March 19, 1996

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Dear Greg,

What a terrible story about Anthony Bouckoms! I liked him very much; last November I invited him to join me and Charlie Welch in an ECT symposium at the Boston APA meeting. He did well, and he told us of the changes in progress at Hartford Hospital. Pity, such an untimely personal and professional loss; more than doubly sad for his wife and remaining children.

I have read the article on catatonia with interest. My suggestions -- I cannot read any article without and editorial pen in hand -- are in the text and margins. Overall, I think the essay presents the experience well. But, if we are to be of service, we should be more definite about the conclusions which we wish to leave as the 'messages'.

The first message is to recognize catatonia, and that is done well. Andy and George will probably wonder why you do not recommend the use of their handiwork -- the catatonia rating scale. By the time you get this note, it will be published [it is in the March number of the *Acta*]. Instead of tables 4-6, should you not adopt the rating scale and test method? We will surely have no objection to such use. [There is one small point -- both George and Andy saw the scale as their 'special' handiwork and in the text of the article labeled the scale as the 'Bush-Francis Catatonia Rating scale' or BFCRS -- a cumbersome title that will not sell well. I believe you can adopt the scale without the mnemonic label, as I have done elsewhere.]

Similarly, you may wish to amplify the paragraph discussing the Bush *et al* experimental findings in incidence of cases and treatment results, much as you do for the Ungvari and Rosebush data (pg 16).

The discussion of the theory of the mechanisms in NMS, catatonia, and malignant hyperthermia deserves a clearer message (pg 23). When NMS was discerned as an entity in the late 1970s, it was seen as similar to malignant hyperthermia, leading to the suggestion that dantrolene be tried. But no commonality between MH and NMS has been demonstrated except the superficial appearance of the syndromes. Further, the evidence for the efficacy of dantrolene alone in NMS is anecdotal at best. (I am not convinced that it does anything material in CNS disorders.) It does no good to continue to recommend its use in NMS.

In the theoretic argument (pg 24), you argue for a specific site for pathophysiology. Perhaps you would include a statement that one should assess PET with xxxx as the ligand or SPECT for lesions in yyyy and/or zzzz. Such specificity may bring you more attention than the more general, non-specific loci recommended now. [I believe Mickey Taylor did himself a disservice by hsi trepidation in not seeking to argue for more specificity; as a result his work is usually seen as 'something happens to the brain, more in the front than the back, I think . . .]

Finally, the summary argument -- which is usually the most read- should be clearer about what you recommend as a course of treatment. The journal is 'critical care medicine', read, I assume by clinicians. What I have learned since we treated our patient on 16N together is that that all neuroleptics need to be discontinued promptly; that supportive measures instituted; and that lorazepam needs to be 'pushed'. My present dosage range for lorazepam is up to 16mg/day. I see no need to ever consider bromocriptine or dantrolene -- if lorazepam fails, ECT is the definitive treatment. If the diagnosis of catatonia is secure, such Rx will surely optimize the best for the patient.

I have also pencilled in corrections for some of the citations.

On a more personal level, I do not need to be a co-author of another paper. I am delighted to help in this effort -- and will gladly review a penultimate draft -- and all I warrant is a note of thanks in the acknowledgements.

Many thanks for the opportunity to read this review. My best regards.

Sincerely yours,

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Professor of Psychiatry
and Neurology