Squad was summoned; estimated blood loss 30 fl. oz. (850 ml.). After setting up an intravenous transfusion vaginal examination revealed the fundus of an inverted uterus palpable in the vagina. After rapid resuscitation manual replacement of the uterus under general anaesthesia was performed and the patient made a complete recovery.

While this last case might possibly have been an instance of the very rare condition of spontaneous inversion of the uterus, it more probably reflects the way in which the third stage was managed and I think there is little doubt that cord traction played a part in the production of acute inversion in the two other cases. It is hardly necessary to add that all personnel concerned with the management of the third stage of labour are repeatedly warned that cord traction is *never* permissible unless the uterus is firmly contracting under the influence of the oxytocic drug administered, which in our cases is usually Syntometrine. The two midwives and the doctor concerned in the cases described above all stated that the uterus was firmly contracted before attempts were made to deliver the placenta.

Acute inversion of the uterus in the third stage of labour has always been regarded as a very rare but serious complication. There seems little doubt that this hazard is increased if an active policy of management of the third stage of labour is practised.—I am, etc.,

General Infirmary, Salisbury. M. R. FELL.

Detection of Latent Pernicious Anaemia

SIR,—In a recent leading article (21 May, p. 1249) it has been stated that the detection of parietal cell antibodies is not of diagnostic value in pernicious anaemia because these antibodies are not specific for this condition. The detection of intrinsic factor antibodies was considered a useful contribution to the diagnosis of suspected and of early or " latent " cases of this disease. Because pernicious anaemia can be diagnosed relatively easily by means of the other available methods, the diagnostic use of the immunologic procedures was not advocated. Immunologic studies usually give no additional information of diagnostic significance when patients present themselves with—sometimes irreversible—clinical manifestations of vitamin-B_{12} deficiency, but if one wants to prevent the development of impaired health in people who may acquire pernicious anaemia the method for the detection of parietal cell antibodies may have some practical value.

This opinion is based on the results of the studies of Adams *et al.*[1] and of our own group,[2 3] who found that the detection of parietal cell antibodies in patients without pernicious anaemia usually is indicative of the presence of chronic gastritis. In a more recent study[4] we investigated the histological appearance as well as the intrinsic factor secretory capacity of the gastric mucosa in 69 parietal cell antibody-positive subjects who were not suffering from pernicious anaemia. Among them there were 19 individuals with and 50 without hydrochloric acid in their gastric juice after stimulation of their gastric secretion with 0.5 mg. histamine diphosphate given by subcutaneous injection. In addition we tested the sera of these subjects for the presence of intrinsic factor antibodies and estimated the serum vitamin-B_{12} level in 33 of them. The different situations observed within the group of 69 parietal cell antibody-positive individuals were arranged according to the degree of the morphological and functional changes of the gastric mucosa. This disclosed a clear gradient from functionally intact fundus glands without detectable structural changes to complete atrophy of these glands with loss of hydrochloric acid and intrinsic factor secretion. In some subjects with severe gastric changes low serum vitamin-B_{12} values were found to be present. Histologic investigations of the gastric mucosa in parietal cell antibody-positive individuals with achlorhydria showed diffuse changes which contrasted with the focal nature of the chronic gastritis in a group of 33 achlorhydric subjects without parietal cell antibodies. Despite the impairment of hydrochloric acid secretion in the latter subjects all but two of the 26 individuals in which a urinary excretion test was performed were able to absorb a normal amount of vitamin B_{12}.

On the basis of these observations we believe that parietal cell antibody-positive individuals do have a particular type of chronic gastritis which interferes with hydrochloric acid secretion and intrinsic factor production, and which, therefore, may terminate in the development of pernicious anaemia. Apparently parietal cell antibodies already occur in the very early stages of this type of chronic gastritis, which is genetically controlled.[4] Antibodies to intrinsic factor