THE ABORTIVE EFFECT OF LOCAL ANESTHETIC AGENTS IN PROVOKED CLUSTER HEADACHE ATTACKS

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Summary

It is well known that the administration of the vasoactive agent nytroglycerine is able to trigger spontaneous-like attacks in most cluster headache (CH) sufferers. There is little evidence on the effect of locally administered anesthetics such as cocaine and lidocaine in aborting nytroglycerin-mduced CH attacks. Thirteen patients suffering from CH, diagnosed according to the IHS criteria (6 with episodic CH, mean ± SD age 36.8 ± 5.6 yrs, and 7 with chronic CH, mean ± SD age 40 ± 10.8 yrs) were studied. They underwent anterior rhinoscopy to rule out endonasal pathology or malformations. All patients were given trinitrine 0. 6 mg sublingually and were carefully monitored during the test. The intensity of pain was scored using a visuo-analogic scale (range 0-10). Seven patients (2 with episodic CH and 5 with chronic CH) experienced a spontaneous-like attack within one hour. Once pain severity had reached 5 or more on the scale, in each patient a solution containing cocaine 1g hydrochloride (A) or 10% lidocame (B) or placebo (C) was randomly applied under anterior rhinoscopy to the area corresponding to the sphenopalatine ganglion of either the nonsymptomatic or the symptomatic side, for 5 minutes. The latency of any observed anesthetic effect and modifications of neurovegetative simptoms were carefully recorded. Complete extinction of the attack occurred after 35 ± 11.3 mins for solution A, 42.5 ± 8.8 for solution B and 53.7 ± 10.3 for solution C, the latency for solutions A and B being significantly lower than placebo ($p<0.01$ and $p<0.05$, respectively). This study suggests that both lidocaine and cocaine locally administered to the sphenopalatine fossa may be useful in the abortive treatment of CH attacks. Lidocaine also appears to be preferable to cocaine with regard to possible risks of addiction. Moreover, these findings further support the involvement of the sphenopalatine ganglion in the pathogenetic mechanisms of CH attacks.

Introduction

The pathogenetic mechanisms of cluster headache (CH) are still poorly understood. Among the various theories, the involvement of the sphenopalatine ganglion in the generation of pain has been originally proposed (1) and the sphenoidal sinus has been since regarded as an area central to the pathophysiology of the disorder (2). Very little is known about the possible effectiveness of anesthetic substances acting locally at this level. Few studies have suggested a prompt abortive effect of agents administered in the proximity of the sphenopalatine ganglion on spontaneous/provoked CH pain (3, 4). Since nitroglycerin is a long known substance able to precipitate CH attacks (5, 6), in the present study we have used this model of provoked pain in CH patients in order to: 1) test the effect of lidocaine and cocaine locally administered on pain intensity, and 2) further elucidate whether the sphenopalatine area may be actually involved in CH attacks.

Subjects and Methods

The study group consisted of 13 patients, 11 males and 2 females, suffering from CH in active phase (6 with episodic CH, and 7 from chronic CH), diagnosed according to the IHS criteria (7). Their mean ± SD age was 38.5 ± 5.6 years,

while symptom duration was of 7.6 ± 4.3 years (age of onset 30.9 ± 7.9). The characteristics of the population studied are reported in Tab. 1. All patients underwent anterior rhinoscopy to rule out endonasal pathology or malformation.
The induction test was performed as follows: 0.6 mg nitroglycerin were administered sublingually, and patients were carefully monitored throughout the study. Patients were instructed to score the intensity of any provoked pain according to a visuo-analogic scale (range 0-10), every 5 mins. The possible occurrence of pain as well as local/general autonomic signs and symptoms was recorded using a dedicated chart. Three different solutions were employed, one containing 1 gr cocaine hydrochloride, one 10% lidocaine, and one saline, respectively.
Only in the patients experiencing a typical, spontaneous like CH attack, once pain intensity had reached 5 on the scale, the solutions were applied randomly using a cotton swab (in 3 separate, consecutive sessions) to the sphenopalatine fossa, under anterior rhinoscopy, on either the symptomatic or the contralateral side. Swabs were left locally for approximately 5 mins. Any change in pain intensity after the administration of each solution was carefully recorded, as was the latency of the observed anesthetic effect. Extinction time was taken as the time elapsing from the administration of a solution to the complete disappearance of any pain (0 on the visuo-analogic scale).

RESULTS

Seven out of the 13 patients (2 with episodic CH and 5 with chronic CH) experienced a nitroglycerin-induced headache attack, (a mean latency 46 mins), with spontaneous-like features and pain intensity of 5 or over.

The temporal profile of pain intensity for each solution in these 7 patients is reported in Fig. 1. After both cocaine and lidocaine, pain intensity decreased more promptly than after placebo, and the effect was more evident within the first 20 mins, particularly in the case of cocaine. As shown in Fig. 2, complete absence of

Table 1: Clinical features of patients

N.	AGE	SEX	PAIN SIDE	CH TYPE	DURATION (YRS)
1	36	M	L	SECONDARY CHRONIC	14
2	56	M	L	SECONDARY CHRONIC	15
3	31	M	R	PRIMARY CHRONIC	5
4	53	M	L	PRIMARY CHRONIC	4
5	34	M	L	SECONDARY CHRONIC	7
6	35	M	R	EPISODIC	13
7	41	M	R	EPISODIC	10

Figure 1: The effect of local Cocaine (——), Lidocaine (···) and Placebo (– –) on induced by nitroglycerin in 7 CH patients. Values are expressed by Mean ± Standard Error.

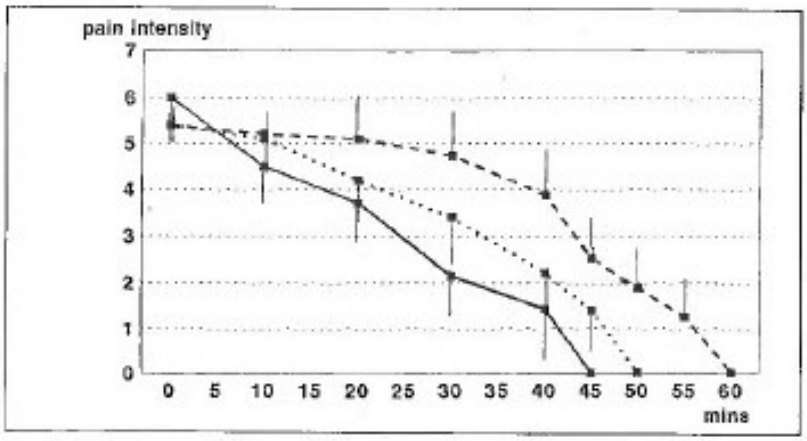
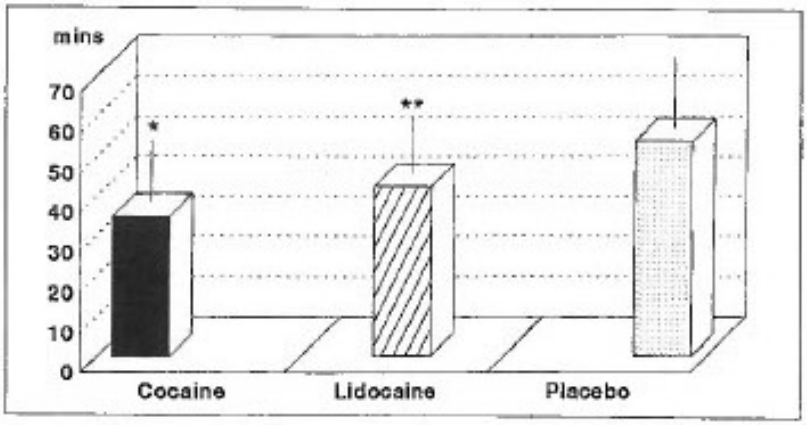


Figure 2: Mean extinction time ± Standard deviation with Cocaine, Lidocaine and Placebo in Cluster Headache; p<0.01 vs lacebo, ** p<0.05 vs placebo



pain occurred after 35 ± 11.3 mins for cocaine, 42.5 ± 8.8 mins for lidocaine, and 53.7 ± 10.3 mins for placebo, the extinction time for the latter being slightly shorter than that of the usual unprovoked attacks. The latency of cocaine and lidocaine was significantly lower than that of placebo ($p<0.01$ and $p<0.05$, respectively).

Discussion

In this study, nitroglycerin administration was able to induce a significant pain attack in 7 out of the 13 CH patients (54%), a proportion slighty lower than that observed by other authors (6). The effect of nitroglycerin is now thought to occur via formation of the potent vasodilatator nitric oxide (NO) (8). Intranasal application of cocaine was able to attenuate pain intensity within 5 mins, while lidocaine induced a significant effect starting after 10 mins. Both substances preved to be more effective than placebo in attenuating CH pain, with a significantly lower extinction time. These findings in line with previous reports (3, 4), suggest that cocaine and lidocaine locally administered to the sphenopalatine fossa may be useful in the abortive treatment of CH attacks. Althought slightly less prompt than cocaine in its anesthetic effect, lidocaine may be preferable to the latter with regard to possible risks of addiction. Our data also lend further support to the view that the sphenopalatine ganglion may be involved in the pain mechanisms of CH attacks.

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