

Arrest of Attacks of Cluster Headache by Local Steroid Injection of the Occipital Nerve

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Introduction

The role of the cervical spine and its associated structures has been generally underestimated in the causation of headache. And yet it has long been accepted that cervical spondylosis can cause pain referred to the orbit and frontal regions. Experimental stimulation of various structures of the neck in man can produce pain in the anterior parts of the head. Orbital and frontal pain was produced by stimulation of the C₁ dorsal rootlets [6], forehead pain was elicited by stimulation either of neck muscles close to their insertion to the occiput [3] or midline soft tissues between the occiput and the C₁ vertebra [4]. These clinical observations have been explained on the basis of overlap of central connections between the spinal nucleus of the trigeminal nerve and the upper cervical nerves within the spinal cord. On descending through the pons and the medulla, the spinal trigeminal nucleus becomes both morphologically and functionally associated with the upper cervical segments and its cells form a column continuous with the column of cells forming the posterior horns as far down as C₂ [2] and perhaps as far down as C₄ [8]. In fact, this cervicotrigeminal relay is accepted as the main pain centre for the head, and pain impulses from the neck may thus be referred to the frontotemporal region and vice versa [7].

The pain of cluster headache is experienced in and around the orbit and the frontotemporal area, a distribution remarkably similar to pain arising from stimulation of upper cervical structures. As the main neural structure in that region which conveys sensory impulses to the brain is the greater occipital nerve, it was decided to test the hypothesis whether interruption of that nerve would relieve the recurrent attacks of cluster headache.

Patients and Methods

20 patients suffering from proven cluster headache were selected for study. 8 suffered from the chronic and 12 from the episodic variety of the disorder. Only 3 patients were suffering from the condition for 1 year, and of these 2 belonged to the chronic variety. The remaining patients were suffering from attacks of cluster headache for 4–24 years. No abnormality of the nervous system was detected in any of the patients and no sensory changes were present in the distribution of the occipital nerves.

Injection of the occipital nerve ipsilateral to the side of the headache was made when patients were headache-free. The point of injection was at the accepted point of emergence of the nerve onto the scalp. This is the intersection of the midpoint between the superior nuchal line and the intermastoid line in the horizontal plane, and the midpoint between the mastoid tip on that side and the midline of the neck, in the vertical plane [1]. In the majority of cases there was tenderness on pressure at this point but, where this was not present, a point lateral or medial to it was sought, where such tenderness was present before the injection was given.

The site of injection was prepared by instilling 2–5 ml 2% lignocaine local anaesthetic (LA) into the skin and deeper tissues. This was followed in about 3–5 min by 120 mg (3 ml) methylprednisolone acetate in polyethylene glycol (Depomedrol, Upjohn). The needle was directed 5–10° towards the occiput until resistance of bone was felt, thus making certain that the spinal subarachnoid space was not entered into. The tip of the needle was withdrawn a few millimetres and 1 ml of the solution was deposited. The tip of the needle was then withdrawn to just under the skin and redirected about 10° laterally and again medially, and into each site 1 ml of Depomedrol was deposited. On completion of the injection, the area was massaged vigorously, so as to spread the solution of the steroid and make sure that at least part of it bathed the nerve trunk. Light pressure was maintained over the point of the injection with a wad of cotton wool for a few minutes, until the bleeding from the needle tract stopped and the patient was allowed free.

Patients served as their own controls. 10 patients were given an injection of 5 ml lignocaine during an attack of headache. In 4 cases the headache was made worse, and in the remainder the attack was arrested within a few minutes. 5 patients were given an injection of 5 ml lignocaine between attacks of headache, whilst another 5 were given an intramuscular injection of 120 mg Depomedrol. In all cases attacks of headache returned within 24 h.

Results

Details of responses of patients to the treatment used are shown in tables I and II.

Episodic Cluster Headache. In 7 patients, the duration of relief from headaches varied from 35 to 129 days without recurrence, by which time the bout was considered by the patients to have ended by natural remission. That this was so was confirmed by a survey of the same patients 1 month after the above periods, when they reported that in fact they continued to be free of attacks. 5 patients experienced relief from headaches for 14–37 days before the attacks returned and further relief of 10–29 days was obtained in 4 patients when the injection was repeated (table I).

Table I. Periodic cluster headache. Clinical details of patients and response to steroid injection of occipital nerve

Patient No.	Age years	Duration of disease years	HA side	HA frequency per day	HA duration hours	Response HAF days
1	33	10	R	1	0.5-2	129
2	33	8	R	1	1.5-2	32, 20
3	41	21	R	2-3	0.25-4	0, 35
4	39	5	R	0-1	2-6	90
5	55	4	R	1	0.75-4	90
6	26	12	L	2	1.5-2.5	37, 0
7	31	10	R	1-4	1-1.5	41
8	52	1	L	1-4	0.25-0.5	29, 16
9	46	24	L	1	1-1.5	0, 33
10	43	23	R	2	0.25-2	14, 29
11	58	16	R	3-4	0.5-1	120
12	31	7	L	1-2	1	21, 10

HA = Headache; HAF = headache-freedom.

Table II. Chronic cluster headache. Clinical details of patients and response to steroid injection of occipital nerve

Patient No.	Age years	Duration of disease years	HA side	HA frequency per day	HA duration hours	Response HAF days
1	64	8	L	1-3	1-3	5, 10
2	30	8	L	1	0.5-1.5	20, 56, 38
3	30	10	R	1-3	0.5-0.75	36, 28
4	32	10	L	1-2	1-1	15, 21
5	55	11	L	2	0.5-1	31, 15, 0
6	49	1	R	1	0.5	30, 73
7 F	29	1	R	1-4	3-4	5, 10
8	58	6	L	2	0.75-1	40, 33

HA = Headache; HAF = headache-freedom.

Attention is drawn to the fact that of the 12 patients in this category of cluster headache, 2 obtained no relief of their headaches following the first injection of corticosteroid (patients 3 and 9), but a second injection produced relief lasting 35 and 33 days, respectively. It can only be assumed that the first injection did not deposit the liquid close enough to the nerve and this is not unexpected, in view of the sinuous and inconstant course of the nerve [1].

Chronic Cluster Headache. All patients had at least two injections. The period of relief from headaches varied from 5 to 73 days, but headaches returned in every patient including the one (patient 2) who has had three injections so far (table II). Only 1 patient failed to respond to an injection and this occurred on the third occasion (patient 5).

The only side-effect complained of by 4 patients was a dull, aching occipital headache of varying intensity, lasting 6–20 h, after injection, but requiring no analgesics for its relief. Mild and subjective depression of pain and temperature sensation over the occipital area of the skull was noted by 9 of the 20 patients within 10 min following injection of the LA and corticosteroid, and this persisted by the end of the first week in 7 patients. It is to be noted that 18 of the 20 patients in the study experienced arrest of their headaches following the initial injection of Depomedrol.

2 patients suffering from the chronic variety of the disorder (patients 1 and 4, table II) were subjected to surgical division of the occipital nerve on the symptomatic side. The first patient was headache-free for 6 months and his headache returned with increasing frequency over the subsequent 4 months, whilst the second patient was symptom-free for only 10 days.

Discussion

The present study demonstrates that local corticosteroid injection into the region of the occipital nerve ipsilateral to the side of cluster headache is capable of arresting the bout of attacks for a period ranging from 5 to 73 days. Results in the group suffering from the periodic variety of the disease may not be so reliable, as bouts of headache can stop at any time by natural remission, making the decision as to the duration of the benefit of this particular treatment difficult. However, in 4 of the 12 patients the headaches returned and on repeating the injection beneficial effect was again obtained ranging from 10 to 29 days, proving that the treatment and not natural remission was responsible for the temporary arrest of the attacks. On the other hand, the fact that the headaches were arrested immediately following the injection only serves to confirm the effectiveness of the treatment in every patient. In the group suffering from the chronic variety of the disease, there appears little room for doubt as to the value of the procedure, since natural remission is exceptional in such cases, and in any case every patient had a second injection of corticosteroid into the region of the occipital nerve, as the headaches returned following an initial period of headache relief of variable duration.

That interruption of neural pathways produces relief of attacks of

cluster headache is not a new observation. *Harris* [5] reported successful relief of attacks in 5 cases by alcohol injection of the Gasserian ganglion. Other procedures used over the years have included avulsion of branches of the trigeminal nerve, radiofrequency lesions of the appropriate division of the nerve, retro-Gasserian root section and division of the greater superficial petrosal nerve. *Watson* et al. [9] reviewed the literature and reported their own experiences of 61 procedures in 20 patients with chronic cluster headache. They concluded that although a variable degree of relief was obtained by each of these procedures in the majority of patients, only exceptionally was such relief long-lasting.

The contribution of this study to the management of cluster headache lies in the fact that corticosteroid injections into the region of the occipital nerve ipsilateral to the headache will relieve the bout of attacks in every case, though temporarily, by blocking transmission of pain impulses along the nerve to the cervicotrigeminal pain centre. If attacks of cluster headache are assumed to be due to paroxysmal activity of the spinal nucleus of the trigeminal nerve as a result of impulses received from its many sensory connections, it appears that impulses arriving along the C₂ and C₃ sensory roots (the components of the occipital nerve), are the most significant and therefore their interruption leads to pain relief more regularly than interruption of other neural pathways employed so far.

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