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CORPUS CALLOSUM DYSFUNCTION IN SCHIZOPHRENIA

DEAR SIR,

May we account for the difference between our original findings, and those of Professor Shagass and his colleagues? As we reported some time ago (*Journal,* November 1982, **141,** 535), our stimulus was not just simple vibration as used by Shagass *et al,* but included a larger amplitude sudden finger extension; and we reported that this latter was the critical component for producing the evoked potentials. We accept, therefore, that our work was not directly comparable to that of Salamy (1978).

The relevance of stimulus parameters is shown by the results, remarkably similar to ours, reported by Gulmann *et al* (1982), who used a standard electrical stimulus to the median nerve, and who did not use a common vertex reference. Their main finding was of a substantially reduced conduction time, though this only achieved significance in one direction. Their median conduction time from left to right of 16 m/secs in schizophrenics could not possibly have been produced by a normal pathway as it would have required conduction velocities of more than 90 metres/second and far thicker myelinated fibres than have been described in the human corpus callosum (Swadlow *et al,* 1979), which supports our hypothesis of an abnormal ipsilateral pathway in schizophrenia. Connolly (*Journal,* April 1982 , **140,** 429–30; May 1983, **142,** 536) has failed repeatedly to understand the concept of a "negative" transmission time, though this convention was also used by Gulmann and his colleagues to denote an ipsilateral response occurring earlier than the corresponding contralateral one.

The crucial work on the corpus callosum was performed in a patient with an angiographically proven tumour in this area (Goya, 1976). When either median nerve was stimulated electrically, only a contralateral response could be recorded, the delayed ipsilateral response expected in normals being abolished completely. Not only does this demonstrate that the ipsilateral response in normals is not just an artefact produced by an active reference electrode, but that it is dependent on the integrity of the corpus callosum, thus validating the contralateral-ipsilateral latency difference as a measure of conduction time. It also gives the lie to Professor Shagass' confident assertion that such a conducting pathway does not exist, a view derived apparently by over-generalisation from the anatomy of monkeys.

Shagass has re-iterated Connolly's clanger (*Journal,* April 1982, **140,** 429–30) in believing that patients with agenesis of the corpus callosum have split-brain symptoms and signs, which they most certainly do not (Sperry, 1968). Furthermore, neuropathological work published subsequent to, and perhaps partially stimulated by our predictions, has shown gliosis of the corpus callosum in late onset schizophrenia (Nasrallah, *Journal,* July 1982, **141,** 99–100), and thickening in early onset disease (Bigelow *et al, Journal,* March 1982, **142,** 284–7).

Further research seems indicated in this controversial field. One of us has been using visual evoked potentials as an independent measure of corpus callosum function, and these results will be submitted shortly.

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