characteristics of all other learning curves, then the conclusions of the rehabilitation studies are unsupported. This, I submit, is poor logic. Even if the limiting contentions were true—and Phillips has produced no good evidence that they *are* in relation to all the studies—this could only mean that alternative explanations *might* account for the direction of the findings if and when appropriately tested, because Phillips has not drawn on internally conflicting or contradictory figures or test results. It would be poor logic on my part now to suggest that the introduction of Workshop Therapy to all mental hospitals at an even more realistic level should be halted pending definitive answers to some remaining theoretical issues.

Yours faithfully,

VERNON HAMILTON.

Postscript. Miss Phillida Salmon, co-author of the original paper (Hamilton and Salmon, *J. Ment. Sci.*, 1962, p. 505) wishes to associate herself with these comments.

DEAR SIR,

May I comment briefly on three of the points raised by Dr. Hamilton?

1. He now cites another study (Hamilton, *Brit. J. Psychiat.*, 1963) in support of the superiority of workshop therapy. Unfortunately there are seventeen inconsistencies, implying at least twenty-one errors, in its Tables (details available on request), and without reliable data it is not possible to know what to make of it. In any case, I argued not against the *proposition* that workshop therapy is superior (which may very well be true), but against the *inference* from the results of Hamilton and Salmon (1962) and Hamilton (1964) to the proposition, i.e. that it was unsupported by the evidence.

2. He argues that the initial between-group differences in each study are insufficiently marked to be responsible for the different trends shown by the groups. But these trends are themselves of doubtful significance. I think it would be better science not to interpret them at all, but if they are to be considered, as he has done, then largely insignificant initial between-group differences should, in all consistency, be considered as well.

3. In his final paragraph he reformulates my arguments, and submits that his reformulation is poor logic. I agree: but it does not accurately represent my position, which I had better clarify. There were, in the above pair of studies, two separate sources of possible bias in favour of the workshop group: its larger N and its lower initial position on a negatively accelerated improvement curve. It is not possible to say whether the (doubtfully significant) greater improvement trends in the workshop group resulted from one, or the other, or both, or from a real superiority of workshop therapy, and the inference that the latter is the case is unjustified.

Yours faithfully,

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PHENOTHIAZINE EFFECT ON HUMAN ANTIBODY SYNTHESIS

DEAR SIR,

Drs. J. C. Saunders and E. Muchmore have done good service to your readers by bringing to their attention an important but still little recognized potential hazard of phenothiazine medication. Their paper (12) unfortunately omits to quote experimental work which has already been carried out in this area. The significant contribution which they themselves have made cannot therefore be viewed in proper perspective. Moreover, they may give the impression that their findings lack support from other quarters.

Eight years ago Goldman (3) reported that infections are numerically the most frequently encountered complications in the institutional use of chlorpromazine and reserpine, while one year before that Rosenow (11) reported that he found the titre of antibodies to a haemolytic streptococcus to be significantly lower in patients on chlorpromazine medication than in a comparable non-tranquillized group of patients.

Some of the early experimental work with various species of animals and various pathogenic organisms (1, 2, 6, 9, 10) led to inconsistent conclusions.

More recently, this writer (4) has shown in a carefully controlled experiment that in mice to which were administered various dosages of chlorpromazine and *S. enteritidis* inocula increased daily dosage of chlorpromazine shortened the average length of survival for each level of *S. enteritidis* inoculation. The relationship was highly significant ($p < \cdot 005$). Blood cultures taken from various groups of mice during the experiments demonstrated an earlier onset as well as a more prolonged *S. enteridis* bacteraemia in those infected mice which were on chlorpromazine medication. A similar finding was previously reported by Maral and Cosar (7) in their experiments on tranquillized rabbits inoculated with pneumoccoci. These observations have subsequently been confirmed by other workers.

Although interference with the immune response of the organism is implicated, other mechanisms, probably of equal importance, are also involved. Among these one may mention, for example, impairment in the function of the reticulo-endothelial system (8) and impairment in the phagocytic action of leucocytes (5).

It would appear, then, that chlorpromazine and probably also other phenothiazine compounds have a very definite effect on the capability of the body to mobilize various biological defences, including antibodies, against infection, and that due consideration ought to be given to this factor before and during the administration of such medications.

Yours faithfully,

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