I welcome, however, Dr. Bieber's unexpected announcement that he and his co-authors "did not claim that the shift [to heterosexuality] was permanent". He stresses now that all they reported was the temporary sexual status of the patients at the termination of treatment.

It certainly was not made clear in the book that the therapeutic claim applied only to the period before treatment came to an end, and that there was no follow-up of the patients. In fact, the reader was led to think otherwise. In Table XI-1 (p. 276), for instance, the authors speak of the sexual status of patients as reported "at the *final follow-up inquiry* in June, 1960" (the investigation having been started in June, 1952). When describing Case No. 166 (p. 296), the authors are even more explicit: "At last follow-up, three years after completion of psychoanalysis, the patient reported . . .". (My italics in this paragraph.)

I realize now that the authors studiously avoided using the word "cure", but the way they expressed themselves could easily be misunderstood. Take this statement (p. 318), for example: "Many homosexuals became exclusively heterosexual in psychoanalytic treatment." It will be interpreted as claiming actual cures, as we have all become conditioned to the assertion that psychoanalysts only report therapeutic results which are reliable and lasting.

I am sure the authors will be quoted as having demonstrated that many homosexuals can be cured through psychoanalysis. A follow-up enquiry would therefore seem to be all the more obligatory now to put the record straight. As there may be justifiable objections to independent observers, I suggest that the analysts might get in touch with their former patients to ascertain their present sexual status. Some seem to have done so already. It is only a second-best solution, but it has the advantage of being more easily organized.

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DISFRANCHISED?

DEAR SIR,

As far as absent voter arrangements for hospital patients were concerned, it appears that H.M.(55)65 was still the authoritative document at the time of the recent General Election. Under this authority a patient in a psychiatric hospital situated within the same parish, borough or urban district as the qualifying home address at which he was on the electoral roll, was not entitled to absent voter facilities if he could not be allowed out without detriment to his own health or the public's interest, to attend a polling station either alone or with friends or relatives.

The same restriction did not apply to the physically ill, and yet it is conceivable that under these provisions an informal patient, for example, with severe phobias of crowds and open spaces, who had been advised to remain in hospital for the time being in the interests of his own health, could thus have been unreasonably disfranchised. There might also have been some detained patients, not allowed out of hospital in the public's interest, who were both capable and desirous of expressing their political opinion, although similarly excluded from doing so.

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CALCIUM THERAPY IN DRUG-INDUCED PARKINSONISM

DEAR SIR,

In a recent article (Lichtigfeld, 1964) it was proposed that the interference with the action of calcium might be a factor in the production of druginduced Parkinsonism. Evidence was presented supporting the view that hypo-calcaemic states may interfere with nerve cell function in the basal ganglia. It was concluded that calcium administration may control some of the extra-pyramidal symptoms caused by tranquillizers.

Further to this idea, some work done on postencephalitic Parkinsonian patients lends weight to the above suggestion. The use of maintenance doses of calcibronat has been shown to reduce the number of post-encephalitic oculogyric crises occurring in these patients (Duensing and Meyer, 1938; Renner, 1941; Brandt and Brandt, 1956). Intravenous injections of calcibronat often aborted the crises and sometimes shortened them (Duensing and Meyer, 1938). Whether the bromide component is absolutely essential to the success of the combination is open to question, as the following case history illustrates. Duensing and Meyer mention a case (No. 10) that did not respond to bromide therapy. Injections of calcium were then given on alternate days for some weeks, and this treatment regime produced two 16-day periods of complete freedom from crises over a three-months period. The attacks resumed their old frequency when calcium medication ceased, but they responded to calcibronat when this therapy was started at a later date. Therefore it is likely that the addition of other sedatives to the calcium would have as good an effect as the bromide.

The site(s) of action of calcium in the brain has been the subject of much work. Apart from sites previously mentioned, it appears that the calcium content of the ventricular fluid may also play an important role. The experimental work of Demole is quoted by Duensing and Meyer. Demole showed that calcium salts injected into the region of the wall of the third ventricle produced sleep. The work of Huggins and Hastings has been mentioned previously. They showed that by injecting isotonic sodium citrate intra-cisternally in dogs, marked opisthotonus was produced.

These results point to a possibility that antiparkinsonian drugs may exert an action in the region of the peri-ventricular grey matter; an action that may be responsible, as in the case of calcium, for some of their therapeutic activity. Tranquillizers have been shown to be highly active when given by the intra-thecal route (Verster, 1963). This action could also be mediated via the peri-ventricular grey matter.

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