(*Brit. J. Psychiat.*, February 1965, 195) in which he explicitly states: "The sexual state at termination was reported and *that was all we reported*. We did not claim that the shift was permanent" (Dr. Bieber's italics). They did not even attempt to find out how long the shift lasted. Weeks? Months?

Dr. Clifford Allen states quite properly that his "successes were confirmed by follow-up". I therefore presume that he is as interested as I am in establishing the truth, and that he will join me now in requesting Dr. Bieber and his colleagues to round off their investigation by a follow-up study. But will they have the courage to do so? After all, this might bring down to realistic proportions the therapeutic successes they have never claimed, but which are attributed to them so generously.

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THE EFFECT OF SODIUM AMYTAL ON AUTONOMIC AND MUSCLE ACTIVITY IN PATIENTS WITH DEPRESSIVE ILLNESS

DEAR SIR,

We are prompted to comment on the recent paper by Martin and Davies, "The Effect of Sodium Amytal on Autonomic and Muscle Activity in Patients with Depressive Illness" (February, 1965, pp. 168–175). In this and in earlier study (1962), the authors reached the unwarranted conclusion that the digit-doubling method of determining sedation threshold is unsatisfactory.

As introduced by us in 1960 the procedure involved combining the digit-doubling task with intravenous sodium amytal administered as a continuous infusion. In their first attempt to repeat the work, Martin and Davies, using instead a discontinuous injection procedure, not surprisingly found end-points of sedation difficult to detect, due to fluctuations in consciousness. Changing, in their second experiment, to a continuous infusion method, the authors rather surprisingly abandoned the digit-doubling technique on the grounds of its previous inefficiency! In fact, the only study to replicate the original procedure exactly (Moffat and Levine, 1964), substantially confirms our own experience over several years in nearly 300 subjects that the technique is a simple, reliable method of determining the sedation threshold.

Surprisingly, too, Martin and Davies do not discuss the peripheral action of sodium amytal which is known (Goodman and Gilman, 1955) to impair transmission through autonomic ganglia and have a direct influence on blood vessels. Such effects may seriously invalidate the use of barbiturates for manipulating "arousal level" as monitored via autonomic indices.

Finally, may we add that it is difficult to evaluate the study adequately in view of the imprecise description of the clinical material and the lack of clarity in presenting the statistical analysis, particularly the correlations between the various physiological measures.

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DEAR SIR,

Our conclusion that the digit-doubling method of determining sleep thresholds is unsatisfactory for use *with depressive patients* is based on our data. Neither Claridge and Herrington nor Moffat and Levine used depressive patients. Is it our conclusion that is unwarranted—or possibly theirs?

Our findings would surely surprise no one, since co-operation, retardation and verbal responsiveness are severely impaired in some depressive patients, as indicated in our first paper (1962, pp. 469 and 472) and our second (1965, p. 171), and as discussed independently by Moffat and Levine. Even when good co-operation is achieved initially from depressives, they often find it "too much of an effort" to continue with the digit-doubling task. Subsequent checks have shown that a poor and erratic performance on this task occurs with severely depressed patients even *in the absence of sedative drugs*.

It obviously needs to be stressed that we have never aimed to replicate the work of Claridge and Herrington, but that of Shagass on *depressed* patients; however, we gladly incorporated their digit-doubling technique in our first experiment in the hope that it would introduce a more objective method of determining sedation thresholds. We abandoned it because it became obvious that the method was inapplicable to some of our depressives.

It would be useless to discuss Claridge and Herrington's point on "arousal" since we were not concerned with this concept.

Finally, the problem of evaluating studies which are compelled from lack of publication space to drastically limit data presentation is one with which we sympathize. Needless to say we should be extremely glad to supply Claridge and Herrington with the detailed clinical descriptions and statistical analyses which we were compelled to cut from our first draft of the paper.

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ACEDIA: ITS EVOLUTION FROM DEADLY SIN TO PSYCHIATRIC SYNDROME

DEAR SIR,

In common with many psychiatric authors, Dr. Altschule (*Brit. J. Psychiat.*, February, 1965, pp. 117–119) pays little attention to those for whom the spiritual disorders he discusses are not reducible to psychiatric syndromes. There are still many people for whom the title of the paper might contain the word "devolution" rather than "evolution", because of a debasing and falsification of concepts. It is a trifle too bland to imply that knowledgeable opinion agrees with the assumed improved concepts of modern and psychodynamic psychiatry in these matters. The question of personal responsibility for the mental attitudes discussed is assumed to be answered on some basis of automatism, as in psychiatric syndromes.

Authors dealing with such matters might give more weight to the fact that there is still much alternative theory embracing issues of choice and moral responsibility; theory often closer to the original concepts, towards which a patronizing attitude is too often shown in psychiatric writings.

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Dr. Mark D. Altschule writes:

"I strongly agree with Dr. Flanagan in the main, and especially with his closing paragraph. As regards the rest of his letter, most of it is irrelevant: the paper was an *account* of what has happened and not a *judgment* on what has happened. Dr. Flanagan falls into a serious error in the last sentence of his first paragraph when he assumes, if I read him correctly, that the aetiology of all psychiatric syndromes involves some form of automatism."

E.C.G. ARTEFACTS AND POLARIZATION OF THE BRAIN

DEAR SIR,

In view of the three very interesting articles which you published on direct current polarization of the human brain (November, 1964, pp. 768–799), I thought that an interesting artefact which we came across in a somewhat similar endeavour might be worth while mentioning. Some time ago we became interested in the possible psychic effects of passing low levels of direct current through the human brain and did so in a few subjects. Unfortunately we observed them too briefly and superficially to note the interesting effects reported in the previously-mentioned articles. Amongst other physiological parameters, these patients' electrocardiograms were monitored, and an effect was noted which may be of interest to those considering utilizing this technique. A polarizing current was passed through our subjects via a cranial electrode in the shape of a skull cap and an electrode plate at the base of the spine. When the current was turned on we noted an instantaneous deflection of the SP segment of the E.C.G.; when the current was switched off this effect instantaneously disappeared. When the head was made positive with respect to the caudal electrode the deflection was upward, and when the head was made negative the deflection was downward. Figure 1 shows the

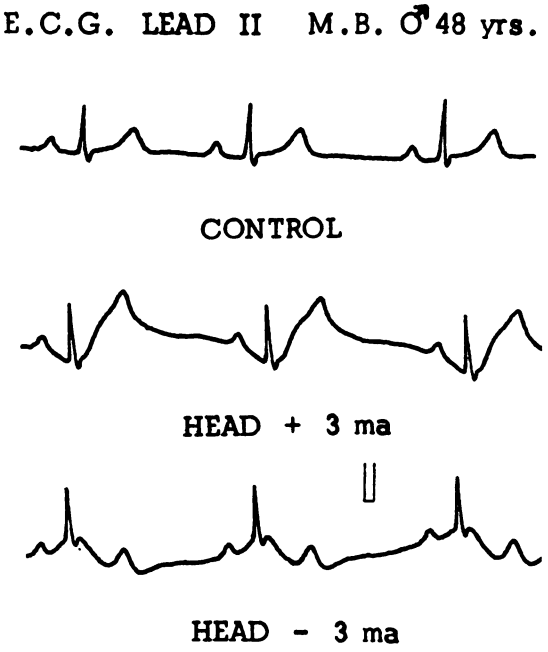

FIG. 1.

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