Figure 1 a) Anteroposterior (AP) and b) lateral skull radiographs showing crossbow bolt at presentation.

ventricle (fig 1b), with evidence of intraventricular haemorrhage but no evidence of a cerebral abscess. Despite initial treatment with flucloxacillin and sulphadimidine the pyrexia continued and signs of meningism developed. Penicillin and chloramphenicol were added, after which he showed a progressive recovery over a five day period.

Lumbar punctures performed during this period showed an increasing polymorph count to a maximum of 1400/ml and continued to show a haemolysed supernatant with no visible organism on microscopy. Culture of the initial lumbar puncture CSF

grew *bacillus subtilis* after one week but was considered to be of doubtful significance as a primary pathogen. It was, however, subsequently cultured from two further specimens of CSF. It was sensitive to chloramphenicol and penicillin but resistant to sulphadimidine. The organism also possessed a haemolysin which would account for the persistently haemolysed supernatant. Treatment with chloramphenicol was continued for three weeks and penicillin continued for six weeks, at which time lumbar puncture was normal. The patient made a complete recovery with no neurological deficit and was discharged for psychiatric treatment.

The "non-pathogenic" members of the genus *bacillus* are spore-bearing, aerobic and usually gram positive saprophytic organisms which are ubiquitous in distribution.¹ They have been described as a rare cause of both local and disseminated infections.² Some species are used as test organisms for assessing the efficiency of ethylene oxide and ionising radiation sterilisation techniques. In particular *B. stearothermophilus* spores which are heat resistant to 121°C, are used to test autoclave function.¹ They are commonly isolated from clinical material and classified as "contaminants". As a result of a complex taxonomic relationship such non-pathogenic isolates are reported as *bacillus subtilis*, and a specific identification is not usually performed.² The structure most commonly involved is the eye, although epidemics of food poisoning have been ascribed to it.³

The most serious orbital infection, a fulminating panophthalmitis, appears to follow penetrating trauma with direct inoculation of the organism into the vitreous humour.⁴ Disseminated infection as a result of primary infection with *bacillus subtilis* is rare, but presents as meningoencephalitis, meningitis, endocarditis and septicaemia in the twelve recorded cases.² Nine of these had involvement of the central nervous system which in four cases followed a spinal anaesthetic procedure. It would appear that the organism is introduced during such procedures directly into the subarachnoid space, where the defence mechanisms of the body are minimal. Interestingly, the organism does not appear to cause opportunistic infections in debilitated patients and therefore supports the theory that direct inoculation into a poorly defended area allows a weakly virulent organism to become pathogenic.²

In our case the organism appears to have gained entry from the wooden bolt by virtue of the penetrating nature of the injury. Although of low virulence, disseminated infections appear to have a significant mortality of 50% in the 12 cases previously recorded,² and our patient was therefore fortunate to have shown a complete recovery.

Although a single dose of a broad spectrum antibiotic was given perioperatively this was obviously not sufficient to prevent infection and in penetrating injuries of this type it is necessary to complete a full course of high dose prophylactic broad spectrum antibiotics to minimise the risk of colonisation. Of further note in this case is the risk of major arterial damage. The crossbow bolt penetrated the brain anterior to the middle cerebral artery and crossed the midline structures. Although not performed in this patient, cerebral arteriography is advisable in any patient before removing a foreign body which has penetrated the skull.

This case illustrates the need to consider

bacillus subtilis as a primary and potentially serious pathogen following penetrating head injuries and this organism should therefore not be dismissed merely as a laboratory contaminant.

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We thank Mr J Bartlett, Consultant Neurosurgeon, Southeast Regional Neurosurgical Unit, Brook Hospital, London for permission to report his patient, and the Department of Clinical Photography, Royal National Throat Nose and Ear Hospital, London for their preparation of the illustration.

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MATTERS ARISING

Alleviation of acquired stuttering with human centremedian thalamic stimulation

I would like to comment on the article by Bhatnagar and Andy¹ which claimed that thalamic stimulation was effective in the treatment of an acquired stuttering. Their findings could have very substantial theoretical and clinical implications, but they rely on seemingly insubstantial and questionable data.

The authors report on a 61 year old male with a 20 year history of trigeminal pain, beginning with a tic douloureux, who ultimately developed a severe stuttering. The stuttering had a presumed subcortical origin and progressively deteriorated. The patient's self-administered thalamic stimulation, via an implanted electrode, was claimed to have had "a remarkable beneficial effect on the speech dysfluencies." The basis of this claim was a comparison between stutterings counted during preoperative conversation and oral reading tasks, and stutterings counted during a 10 minute postoperative conversation. This information was supplemented by self ratings of "dysfluencies" before and after the implant. It was also claimed that the subject had been "free of his

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 centromedian thalamic stimulation. *J Neurol Neurosurg Psychiatry* 1989;52:1182-4.
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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. Pre-operatively 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. Post-operatively, 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 network³ (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.
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The observations of Bhatnagar and Andy¹ 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-parafascicular 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 authors¹ 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.
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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 Bailey¹ 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