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Matters Arising: Bhatnagar and Andy Reply

Subhash Bhatnagar
Marquette University, subhash.bhatnagar@marquette.edu

Orlando J. Andy
University of Mississippi Medical Center

stutter" for the past two and a half years while using self-delivered thalamic stimulation.

The history of stuttering treatment is a record of dubious claims for therapy benefits with documentation that is little better than that provided by Bhatnagar and Andy.[2] For instance, these authors provide absolutely no details on where or how the patient's speech data were collected, the reliability of those data, or more importantly, the quality of the subject's speech. Among the best known features of stuttering are its reactivity and the ease with which it is alleviated when the speaker employs an unusual manner of speech production. This has also been found to be true of acquired stuttering.[3] Because these variables may confound treatment effects, it is essential that they be carefully controlled in any investigation of a stuttering treatment.[4] The authors also claimed that their patient's "improved" speech was sustained for two and a half years with some continuing and unspecified level of self stimulation.

Quite apart from the need to document the functional value of the treatment during this period, it is impossible to assess the merit of the authors' report without carefully collected speech performance data.[5] It is to be hoped that the authors will provide much more evidence to justify such a monumental claim for treatment efficacy.

ROGER J INGHAM
Department of Speech and Hearing Sciences, University of California, Santa Barbara, California 93106, United States

1 Bhatnagar SC, Andy OJ. Alleviation of acquired stuttering with human centremedian thalamic stimulation. *J Neurol Neurosurg Psychiatry* 1989;52:1182–4.
2 Ingham RJ. *Stuttering and behavior therapy*. San Diego: College Hill, 1984.
3 Rosenbek J, Messert B, Collins M, Wertz RT. Stuttering following brain damage. *Brain and Language* 1976;6:92–6.
4 Bloodstein O. *A handbook on stuttering* (4th Ed) Chicago: National Easter Seal Society, 1987.
5 Ingham RJ, Costello JM. Stuttering treatment outcome evaluation. In: JM Costello, A Holland, eds. *Handbook of speech and language disorders*. San Diego: College-Hill, 1986: 313–46.

Bhatnagar and Andy reply:
Dr Ingham questions the validity of the reported observations of the facilitatory effect of thalamic stimulation on acquired stuttering on the grounds of unreliability of data, lack of patient follow up and questionable remission of stuttering. His questions are based on a misunderstanding of the reported data and the following comments are made to clarify his inquiries.

Data collection: Contrary to Dr Ingham's perception, this patient's medical condition and communicative skills were carefully monitored for weeks before the surgery and his behaviour was objectively assessed. Comprehensive neurolinguistic assessment, completed as part of a larger research project, included auditory comprehension, lexical retrieval, verbal and nonverbal memory, lexical association, expressive language, nonverbal reasoning and motor speech. Preoperatively his stuttering was severe and did not fluctuate, consequently a ten minute segment of his spontaneous verbal output along with performance on a structured reading task was used as a representative sample of his dysfluency before and after surgery. Postoperatively, the patient was consistently observed during the hospital stay and has been followed as an outpatient for the past five years.

Neurolinguistic testing has been repeated to evaluate the effect of left thalamic stimulation on language and cognition. Thalamic stimulation eliminated his motor speech spasms; with his verbal output being spontaneous, natural and free of dysfluency, there is no need for further evaluation. The pre- and postoperative language/speech evaluations were completed by one of us, a certified speech language pathologist. The total amount of time spent jointly by the authors observing this patient would amount to more than 40 hours, not to 10 minutes as interpreted by Dr Ingham.

The objectivity of the facilitational effect. Ingham's equation of this physiologically evident, scientifically demonstrated and objectively measured ameliorating effect of thalamic stimulation on speech dysfluency, with "dubious claims for therapy benefits", is inaccurate. It should be noted that in all those "dubious claims" of stuttering treatment, patients knew in advance that the goal of the treatment was the elimination of stuttering and therefore they had preconceived expectations. Further, the benefit entailed by the devices had faded after subjects became accustomed to them. The patient in question was stereotactically treated for intractable pain and absence attacks. Neither was the treatment geared to treat speech dysfluency nor was there any expectation on the part of the patient; not even the authors had reasons to believe of such a possible effect. The observed amelioration of speech dysfluency was a secondary benefit of the mesothalamic stimulation undertaken for pain control, and no relapse of speech dysfluency had occurred with continuous usage of the thalamic stimulation.

The patient does not speak with any "unusual manner of speech production", as argued by Dr Ingham. The patient's speech is spontaneous, natural and requires no groping efforts and constant monitoring of speech as before surgery. Furthermore, the stimulation had a positive effect on language functions, memory, attentiveness and self-concept.[1] The patient has been free of dysfluency for the five years of self-stimulation with no relapse of either pain or stuttering. If the speech gain has lasted for this length of time, it is highly unlikely that this elimination of stuttering has resulted from an unusual motor speech pattern or gimmick (placebo effect). Further, we have observed similar facilitating effects of the thalamic stimulation on acquired stuttering in some other neurosurgical patients (in preparation).[2]

Hypothesis formation: The authors made no claim that this amelioration effect was a psychological or organic phenomenon nor did we imply a prescription for acquired speech dysfluency. We only reported observations that the thalamic stimulation had suppressed the pre-ictally present abnormal mesothalamic discharges and subsequently had controlled the pain; this also had secondarily resulted in the elimination of acquired stuttering. Since the pre-nuclear reticular network[3] (PNRN) is located here, it is likely that the mesothalamic modulation of the PNRN had a role in the elimination of speech dysfluency (motor speech spasms). Support for this assumption has come from the additional observations of amelioration of acquired stuttering in other patients secondary to similar mesothalamic mechanism.

SUBHASH C BHATNAGAR
ORLANDO J ANDY
Department of Speech Pathology and Audiology, Marquette University Milwaukee, Wisconsin 53217
Department of Neurosurgery, University of Mississippi Medical Center Jackson, Mississippi 38216, United States

1 Bhatnagar SC, Andy OJ, Korabic EA, *et al.* The effect of thalamic stimulation in processing of verbal stimuli in dichotic listening tasks: A case study. *Brain Lang* 1988;36:236–51.
2 Bhatnagar SC, Andy OJ. Stuttering acquired from subcortical pathologies and its alleviation from thalamic perturbation; evidence from four neurosurgical subjects. (*In preparation*).
3 Lorente De. Analysis of the activity of the chains of internuncial neurons. *J Neurophysiol* 1938; 1:210–44.

The observations of Bhatnagar and Andy[1] concerning central influences upon stuttering raise some important questions. The authors provide no anatomical evidence yet confidently identify a proposed structure in the brain as the locus of the effect that they have observed. The general concept of altering such a very difficult problem by brain stimulation is of great interest though a note of caution must be sounded in the absence of histological confirmation.

It would have been useful to have known if there were any changes in cardiovascular parameters, such as heart rate or blood pressure, with stimulation since in the rat electrical stimulation of the centromedian-parafasicular complex causes a marked tachycardia and a large pressor response.[2] Recently it has also been shown that stimulation in this region results in dissociated changes in cerebral blood flow and cerebral metabolism.[3] The careless use of the terms cerebral blood flow and cerebral metabolism by the authors[1] suggests that they are interchangeable; they are not. The iodoamphetamine method measures cerebral blood flow not cerebral metabolism, the two should not be confused. It is particularly ill-advised in this setting where strong experimental evidence has demonstrated that they do not change in parallel.

PETER J GOADSBY
The National Hospitals for Nervous Diseases, London W9 1TL, United Kingdom

1 Bhatnagar SC, Andy OJ. Alleviation of acquired stuttering with human centromedian thalamic stimulation. *J Neurol Neurosurg Psychiatry* 1989;52:1182–4.
2 Mraovitch S, Lasbennes F, Calando Y, Seylaz J. Cerebrovascular changes elicited by electrical stimulation of the centromedian-parafasicular complex in the rat. *Brain Res* 1986;380:42–53.
3 Mraovitch S, Seylaz J. Metabolism-independent cerebral vasodilation elicited by electrical stimulation of the centromedian-parafasicular complex in the rat. *Neurosci Letts* 1987; 83:269–74.

Bhatnagar and Andy reply:
This response clarifies the anatomical mechanism relating to brain function and other questions posed by Dr Goadsby.

Stereotactic method: The single human brain used by Schaltenbrand and Bailey[1] was used as the reference for the stereotactic coordinates to identify the electrode localisation site in our patient. Thalamic studies reveal increased anatomical variability with

increasing distance from the mid line.[2] The electrode tip in this case is at coordinates (F 7·7, H 0·0, L 6·8 mm). The bare component of the unipolar electrode extends 3 mm rostral to the tip at a 45 degree angle. In the sagittal plane the variability is approximately 2 mm. In this case therefore the possible structures which may be involved are the centromedian nucleus and parafascicular nucleus of the thalamus, the prerubral field of the mid brain, the medial part of the VPM nucleus of the thalamus, the habenulo-interpeduncular tract, the field H of Forel and the rostral components of the brainstem reticular formation.

Electropathological discharges and localisation: The EEG discharges observed in this case and used for final electrode placement were characteristic of discharges observed in other patients being similarly treated for chronic pain syndromes. The discharges have included an area in the rostral mesencephalon in contiguity with the posteromedial thalamus in which the centromedian nucleus was most frequently implicated. Since the discharges are generated by cellular elements and not by fibre tracts, and since attenuation of the discharges is accompanied by improvement without sensory and motor impairments, it was theorised that the observed effects were due to implication of neither the fibre tracts nor thalamic sensory-motor nuclei.

Clinical reports without histological control: It is unfortunate that precise anatomical localisation of an electrode cannot be given in the present case, nor in any other clinical reports which by necessity are dependant upon a stereotactic atlas of the human brain.[1] The most that one can expect in a brief case report, is to diagrammatically illustrate the anatomic site of localisation and to identify the site by the structure or anatomic system whch most likely accounts for the results. In our report, it is expected that the readers will refer to the diagrammatic insert and realise, as did Mr Goadsby, that in all probability the centromedian nucleus is not involved to the exclusion of other structures.

The same reporting methodology was apparently used, in the rat studies quoted by Mr Goadsby, on cerebrovascular changes elicited by electrical stimulation of the centromedian-parafascicular complex in the rat. A non critical reader may think that the (C-M) complex is exclusively responsible for the findings. Although the electrodes are in the (C-M) Complex, the tractus retroflexus of Meynert may also be implicated for the following reason: histological diagrams of the electrode localisations reveal several points next to the tractus retroflexus of Meynert, which runs through the (C-P) complex. That tract, in part, conducts impulses from the anterior diencephalon and septal area, structures which are associated with BP elevations in response to high frequency discharges. Furthermore, after establishing threshold stimulation sites for BP elevations of $< = 10$ mm Hg and then stimulating at the same sites with parameters $3\times$ threshold, makes one wonder whether those fibre tracts were also implicated at those relatively high levels of stimulation. In addition, 1·5 h after anaesthesia may be insufficient clearance time to obviate chloralose hyperexcitability effects.

Blood flow: The increased cerebral blood flow from stimulation of the centromedian-parafascicular complex observed, by Mraovitch and Seylaz (1987) and Mraovitch *et al* (1986) in the rat, is of interest in view of our demonstrating increased thalamic blood flow without obvious cortical involvement. One is tempted to speculate that the underlying mechanism is the same in both instances despite the deficiencies in specific anatomical localisation. The evaluation of local blood volume or local glucose utilisation depends on the radiolabelled agent used in SPECT. Iodoamphetamine (IMP) used in this case primarily evaluated rCBF, but probably also indirectly reflected local cell functions or metabolic state.[3]

Blood pressure: Acute changes in BP, as reported in the rat, were not noted in this patient nor any other during stimulation in the general area of the centromedian nucleus, even at threshold stimulation for sensory-motor responses. Spontaneously and artificially induced after-discharges also were not accompanied by BP changes.

1 Schaltenbrand G, Baily P. *Introduction to stereotactic atlas of the human brain.* Thieme: Stuttgart, 1959.
2 Andy OJ, Jurko MF, Sias FR. Subthalamotomy in treatment of Parkinsonian tremor. *J Neurosurg* 1963;**20**:860–70.
3 Lee RL, Hill TC, Hollman BL, Clouse ME. An isopropyl (I–123)p-Iodoamphetamine (IMP) Brain Scans with SPECT: Disordance with Transmission Computed Tomography. *Radiology* 1982;**145**:795–9.

BOOK REVIEWS

The Right Cerebral Hemisphere and Psychiatric Disorders. By JOHN CUTTING. (Pp 479; £45.00.) Oxford University Press. 1990. ISBN 0-19-261764-8

The fascination with the functional differences of the two cerebral hemispheres lives on. We have come a long way from the quasi-phrenological theories that attempted to localise discrete functions to specific areas in the right or left hemisphere. More appealing views of the left hemisphere as an analyser and the right as a Gestalt processor have also been superseded by models based on the study of split brain patients and the use of dichotic listening and tachistoscopic techniques to direct information to either hemisphere. One such model put forward by Kosslyn considers that the left hemisphere analyses information along categorical lines, like a library of words or objects, whilst the right hemisphere is more like a guide-book that allows us to get information from the library.

Cutting has found inspiration in Kosslyn's views and his perambulations across the two hemispheres are done with his guide-book firmly in hand. The result of his effort is a unique book that provides a detailed and scholarly review of hemisphere function that will be impossible to find anywhere else. The book is divided into three sections. The first deals with the evidence of the differential functions of the two hemispheres and includes an excellent historical review. The second deals with focal neuropsychiatric symptoms in the light of differential hemisphere function and includes a useful chapter on tests of hemisphere function, and the third explores the role of hemisphere differences in the causation of psychiatric disorders.

In my view the main strength of the book lies in its second section which skilfully explores the common ground between many psychiatric and neurological phenomena which, at times, have been artificially separated. Various disorders of awareness, language and thought and other symptoms such as delusions are dealt with here. New insights into phenomenology are abundant and I can easily envisage coming back to it in search for an explanation, when puzzled by clinical cases. The last section is perhaps best seen as food for thought and it is less likely to stand the test of time.

Cutting firmly believes that a hemisphere imbalance, with impaired functioning of the right hemisphere, is at the root of schizophrenia. The evidence for this, as Cutting himself points out, is far from conclusive and the recent imaging and neuropathological studies have failed to provide the desired proof. In fact, finding a coherent explanation to encompass the evidence implicating abnormalities in various cerebral sites in schizophrenia is one of the greatest challenges facing psychiatry; and hemisphere imbalance is unlikely to be a satisfactory explanation. The evidence is even less convincing for affective illness and autism. These problems do not detract from the interest of the book, but add to the hope that Cutting will again be tempted to write on the subject when, in a few years time, the biology of psychiatric illness will be better understood. All those interested in the complex relations between brain and mind should read this book.

MARIA A RON

Clinical Neurophysiology of the Vestibular System. 2nd Edition. Contemporary Neurology Series. By R W BALCH AND V HONRUBIA. (Pp 301; Price £38.79). Philadelphia, F. A. Davis Co., 1990. ISBN 0-8036-0584-6.

The first major investigations of clinical disorders of the vestibular system were carried out by Robert Barany in 1907. Since this time, and especially in the last 15 years a veritable deluge of tests has been applied to function and reflexes arising in the vestibular apparatus. Neuro-otology has evolved as a new specialty, but sadly the refinements of clinical diagnosis and treatment of the dizzy patient have lagged behind. Indeed only a handful of diagnoses are used by most audiologists and neurologists. The commonest are: acute vestibular neuronitis, benign positional vertigo, the clinically ill-defined and much overdiagnosed Menière's syndrome, and a variety of clinical assumptions in later life yielding labels such as cervical spondylosis and vertebrobasilar insufficiency often in tendentious fashion.

There is little doubt that the dizzy patient will at least receive a more accurate diagnosis if suitably investigated in a clinically directed neuro-otology laboratory, even if he or she emerges with specific drug or surgical therapies which are often disappointing.

The second edition of this book from UCLA appears some eleven years after the first. It is conventionally divided into three