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.

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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.

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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.

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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