"Cervicogenic headache": Clinical manifestation

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The main criteria of "cervicogenic headache" are considered to be as follows: relatively rare and long-lasting *unilateral* attacks of severe headache, although seemingly of a non-excruciating character, signs of neck involvement, and lack of "cluster pattern". In the present communication, the clinical manifestations in 11 patients fulfilling these criteria are described. All 11 patients selected in accordance with these criteria proved to be females, the age at onset ranging from 6 to 40 years (mean, 30 years). The mean duration of symptoms was 13 years. Six patients had had previous head/neck injuries. All patients had pain periorbitally, in the temporal region, and in the low occipital region (nape of the neck); less frequent were frontal, parietal, and facial pain and pain in the upper part of the occipital region. The duration of attacks was from 3 h to 3 weeks, and the interval between attacks lasted from 2 days to 2 months. The commonest accompanying phenomena were phonophobia, dizziness, ipsilateral eyelid edema, ipsilaterally blurred vision, and irritability. Some of the patients also had nausea ($n = 7$) and vomiting ($n = 6$). On physical examination, slight to moderate reduction of movements in the neck was noted, and five patients had ipsilaterally reduced sensation for touch in the trigeminal area. All the patients except one were severely afflicted. Attacks could, in addition to occurring spontaneously, be precipitated in all patients by head movements or by pressure at specific points in the neck. □ *Barré syndrome; cervicogenic headache, clinical manifestations; cluster headache, differential diagnosis; "migraine cervicale"; migraine, differential diagnosis*

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In 1925 Barré (1) introduced the term "syndrome cervical sympathique posterieur", linking headache causally with a presumed "posterior" sympathetic deficiency. Many reports on headache with a possible origin in the neck have appeared since then. The subject whether headaches may originate in the neck is, however, still controversial. Many migrainologists, although in principle accepting that neck abnormalities can cause headache from time to time and in various forms, have generally not been inclined to believe that any distinct, clinically recognizable type of headache should have its origin in the neck. Sjaastad et al. (2) in 1983 reported on 22 patients with a rather uniform headache from a clinical point of view. Each of the patients had some evidence that the neck was the origin of a headache proper, and we introduced the term "cervicogenic headache" for this type of headache.

It is the principal purpose of this paper to describe the *clinical* manifestation in a limited but carefully chosen group of patients with cervicogenic headache. We have carried out a rather thorough study in this group of patients. The results pertaining to other investigations in these patients will be presented in forthcoming articles.

Materials and methods

On the basis of what was considered to be the crucial traits in the first five cases that we observed with this clinical picture, we listed a set of inclusion criteria, set out in Table 1. Over the last approximately 4 years we have seen more than 60 patients with a tentative diagnosis of cervicogenic headache in our clinic. Only what we felt were typical cases in accordance with the inclusion criteria were included in the present study. Cases with an obvious psychic overlay were excluded. We finally chose 11 patients for this study; they were first seen by us in the period 1982–1983

Table 1. Inclusion criteria.

1. Unilateral headache ($n = 11$)
2. Headache usually of a non-continuous nature, with relatively rare* and long-lasting† attacks ($n = 11$)
3. No "cluster" pattern of attacks ($n = 11$)
4. Signs of neck involvement ($n = 11$). At least one of the following two possibilities:
   a) Ipsilateral, diffuse neck/shoulder/arm pain ($n = 10$) and/or
   b) Provocation of attacks by neck movements ($n = 10$)
5. A headache that during the typical attack, although occasionally being annoying to the point of making bed rest mandatory, does not seem to be of excruciating severity ($n = 11$)

The number of patients who fulfilled each of the requirements is given in parentheses.
* ≤One attack per day, and usually less than two attacks per week.
† The longest attacks should exceed or be equal to a duration of 1 day—that is, more long-lasting than the attacks in the cluster headache syndrome.

and have since been followed up prospectively. The chosen patients were interested in and willing to take part in this program.

All the patients have been interviewed and examined on numerous occasions by at least two of the authors independently (partly by all three). All the patients have also been observed during spontaneous attacks. The reason why we had to limit this study to 11 patients is the following: These patients were to go through an extensive investigation program, including cerebral angiography and cervical myelography, necessitating multiple visits to the hospital in the course of a long period. Such a program would be impossible for us to carry out in 50–60 patients. We feel, however, that the 11 carefully chosen patients are representative of the core of this material. Any selection other than on the basis of the mentioned criteria and characteristics was not made. The fact that, for example, the 11 patients proved to be females is accordingly a finding or result (see later) and not a premise.

All the patients except one (no. 11) were relatively severely afflicted, five of them having obtained a disability pension owing to their headache. The patients had invariably consulted many physicians and dentists previously. All patients had had their teeth examined, and in several of them extractions had been carried out on the assumption that the pain could have a dental origin. Several of them had previously also been *suspected* of being hysteric or neurotic.

Results

Sex and age

All the patients included were females. This is in conformity with the tendency in our first group, in which the ratio between females and males was 2.1. The age range was from 25 to 59 years, with a mean of 43 years. The age distribution at the beginning of the study is shown in Fig. 1.

Occurrence of additional headache and of familial headache

Three of the patients had had headache of a global character in attacks that subsided during adolescence. No further definite details are known about these attacks. One of the patients (no. 1, aged 38 years) had had attacks of classic migraine since the age of 15 years. The migraine attacks had subsided over the past decade.

In three cases there was no known family history of migraine or headache. In three cases one of the grandparents was suspected of having migraine ($n = 2$) or headache other than migraine but of some severity ($n = 1$). With regard to the five remaining cases, those representatives of these families who were thought to have the case history most resembling migraine were interviewed.

In one case (a son) the diagnosis seemed to be common migraine. In two cases headache other than migraine seemed to be present. In two cases the headache seemed to be

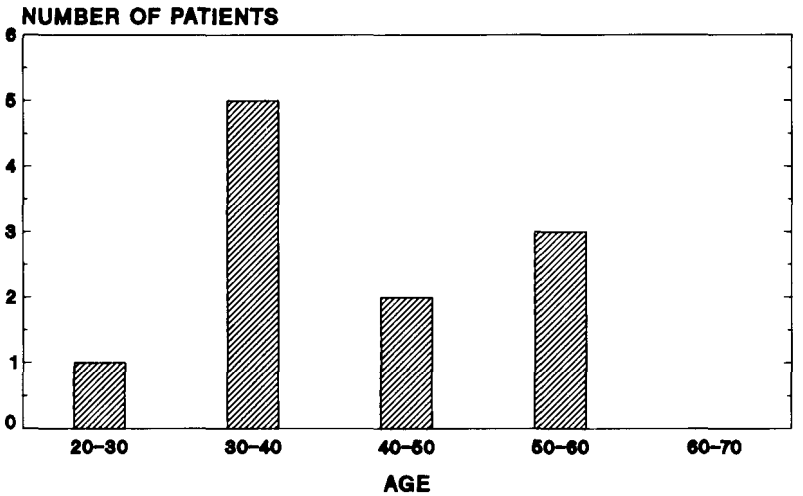

Fig. 1. Age distribution.

unclassifiable—possibly migraine, possibly tension headache.

In summary, therefore, in five families there did not seem to be migraine cases. In three families there was headache in *previous* generations, possibly migrainous in two cases. In two families there was headache of a questionable nature—possibly migrainous, possibly of tension headache nature; in one family there was probable migraine.

Start of symptoms

The age of onset of symptoms varied from 6 to 40 years, with an average of 30 years (Fig. 2). Headache duration ranged from 3 to 53 years, with a mean of 13 years (Fig. 3). It should be noted that in two of the patients headache dated back to childhood—that is, to the age of 9 and 6 years, respectively. The patients themselves (25 and 59 years old, respectively, when the study was initiated) are convinced that their headaches have continued unaltered since childhood. We report these statements with due scepticism, since the memory for headache is difficult to evaluate (3).

Six of the 11 patients could remember previous head/neck injuries. In four of these

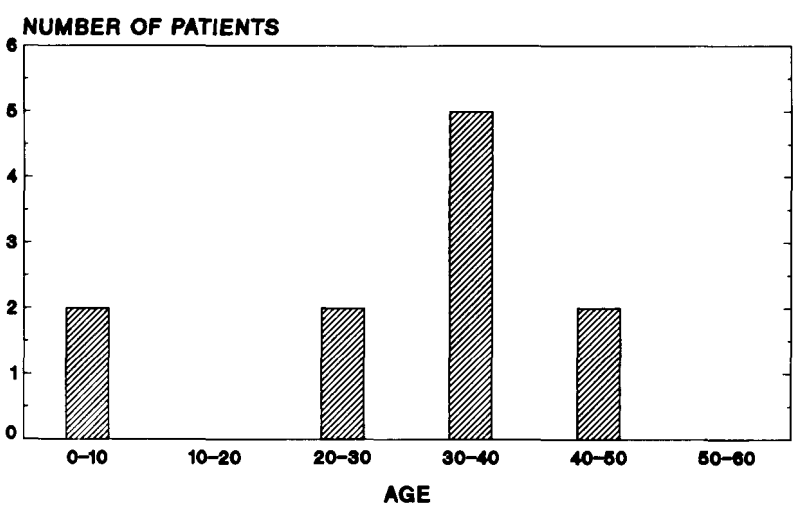

Fig. 2. Age at onset of symptoms.

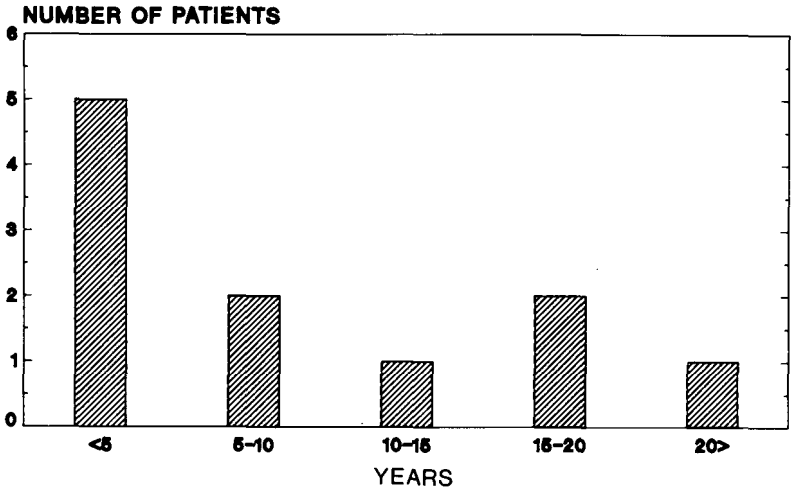

Fig. 3. Duration of pain at start of study.

patients there was a rather close temporal association between the injury and the onset of headache; that is, it appeared abruptly less than a month after the trauma (Table 2). In one of these patients, a typical whiplash trauma seemed to have taken place. In another the onset was temporally related to a minor gynecological procedure under general anesthesia. It may of course be argued that this was no *regular* neck trauma. The sequence of events may well have been a different one in this case. We have, nevertheless, classified this as possibly being a neck trauma on the basis of the patient's account of a painful, stiff neck postoperatively, even though nothing has been recorded in the hospital charts.

In five other patients (two with and three without previous head/neck trauma) there was also an abrupt onset with an initial, full-blown attack. One patient had a more insidious onset with a build-up over a 2-year period before full intensity of attacks was attained. One patient, having had her symptoms for about 26 years, was uncertain about the onset and whether it was acute or the symptoms had come on more insidiously.

Attack pattern

Attacks generally are relatively long-lasting, typically of 1–3 days' duration. The expression "relatively long-lasting" is used because cervicogenic headache is viewed as a unilateral headache, and a comparison is automatically being made with the other unilateral headaches—that is, cluster headache and CPH—in which the duration rarely

Table 2. Onset of symptoms.

In connection with	No. of patients
Known head/neck trauma*	6
Acute onset, in temporal relation to trauma	4
Acute onset, without temporal relation to trauma	2
No known trauma	5
Acute onset	3
Insidious onset	1
Uncertain information	1

* In one of the cases, in connection with a gynecological operation (see text).

Table 3. Temporal pattern.

Patient no.	Duration of attack		Duration pain-free interval	
	Shortest	Longest	Shortest	Longest
1	4 h	3 w	3 w	5 w
2	3 h	5 d	1 w	2 w
3	1 d	3 d	2 w	3 w
4	1 d	4 d	1 w	2 m
5	1 d	3 d	1 w	2 w
6	12 h	3 d	1 w	1 m
7	12 h	1 d	2 d	1 w
8	2 d	1 w	2 d	3 d
9	3 d	5 d	10 d	3 w
10	3 d	5 d	5 d	9 d
11	1 d	3 d	1 m	2 m

h = hour(s); d = day(s); w = week(s); m = month(s).

exceeds 3 h. Attacks may be as shortlasting as 3–4 h, whereas the most longlasting ones may be of 1 week's duration or more (Table 3). In most patients, there is a build-up to maximal severity in the course of hours to days. The pain-free interval is typically of 1–4 weeks' duration, the shortest interval being 2 days and the longest 2 months. In all but two patients there was absolute freedom from pain between the peaks of pain. Interval pain, when present, was reduced to less than 10% of the intensity during strong attacks.

In addition to the short-term variation, there seemed to be a long-term fluctuation of severity in eight patients. Phases of relatively intense pain attacks, relatively long-lasting attacks, and short periods of relatively or absolutely pain-free intervals alternated with phases of moderate attacks and long pain-free intervals. For six of these patients, the phases were of long duration, even years. Two patients seemed to experience seasonal variation with exacerbations during fall and winter. There was no typical night preponderance of attacks as in cluster headache. Nor was it typical that the attack started at any other time of the day, with the exception that not infrequently the patients woke up with a full-blown attack.

The pain

The pain was unilateral in all patients (inclusion criterion) and never changed side either during the solitary attack or between different attacks. By "unilaterality" is nevertheless not meant that the headache can never be felt on the other side: during the most severe attacks, the pain may spread slightly to the other side. It is thus always *most pronounced on the original side*; it is never felt on the opposite side alone. Four patients in our material had pain of this type—that is, slight pain on the opposite side during the strongest attacks, but the pain on the original side always dominated. Seven of our patients had pain on the right side, and four on the left.

The pain was usually characterized as "strong" to "very strong" by the patients. In our estimation the pain was usually hardly

Table 4. Pain distribution.

Patient no.	In/around eye	Facial area	Frontal area	Temporal area	Parietal area	Occipital area	Nape of the neck	Neck
1	+	+		+	+	+	+	+
2	+		+	+			+	+
3	+	+		+			+	+
4	+			+		+	+	+
5	+			+			+	
6	+		+	+			+	+
7	+			+			+	+
8	+		+	+	+		+	+
9	+	+	+	+	+	+	+	+
10	+		+	+			+	+
11	+	+		+			+	+

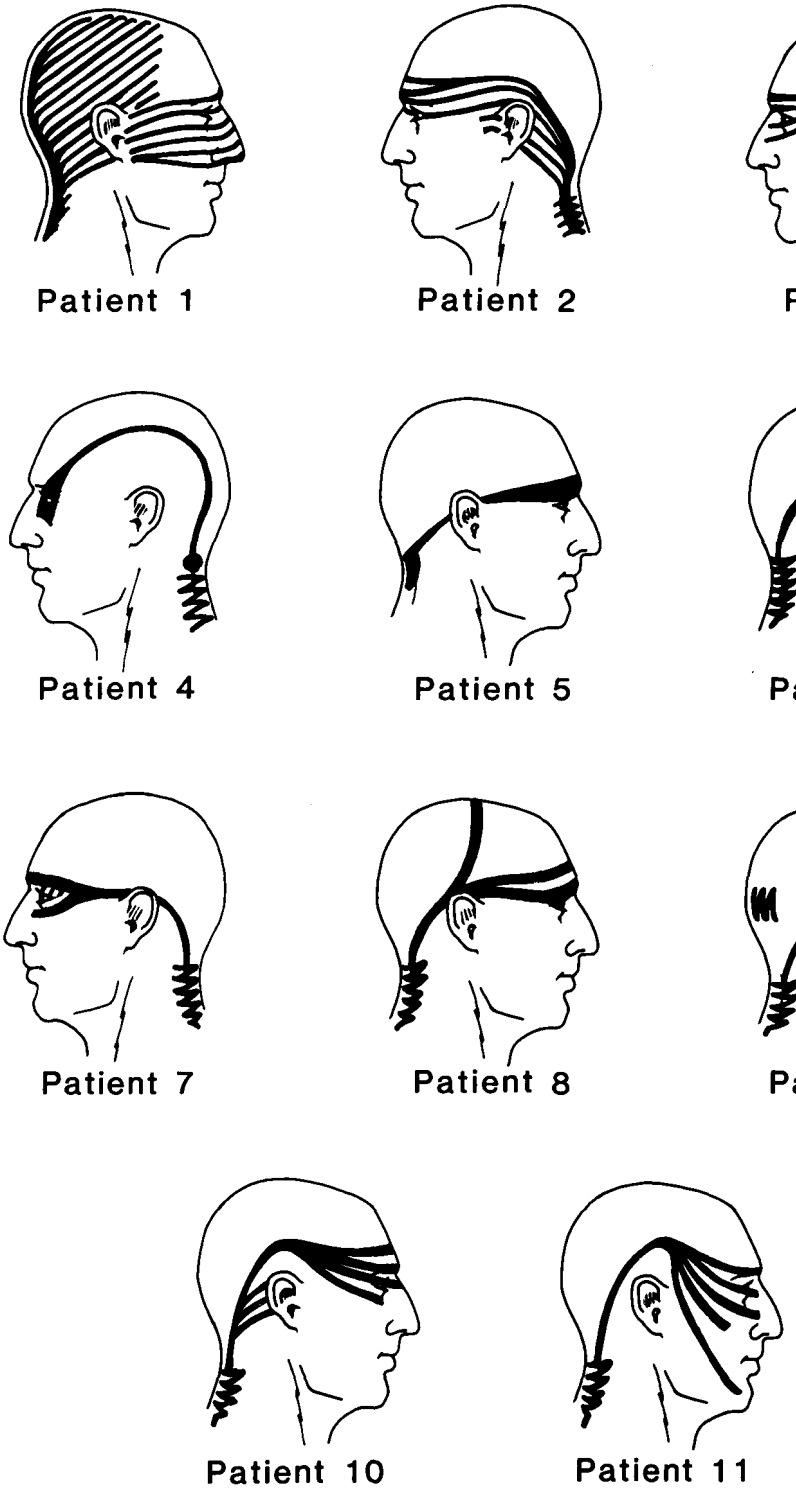

Fig. 4. Pain distribution as given by the patients.

more than moderate to severe (inclusion criterion). The pain was hardly excruciating, as in cluster headache and CPH. This assessment is based on criteria such as the patients' ability to go to sleep during attacks; the number of nightly awakenings; and the ability to be up and about during the usual attacks. Occasionally, the patients had to go to bed during the attacks. A general feeling of "sickness" seemed at times to be more in the foreground than the pain itself. Malaise and inertia were probably at times instrumental in setting the patients out of function to a higher extent than the pain *per se*.

The pain was very constant in distribution, when considering the single patient (Table 4 and Fig. 4). The pain could nevertheless vary in accordance with the *stage* of the headache attack, so that during one part of the attack it might dominate in the vertex area, in another part of the attack in the ocular area. All 11 patients had experienced the strongest pain in or around the ipsilateral ocular, aural, and temporal areas, and the pain also involved the neck and nape of the neck ("nape" is used in the sense of the transition between the back of the skull and the neck). In most patients, the pain was constantly boring and without a pulsatile quality. Four patients reported that the pain could attain a throbbing quality at some stage of the attack (in three patients minimal throbbing; in one a more marked throbbing in the neck at the beginning of attack).

On the whole, the patients experienced no or very moderate relief from the usual migraine medication, including ergotamine. The patient with the previous classic migraine attacks had benefited from ergotamine at the time, but not now. Another patient had also benefited from ergotamine previously, but not in recent years, whereas one patient felt that ergotamine occasionally gave her some relief. After some experimenting, they usually reverted to common analgesics like salicylates and paracetamol, but did not benefit much even from these. Some of the patients had tried indomethacin ($n = 4$; 75 mg/day for ≥ 1 week), piroxicam ($n = 7$; 20 mg/day for ≥ 1 week), and naproxen ($n = 2$; 750 mg/day for a week) in an open fashion. In two of the piroxicam-treated cases, the drug had a moderate effect; in the rest there was no obvious effect.

In only one patient (no. 5) was the pain restricted to the nape of the neck. In the 10 other patients it was also felt in the lower part of the neck. They also felt that the pain in the posterior part was linked with that in the anterior parts. Eight and six patients, respectively, had also noted some pain in the corresponding shoulder and arm. The sensation was mostly diffuse and vague. In none of the patients did the pain have a typical radiating character, so it can hardly be characterized as root pain limited to one or more roots. In two of these cases, the discomfort was more reminiscent of a numbness than of real pain. There was not necessarily a direct temporal association between these sensations and the headache; the shoulder/arm pain might be more or less constant. The headache in those with and those without shoulder or arm pain did not seem to differ appreciably.

Accompanying symptoms and signs

The accompanying symptoms of the attack are listed in Table 5. All patients experienced two or more of the listed phenomena. A general feeling of "irritability" prevailed in all but one patient (who was edgy and difficult to communicate with). Phonophobia was also a rather constant finding. More than

Table 5. Accompanying symptoms and signs.

	No. of cases
Nausea	7
Vomiting	6
Piloerection	7
Photophobia	5
Phonophobia	10
Reduced hearing, subjectively	2
Dizziness	9
Tinnitus	2
Irritability	10
Discomfort in the throat	6
Rhinorrhea, symptomatic side	4
Tearing, symptomatic side	4
Blurred vision, symptomatic side	9
Redness of eye, symptomatic side	4
Edema of eyelids, symptomatic side	8

half of the patients experienced nausea and vomiting. Approximately 50% of the patients had a feeling of uneasiness or discomfort in the throat that was difficult for them to describe precisely. Three patients described it as "discomfort"; two called it a "feeling of dryness in the throat"; two also used the expression "hindrance to proper swallowing", and one described it as a "lump in the throat". We have classified these sensations as "discomfort in the throat".

Two of the patients volunteered the information that they had blurred vision on the pain side during attacks. With regard to seven patients, this information surfaced on specific questioning. In one patient, the blurred vision preceded the attack; in the others it did not appear until the attack had lasted for a relatively long time, or it appeared at the height of the attack. The blurring seemed to be of moderate degree, appeared evenly in the visual field of the ipsilateral eye, and was without any flickering/scintillating qualities.

All the patients have been observed during attack, but only four of them at the height of attack. During a pain attack, all the patients generally looked pale, and they gave an impression of general "illness". Two of the four patients observed at the attack maximum were vomiting. Patients seen during attack generally tried to minimize conversation. In four of the patients we observed a moderate redness of the ipsilateral eye (Table 5), and eight patients had a clinically apparent, moderately expressed edema of both the upper and lower eyelid on the symptomatic side.

Dizziness was described by nine patients; in two of them it was of a more than mild degree; in one of these patients it was invariably brought about by extension in the neck. In one patient, the dizziness was continuous without any particular temporal relationship to the pain attacks. The dizziness did not have any rotational quality in anyone.

In some of our patients we have noted a facial erythema on the symptomatic side during long-lasting attacks, mostly in the infraorbital area. The patients themselves have also noted such erythema. Because of the vagueness of this manifestation, we would not like to express any definite opinions as to the frequency of this phenomenon.

Warning symptoms

Four patients experienced warning symptoms. One patient initially always felt a throbbing sensation in the neck on the pain side. She was then sometimes, but not always, able to abort the attack by taking analgesics and going to bed. In another patient, an attack might be initiated by jabs-and-jolts-like pains in the back of the head. In still another patient, pain of lesser intensity in the usually painful region heralded the attack; one patient was warned of an oncoming attack by blurred vision on the symptomatic side.

Precipitation of attack

All patients except one could precipitate attacks by certain movements of the head. These 10 patients got attacks in connection with prolonged awkward positioning of the head. Typical precipitating positions are those connected with washing the ceiling, polishing the floor, backing a car, sitting next to and turning the head towards someone during conversation, and so forth (Table 6). Some patients woke up with a typical headache attack in the morning and some stiffness in the neck. They felt that awkward positioning of the head during sleep had contributed to the headache. The latency from beginning of a stimulus, such as extension of the neck, until onset of a pain attack usually varied from a few seconds to 5–10 min; occasionally, however, it might take 20–

Table 6. Head movements leading to precipitated attacks.

	No. of cases
Turning of the head	8
Bending forwards	5
Bending backwards	5

A total of 10 patients could precipitate attacks by one head movement or another.

30 min to precipitate an attack. Five of the patients reached attack maximum in less than 1 h; in the rest it was attained within 1–5 h. There is a considerable intraindividual variability in the ability to self-generate attacks.

In six patients, Valsalva maneuvers could precipitate attacks, provided the stimulus was marked enough (such as coughing and bowel movements). Three patients claimed that mental stress also could precipitate attacks, and in four patients moderate quantities of alcohol provoked an attack. There was frequently a feeling of stiffness in the neck and/or crepitation on neck movements.

Physical findings

There were few findings on physical examination. All the patients had slightly to moderately reduced range of motion in the neck, particularly on rotation. This was assessed by inspection only. During attacks, the patients tried to minimize neck movements, and they even resisted passive movements.

In five patients, we found a minimal decrease of cutaneous sensation for touch in the trigeminal area on the symptomatic side during a non-symptomatic period. In one case, a reduced sensation was present only in V_1, in only one in V_2 and V_3; in the rest, complete V involvement was present. There seemed to be a vaguely decreased sensation in the C_2 and C_3 areas on the symptomatic side in one case. In one patient, hyperesthesia was observed above and behind the ear on the symptomatic side during attacks.

Trigger points

In all patients except one, pain with the same distribution and characteristics as during the spontaneously occurring attack could occasionally or constantly be precipitated by firm manual pressure directed toward distinct points in the neck/nape of the neck (Table 7) (the one exceptional patient could precipitate attacks by movements in the neck). In all 10 cases, more than one such trigger point could be identified. By prolonged pressure, manifestations similar to those of an actual attack could be precipitated—that is, a long-lasting pain, outlasting the circumscribed period of pressure application by far. On some occasions, attacks lasting the entire day could be precipitated.

Our impression is that attacks can be precipitated in all these patients provided that the stimulus is strong enough. In some patients, however, attacks could be precipitated on some occasions but not on others. This *may* have to do with a varying susceptibility to attacks. It is our feeling, however, that this, at least partly, resides in difficulties on the part of the examiner in localizing exactly the point most sensitive to external pressure. It may also be due to the fact that it is hard to exert the pressure to the same extent at different times. Inter-examiner differences in examination also may play a not inconsiderable role in this context.

In control individuals, a localized pain may be elicited by a firm pressure at the various "trigger" points (see Table 7), but we have

Table 7. Localization of "pressure" points.

	No. of cases
Midway between external occipital process and mastoid process	8
C_2 area—that is, behind and just below the mastoid process	8
Transverse processes of C_4/C_5	8
Muscle insertion of the external occipital protuberance	1

In a total of 10 patients attacks could be provoked from the pressure points.

never observed a *lasting* pain or anything similar to an attack.

Discussion

Around 1980–1981, we became aware of a few patients with a unilateral type of headache that we were unable to classify when applying the current classification system (4). Since such a group is not depicted in the classification systems presently in vogue; it does not seem to belong to the recognized and accepted headache entities. The rather thorough study of our patients in the course of the last few years has substantiated our original belief that, from a clinical point of view, we are probably faced with a rather homogeneous group of patients. Furthermore, over time it became clear to us that we probably are not faced with a small group. Since it nevertheless does not seem to be mentioned in the classification system in a recognizable form, could there be trivial explanations for this? Could the present headache group be mixed up with other, accepted headache entities, as a consequence of overemphasizing or underemphasizing certain headache characteristics in the diagnostic process? Some of the differential diagnostic possibilities will be discussed. We will in this context consider *unilateral* headaches that may be confounded with cervicogenic headache.

Differential diagnosis versus cluster headache syndrome and trigeminal neuralgia

Our patients seemed to differ clinically from patients in existing headache categories that were characterized by a pattern of repetitive attacks of a strictly unilateral headache—that is, cluster headache, CPH, and trigeminal neuralgia. Attack duration and frequency in our small group seemed to differ from those of cluster headache, and even more from those of CPH and trigeminal neuralgia (see inclusion criteria, Table 1). A typical cluster pattern, as observed in cluster headache, was not found in our group of

Table 8. Comparison with other unilateral headache syndromes.

	Sex preponderance	Attack duration	Usual frequency of attacks	Intensity of pain	Arm/neck/shoulder pain	Precipitation by neck movement	Pressure-point precipitation
Cervicogenic HA	F	Long-lasting	1/week	"More sick than corresponding to pain"	+	+	+
Cluster HA	M	Rel. short	1–3/day	Very intense	+	–	–
CPH	F	Short	>15/day*	Very intense	+	+–	+–
Trigeminal neuralgia	M/F	Ultrashort	Multiple	Very intense	–	–	+

* Maximum individual frequency.

patients. Even in CPH there is a tendency to clustering of attacks ("modified cluster pattern").

As for the signs of "neck involvement", the picture was somewhat more intricate. Several of the basic requirements in our group also pertain to CPH (see inclusion criterion 4 in Table 1). Thus, diffuse pain in the ipsilateral shoulder and arm is not infrequently met with in CPH, and occasional CPH patients can even precipitate attacks mechanically (5, 6). A reduced range of motion in the neck is not customarily found in CPH in our experience. In cluster headache, a mechanical precipitation mechanism has not been found (6).

There were thus *a priori* appreciable differences between our group and the other existing headache groups.

A schematic comparison of the four entities is presented in Table 8. In addition to the differences seen in the Table, we believe that the distribution of pain also clearly differs. In cluster headache and CPH, the pain maximum regularly is in the ocular/periocular area; in trigeminal neuralgia, it usually is in the innervation area of the IInd or IIIrd branches of the Vth cranial nerve. In addition to an "anterior" localization, the pain in cervicogenic headache is also felt posteriorly. Furthermore, in CPH there is an *absolute* response to indomethacin, not found in cervicogenic headache.

We thus feel justified in stating that from a *clinical* point of view the characteristic traits of cervicogenic headache seem to set it apart from the other headaches mentioned so far. It will be necessary, however, also to assess the pathogenesis in this unilateral headache in a search for differences. The results of such a search will be reported at a later stage.

Although cervicogenic headache generally differs clearly from cluster headache, the diagnostic difficulties versus cluster headache may arise in a male patient with relatively short-lasting and frequent attacks. In two of our own cases (one female and one male), the diagnostic difficulties, even with all the supplementary tests available (to be detailed in later communications), have been almost insurmountable.

Relationship to cases described by Hunter and Mayfield and by Barré

In our opinion, the cases described by Hunter & Mayfield (7) are rather similar to ours. They described patients with unilateral headache that was "side-locked", the attacks being rather long-lasting and relatively rare. During relatively severe attacks, the headache could be felt in the entire head. There was ipsilateral tearing, nasal stenosis, and flushing of the face. Some of their patients complained of giddiness and even vomiting. It is noteworthy, however, that Lance (8) has discussed the possibility that Hunter & Mayfield may have seen cluster headache cases. This was one of the reasons why it was felt strongly that the differential diagnosis versus cluster headache really should be given due consideration both in this context and in future research.

Although there have been many reports on headache related to the neck, most such materials to some extent seem to differ from our material. Barré (1) described a headache variety comprising vertigo, tinnitus, and visual disturbances. It was not specifically mentioned whether the headache was unilateral. These features only to some extent conform with the characteristics of cervicogenic headache: Tinnitus is a rare accompaniment of cervicogenic headache. Furthermore, dizziness, although present in a relatively large fraction of our cases, invariably was of low degree. There was no case of true vertigo. In our cases, the headache was strictly unilateral. This is not intended to imply that there may not exist two-sided headache or "unilaterality on both sides" in cervicogenic headache. At present, however, when the etiology of this headache has not been established, and when its status is so dubious, we do not feel that the time has come to include "bilateral" cases in this diagnostic category. We fear that by including bilateral cases there may easily be a "contamination" of the material. The strategy in the exploration of this headache should therefore, as far as we are concerned, be to describe the characteristics of the unilateral cases with both clinical and laboratory variables as thoroughly as possible, before any extension of the group

is allowed. In this manner we will have a firm basis for comparison with bilateral cases and with common migraine cases at some later date.

In addition to Hunter & Mayfield, Raney & Raney (9) may have described patients similar to ours. They described patients with headache caused by cervical disc lesions. "The 'headache' may be located in the scalp, the face, or the cervical, suboccipital, or other regions. It is often unilateral, but may be bilateral". They state that it may be short-lasting or "it may be thought of as a 'sick headache' lasting twelve to seventy-two hours or longer. If severe, it may be associated with nausea or vomiting". They further speculated that visual disturbances that they evidently had found might be caused by extensive drug consumption. It is noteworthy that the head pain was not relieved by section of the root of the trigeminal nerve or by tractotomy, or by both, even when the pain was confined within the zone made anesthetic or analgesic. Cervical sympathetic block did not affect the headache unless the procaine was poorly deposited, so that it affected the cervical muscle and nerve roots, in which case some relief from the headache might result.

In a further search for etiology and pathogenesis in this type of headache, Hunter & Mayfield (7) successfully blocked the homolateral C_2 root and obtained a more lasting and beneficial effect after intradural transection of the root. In a previous report (10) from our group, a good transitory effect was demonstrated by blocking the C_2 root in 9 of 11 patients in this category (not identical with the patients in this report). These blocking procedures may seem to link this headache syndrome with the innervation area of the C_2 root. Bogduk emphasized that if one were to claim a cervical cause of a headache, one should be able to block the pain by anesthetizing the structure in question (11, 12). Jansen & Spoerri (13) in five patients with unilateral frontal pain and retroocular headache found a compression of the C_2 or C_3 root, in three of them due to a venous plexus. Postoperatively, all patients were relieved of pain, and the follow-up period was up to 7 years.

The clinical picture of major occipital neuralgia also in many respects seems to be reminiscent of that in our group (14). The prevalence of this neuralgia is little known and the clinical manifestations rather vaguely outlined.

There is, therefore, some circumstantial evidence to associate unilateral headaches in our group and in patients with similar symptoms (7) with pathology within the C_2 (or C_3) root/innervation area.

On the other hand, Pasztor (15), Kehr et al. (16), and Grønbæk (17), seem to have had considerable success with "uncoforaminectomy" at a lower level in the cervical spine—at the C_5-C_6-C_7 levels—in cases in many respects *similar* to ours, through a "liberation" of the nerve roots and the vertebral artery in this region. We have a feeling that in their cases the vertigo may have been more prominent than in our cases. This may possibly be an indication of a different localization of the pathological condition. The possibility thus exists that our cases and the cases of Hunter & Mayfield, on the one hand, and the last-mentioned groups, on the other, differ in essential respects. Conversely, it may be that intervention at *various* levels in the cervical spine may influence the total sensory input from this area and that this may modify the entire situation in a beneficial manner.

Differential diagnosis versus migraine

Another possibility for diagnostic error as far as our group of patients is concerned is migraine. The term migraine is derived from hemicrania, and, in principle, migraine is a unilateral headache. In 45–65% of the cases of common migraine the headache is said to be unilateral. A typical, although rather infrequently mentioned—and partly forgotten—trait of migraine is that in general the headache changes side within the solitary attack or between attacks. We believe that there are exceptions to this rule and we believe that we have observed such cases. Such cases may nevertheless appear to be rather rare. It should be emphasized in this connection that since there are no tests that can verify or refute a diagnosis of common

migraine, the seeming exceptions to the rule may be more apparent than real. It therefore appears that, whereas in common migraine there generally is a combination of *relatively* short-lasting attacks (1–2 days) and either global or unilateral headache with side shift, in our group of patients the headaches are unilateral, and the attacks are usually more long-lasting (the longest attacks in all cases except one being ≥3 days). The neck symptoms and the precipitation mechanisms are to the best of our knowledge not present in common migraine, although no precise information is available. On the other hand, it may happen that not all patients with cervicogenic headache can precipitate attacks.

The above-mentioned evidence tends to show that this headache is at variance with regular common migraine. This assumption is corroborated by the fact that in our group the usual migraine medications (ergotamine and beta-receptor blocking agents) have no or only minimal effect. However, as long as we do not know the specific pathogenesis and etiology of these headache forms and do not have specific diagnostic tests for them, differential diagnostic problems may conceivably be insurmountable in present-day headache practice. In all probability, several cervicogenic headache cases are at present being classified as cases of common migraine (18).

The intricacy of the differential diagnostic problem versus common migraine is nevertheless evident from the fact that if the migraine criteria of Vahlquist (19) or of Bousser et al. (20) are used, cervicogenic headache would easily pass for migraine (Table 9).

Bärtschi-Roschaix (21–23) described a headache, including both bilateral and unilateral cases, and used the term "migraine cervicale". In the early communications, the *unilaterality* was emphasized (22), the bilaterality being more stressed in later publications. The term may convey the impression that this is a type of migraine with particular cervical symptoms or that the origin of this particular type of migraine is in the neck. Apparently, Bärtschi-Rochaix felt that both conditions applied. The clinical picture described by Bärtschi-Rochaix (22) seems to be rather similar to the picture described by Barré (1). Although there seem to be differences versus the pictures described by Hunter & Mayfield (7) and by our group, there

Table 9. The migraine criteria of Vahlquist (19) and Bousser et al. (20) applied to cervicogenic headache.

Migraine	Cervicogenic headache
A) Vahlquist's criteria: Paroxysmal headache + two of the following four criteria: 1. Nausea 2. Scotoma or related symptoms 3. Unilaterality 4. Familial occurrence, similar headache	Symptoms: Paroxysmal headache + (1) Nausea 2. Unilaterality
B) Bousser et al criteria: Recurrent, idiopathic headache lasting 3 h to 3 days + two of the following five items: 1. Nausea/vomiting 2. Familial occurrence 3. Unilaterality 4. Pulsating headache 5. Phono- or photo-phobia	Symptoms: Recurrent headache lasting 3 h to 3 days (1) Nausea 2. Unilaterality (3) Pulsating headache (4) Phono- or photo-phobia

Cervicogenic headache will probably fulfill the criteria of migraine in >50% of the cases (since, for example, nausea was present in 6 of 11 patients in the present material).

are also many similar features. In spite of the appellation used, "migraine cervicale" might, therefore, possibly have been dealt with in the context of the cervical roots in the present communication.

In summary, it seems to us that cases like ours have been described previously. Earlier materials may partly have been contaminated with cases that do not belong to the group (as may ours!). This is probably one of the reasons why this headache form has not been recognized and accepted as being separate.

There is at present not enough evidence available to make any statements regarding *etiology*. As far as the *localization* is concerned, the possibility exists that the headache we describe could be related to various structures in the neck, presumably the C_1, C_2, or C_3 root fibers, the occipital nerves, and possibly the uncovertebral joints and the vertebral artery. We therefore feel that the non-specific, descriptive term "cervicogenic" (that is, arising in the neck) should be used for this headache at the present.

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