Although this is a small series and the results are based on clinical impressions only, it appears that chlorpropamide is of definite value in the treatment of Parkinson's syndrome. Although so far I have not been able to study the effect of placebos, in view of the nature of the clinical conditions in these patients I am convinced that this is a real effect of the drug and that chlorpropamide is of value in the treatment of Parkinson's syndrome.

Chlorpropamide has also been given similarly to seven cases of chronic long-standing disseminated sclerosis and one acute early case, with distinctly promising results. Of seven cases of angina pectoris, five would appear to have improved. A further controlled trial in these conditions is indicated.

It must be fully realized that so far there has been no evidence of a cure of these various conditions being effected. The effect of the drug has been to produce in many instances a definite amelioration of crippling symptoms, and in the favourable cases much more than has ever been produced by other drug treatment. The mode of action of chlorpropamide in these conditions is unknown, but two possible theories have been advanced. Firstly, Fearnley and Chakrabarti[1] have shown that it will increase fibrinolytic activity in the fasting state, but it is difficult to explain the effect in Parkinson's syndrome after such a short period as five days on this basis. Secondly, Reinert[2] has shown that it will increase the glycogen uptake of cardiac muscle and increase the rate of recovery of anoxic cardiac muscle. In addition this drug decreases lactic-acid production, and these changes may well be responsible for the beneficial effect in angina. These results are encouraging, and there is reason to believe that further work on this and related compounds may lead to a significant advance in the treatment of these conditions.—I am, etc.,

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Keloids after B.C.G.

SIR,—I read with interest the paper "Observations on Vaccinating Schoolchildren with Danish Fresh B.C.G." by Dr. K. Neville Irvine and Mr. A. Barr (October 15, p. 1119). Our views on the local reaction to B.C.G. and its relation to allergy seem to swing like a pendulum. Hertzberg[1] expressed the opinion generally held in Scandinavia that there was a positive correlation between the size of the local reaction and the level of post-vaccination allergy. The experimental studies by the U.S. Public Health Service[2] supported the generally held belief that the immunizing power of B.C.G. depended upon the viability of the vaccine and showed that the best immunity in animals was produced by vaccine that also produced the strongest degree of tuberculin allergy. But in contrast to the opinion expressed by Hertzberg and other B.C.G. workers the size of the local reaction (although a sensitive indication of the total number of bacilli, living or dead) was not a reliable guide to the degree of immunity induced by vaccination. For field evaluation of the results of vaccination, the lesion could not be taken as a measure of the potency of the vaccine.

In recent years we find a renewed interest again in the use of the local lesion as an index of the potency of B.C.G. vaccine in place of the post-vaccination tuberculin test. Heaf[3] has suggested the desirability of measuring the local reaction six weeks after vaccination, and retesting only those in whom it was less than 4 mm. in diameter. Dr. Irvine and Mr. Barr have now more or less repeated the same suggestion.

I am afraid that the method proposed, though undoubtedly welcome, can be of only limited application in the Tropics, because the keloids that occur at the vaccination site in a considerable proportion of cases may mask the true size of the local lesions. In India, Dr. Kul Bhushan, Research Officer, All-India B.C.G. Assessment Team, has observed the occurrence of keloids in large numbers after B.C.G. vaccination. For instance, in his report to the Indian Council of Medical Research for 1958–59[4] he has reported an incidence varying from 1.8 to 59% among the groups examined by him during that year in Assam, coastal areas of Orissa, and Bombay. Unfortunately there is no information in the report regarding their size. The incidence seems to differ in different parts of India, being generally higher in the hot and humid coastal areas than elsewhere. I have therefore been greatly interested in the prevention of this annoying complication, which does not seem to be peculiar to India but is also known to occur in other countries in South-east Asia.

Certain characteristics of the post-B.C.G. keloids may be mentioned. They generally occur at the site of vaccination on the shoulder and not on the forearm after the tuberculin test, although the amount of trauma is identical in both cases. They therefore appear to be different from keloids described in surgical textbooks and are perhaps of different aetiology. They seem to be caused by some ingredient in the vaccine, the B.C.G. bacilli or one of the components of the suspending medium, which is diluted Sauton. I had suggested at the meetings of the B.C.G. Subcommittee of the Indian Council of Medical Research more than once that studies should be set up to determine, if possible, the causative factor by injecting different groups of children (in selected areas where keloid formation was known to be high) with the various components of B.C.G. vaccine and observing the local reactions that may develop. To my knowledge, the suggestion has not been taken up yet.

In this connexion, may I venture to suggest that the development of keloids after B.C.G. vaccination may in some way be related to non-specific tuberculin allergy? My reasons are these. Like keloid formation, non-specific tuberculin sensitivity also is prevalent in low-lying humid areas like the coastal regions of India and less frequent at high altitudes. This may, however, be a chance coincidence. Keloid formation does not appear to be a problem after B.C.G. vaccination in the Scandinavian countries, North U.S.A., etc., where there is little or no non-specific tuberculin sensitivity. Between these two extremes, the incidence of keloids seems to depend on the level of prevalence of non-specific tuberculin sensitivity. I hope those interested in the problem and possessing the necessary facilities will undertake controlled studies to determine the causation of this unpleasant complication.—I am, etc.,

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