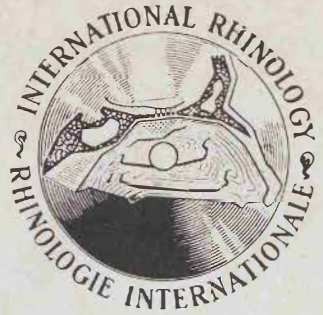

RHINOLOGY

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The nasal patho-physiology of headaches and migraines. Diagnosis and treatment of the allergy, infection and nasal septal spurs that cause them

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INTRODUCTION

This study involves 80 patients suffering from chronic or recurrent headaches, migraines included. They were managed as follows:

1. Control of the infection in the nose and sinuses.
2. Control and immunotherapy of the inhalent and food allergies.
3. Surgical correction of the impacted turbinates with nasal septa, when indicated.

This resulted in permanent relief of the headaches of 1 to 35 years duration.
Review of the literature for the nasal origin of the headache is presented. The mechanism of the onset is believed to be a central antigen-antibody reaction causing release of histamine and neurotransmitters. When these target the nasal mucosa it becomes oedematous and when they target the vascular turbinates they hypertrophy. Thus pain stimuli in the head and neck area will be triggered. Existing inflammation and a deformity of the septum exaggerate this response. Neurotransmitters targeting other distant organs give the acephalgic migraine symptoms. The compromise of the balance of serotonin and noradrenaline centrally accounts for the migraine personality and disturbance in the central autonomic control.
All pharmaceuticals used for the treatment of headaches will be shown to prevent or reverse this allergic-inflammatory mechanism and its sequelae.

PATIENTS AND METHOD

Eighty eight consecutive patients with headaches were included in the study. Eight patients, who did not follow our recommended treatment, i.e. controlling the allergy/surgery as indicated, were used as controls.

Diagnosis

1. Thorough interviews are conducted with special reference to the precipitants of the headache.
2. All patients are clinically and neurologically examined. Laboratory tests, X-rays of the sinuses, CT scan, as required, are done to rule out local or distant organic causes other than in the nose and sinuses (List, 1968).

3. Cocaine solution is applied to the medial surfaces of all the nasal turbinates on the left or right or both sides depending on the location of the headache (Henderson, 1977; Ryan, 1978; Novak, 1984).
   The middle turbinates are treated first. Prior to application of the cocaine the patients are told that it is necessary for proper examination of the nose that this "decongestant" solution be applied. The patients are then surprised at the improvement in their nasal airway, together with disappearance of the headache. This step helps in gaining the confidence of these patients. Anterior rhinoscopy now may show nasal septal spurs, the extent of shrinkage of the turbinates is determined and studied with the Caldwell view of the X-rays of the sinuses (Figures 1 and 2).
   Rhinomanometry may be done to establish asymmetry between the two nostrils and is important for the patients to understand why one side of the head feels different.

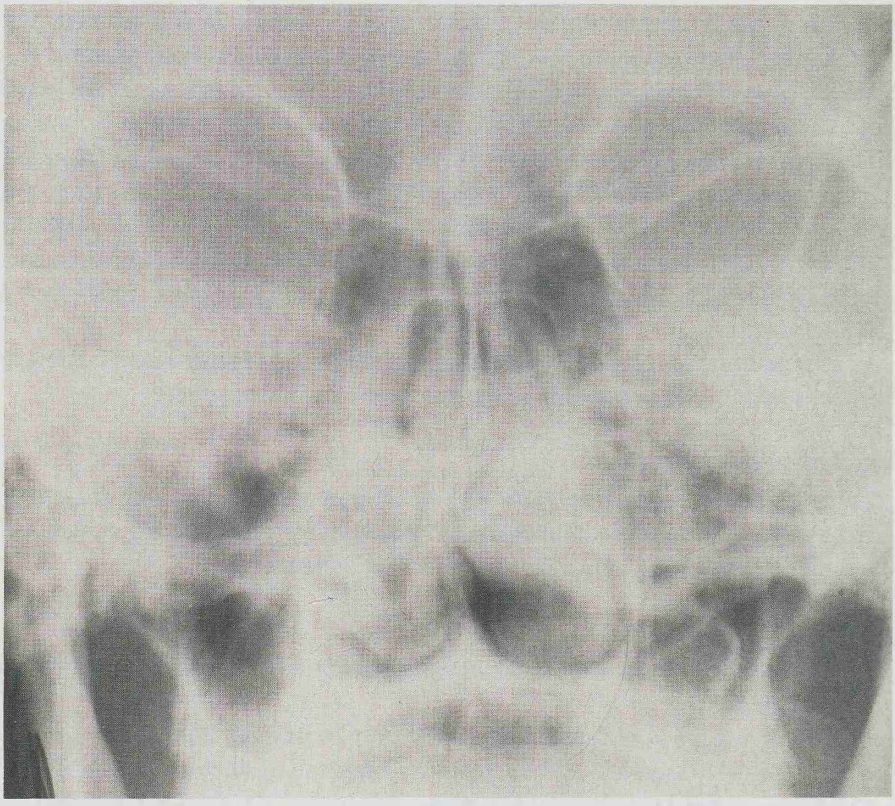


Figure 1.

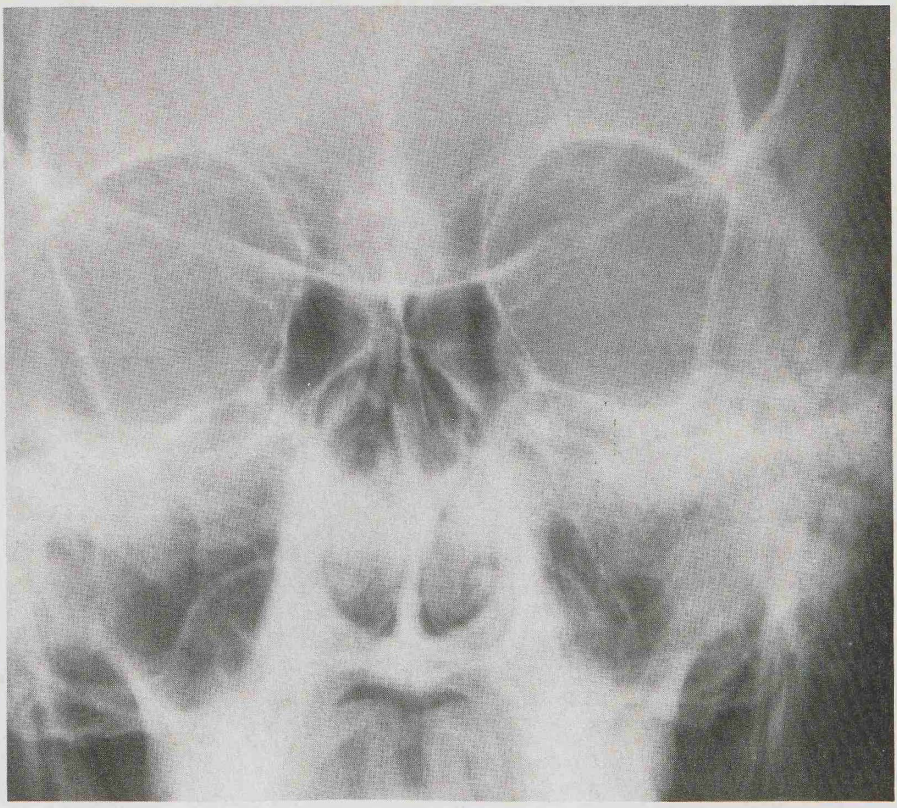


Figure 2.

Figure 1 and 2. The Caldwell view of the X-rays of the sinuses of two patients showing the impacted deviated nasal septa with the hypertrophied turbinates.

4. RAST test (Wide et al., 1972) is done on the patients' serum. Twelve commonly eaten foods together with inhalents that comprise grasses, trees, weeds, molds, house dust mite, dog and cat dander and hair are tested. Hence allergic patients are identified very early.
5. A diet interview is conducted and foods and drinks that are consumed frequently are noted. Intradermal provocation testing with food antigens is done (Willoughby, 1969; Miller, 1972).
   Foods that showed high IgE are tested to single out those that induce the headaches, as some foods that have high IgE may be producing other allergic reactions e.g. urticaria, bronchospasm, vertigo, etc. (Figure 4).
6. A diversified, 4-day-rotating diet is made up for each patient to be followed four weeks. They are advised to eat no more than three foods per meal. They can eat up to six meals per day. Headache inducing foods, detected by the pro-

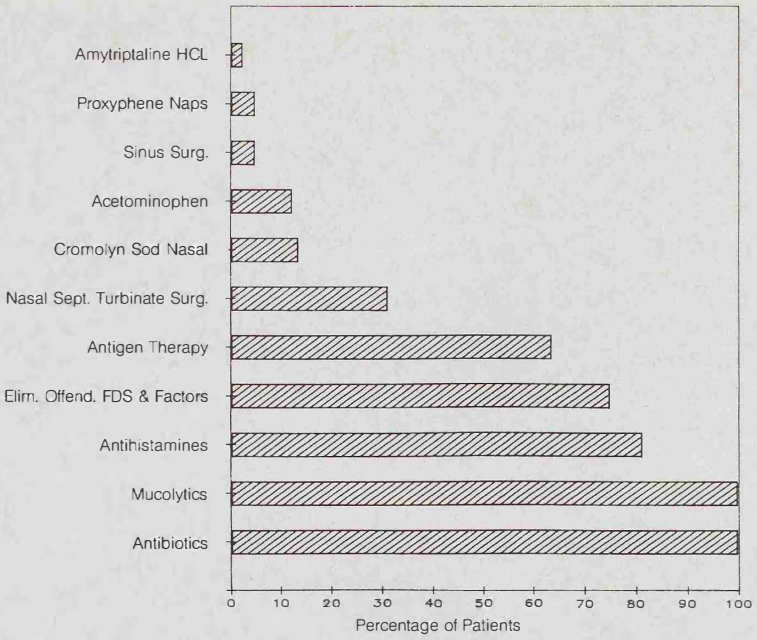


Figure 3. Treatments utilised as indicated by the results of the diagnosis.

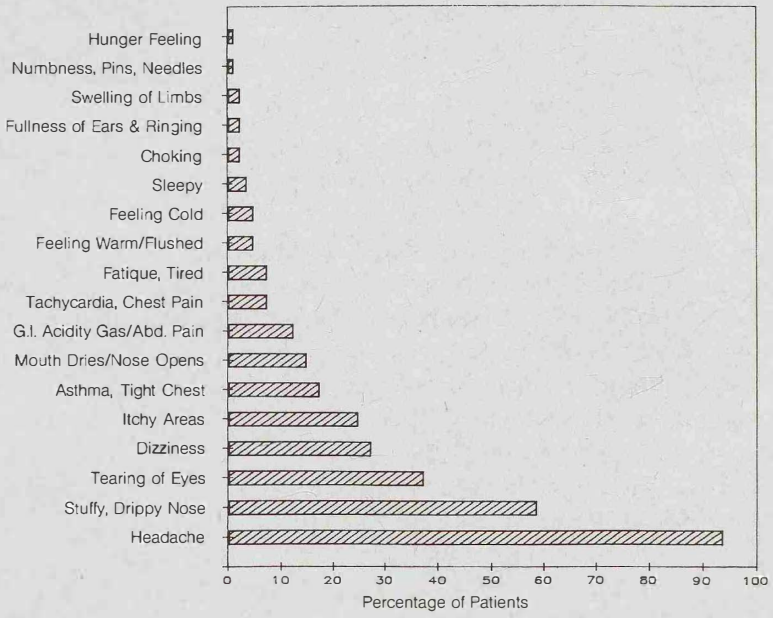
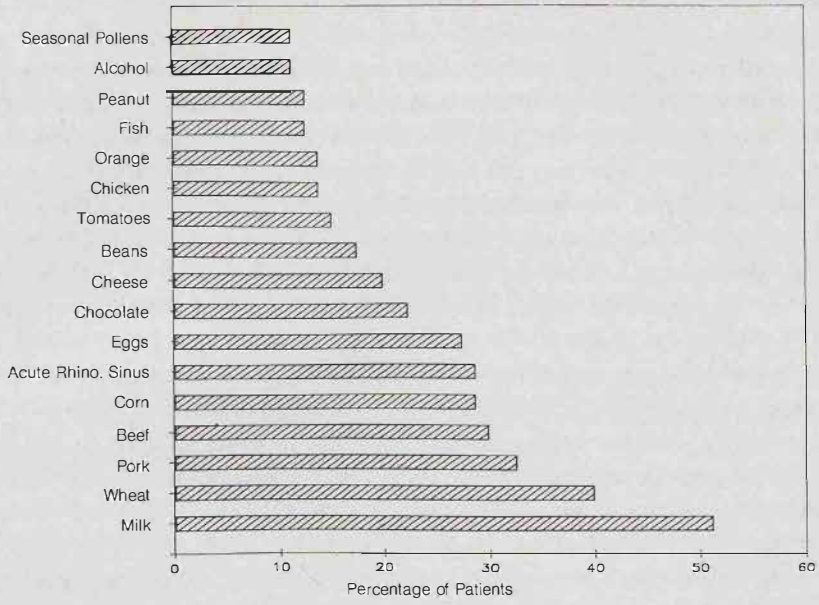


Figure 4. Symptoms reported by patients on provocation with foods and/or food antigens.

vocation method, are completely eliminated. The patient keeps a diary of foods and drinks, or any other association, that caused their headaches (Figures 5 and 6).



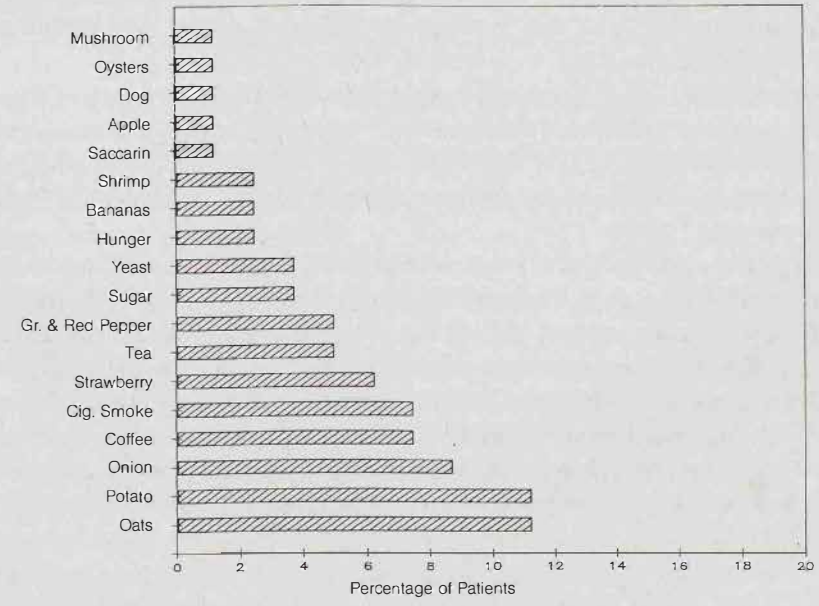


Figure 5 and 6. Precipitating causes of headaches according to patients' diaries.

Any other symptoms also are to be recorded, such as vertigo, urticaria, etc. (Figure 4). After these four weeks the patient would have identified and confirmed the new foods that induce headaches in addition to those that were diagnosed by the RAST and provocation methods.

Treatment

1. All patients were given antibiotics and mucolytics together as per their tolerance. It was found that 7-10 days courses of two or three of the following antibiotics was necessary to control the chronic low grade infective rhino-sinusites. They were Trimethoprim sulfamethoxazole, Cephalexin, Erythromycin, Doxycycline and Amoxicillin. Mucolytics used are guaifenesin (Humibid, L.A.®) and/or iodinated glycerol (Organdin®). These are given for four weeks initially and may be repeated as needed. "INFECTION IN THE NOSE AND SINUSES MUST BE CONTROLLED" (McAuliffe et al., 1943).
   The patients are taught how to test for tenderness of the maxillary sinuses by pressing with their index fingers on the lower parts of the cheeks, and they report whether this is accompanied by coloured nasal discharge and/or nasal obstruction. Further antibiotic and mucolytics treatment are prescribed and this will prevent any future recurrence of the headaches due to infection.
2. Impacted turbinates with a deviated nasal septum are surgically corrected (Ryan, 1976; Koch-Henriksen et al., 1984; Novak, 1984). Advanced sinusitis has to be dealt with surgically if necessary. Ethmoids, sphenoid, frontal and maxillary sinuses have to be free of disease and draining well.
3. Immunotherapy to all food and inhalent allergy is started with the allergic patients (Franklin, 1984; Lehrer et al., 1986).
4. Patients now can eat all the safe foods, those that have severe inhalent allergy are given cromlyne nasal (Nasalchrome®) twice a day and prior to exposure in areas where there is heavy cigarette smoke or visiting friends that have pets. Those that get severe abdominal symptoms are given H_2 blockers, cemitidine (Testa et al., 1986).
5. Patients are also advised to avoid missing meals, avoid fatigue and are advised to sleep 6-8 hours daily. They are made aware that hunger, fatigue, mental conflict and exertion and lack of sleep will cause turbinate engorgement through hypothalamic stimulation, and will make their headaches easier to trigger.
6. Office support is offered by telephone calls and follow-up visits; as patients addicted to certain foods resent the fact that they have to give them up for any length of time. Also these patients have special difficult personalities as will be explained in the discussion section that need much more attention than other individuals.

RESULTS

All eighty patients treated were free of headaches, the duration of these headaches, prior to treatment, was between 1 and 35 years.

Twenty-nine patients had surgery on the nasal septum / turbinates / sinuses. All are well (follow-up periods: 2-6 years). Ten patients had nasal septum deviation but control of their allergies relieved their headaches without surgery. Out of twenty-nine patiens that were operated on, eleven had allergy treatment and eighteen did not need allergy treatment. All were free of headaches (follow-up periods: 2-6 years). Thirthy-nine patients had allergy-induced headaches without septal deformities or sinus pathology (Table 1a, 1b). Allergy treatment alone relieved their headaches. They found out what precipitated their headaches and learned to avoid them. Immunotherapy helped them to develop the phenomena of tolerance. They were also taught not to eat foods they liked repetitively and hence reactions to such foods in the future are prevented.

Table 1a. Distribution of the patients according to their diagnosis; showing those that needed surgery versus those that improved on allergy treatment only.

total number of patients	80	surgery	no surgery
impacted septum/turbinates	33	23	10
impacted turbinates	3	1	2
pathology in sinuses with impacted turbinates	5	5	0
allergic headaches with no deviated nasal septa	39	0	39

Table 1b. Distribution of patients according to the treatment they received, whether allergy, surgery or both.

total number of patients	80	100%
had allergy treatment only	52	64%
had surgery only	11	14%
had surgery and allergy treatment	18	25%

Set meal times relieved the headaches of the two patients, who had hunger-induced headaches. Allergic reactions precipitants were 67.25% (Monro et al., 1980; Wilson et al., 1980; Lehrer, 1986), followed by the inflammation 28.75% (McAuliffe et al., 1943) and last only 4% were due to hunger or other hypothalamic stimulants (Holmes, 1950; Herberg, 1973; Jacob et al., 1982).

Table 2 shows the controls. These are eight patients that did not follow the recommended allergy and/or surgery as their cases indicated.

Table 2. Particulars of the 8 controls that did not follow the recommended allergy or surgery treatment as indicated.

number of patients	description of controls	follow-up period
2	had impacted turbinates/deviated septum; could not have surgery and allergy treatment	4 and 2 years
3	had allergy and food inducing migraines. Failure in breaking their food addiction; could not have immunotherapy	2, 3 and 5 years
2	had allergic headaches. Initial complete relief with diet and immunotherapy. Headaches back on discontinuation of immunotherapy	2 and 3 years
1	had surgery and also had allergy to inhalents and foods. Had complete relief of headache for two years. Now his headaches are back. He refused immunotherapy after surgery.	3 years

DISCUSSION

It was thought that there seems to be a basic biochemical/autonomic imbalance with headache sufferers, showing increase in erythrocyte coline (Nelson, 1986) and increase in sympathetic flow (Kuritzky, 1986), or inherited dysfunction specifically of the cholinergic fibers that predisposes the individual to allergic reactions as stated by Williams (Ballanger, 1977). However the above conditions can be the result of the continuous release of mediators in an allergic patient, compromising the balance of central autonomic control (Wilson, 1980).

The nasal origin of the headaches which are referred to various areas of the head and neck (Sluder, 1927; Vail, 1932) was mapped in detail after stimulating spots in the nasal mucosa over the septum, the turbinates and the sinus ostia by McAuliffe (1943) and Holmes (1950) (Figure 7a-f).

In our sample the application of the cocaine solution on the medial surfaces of the turbinates decreased the intensity of the headaches between 90-100%. It also reduced the size of the nasal turbinates and shrunk the septal mucosa. No attempt was made to reach the sphenopalatine ganglion at all (Meyer, 1970). Cocaine constricts the dilated vessels and blocks re-uptake of norepinephrine at the sympathetic neural junction. Its direct effect on the nerve cell membrane prevents generation and conduction of nerve impulses, the resting membrane permeability is reduced (Henderson, 1977).

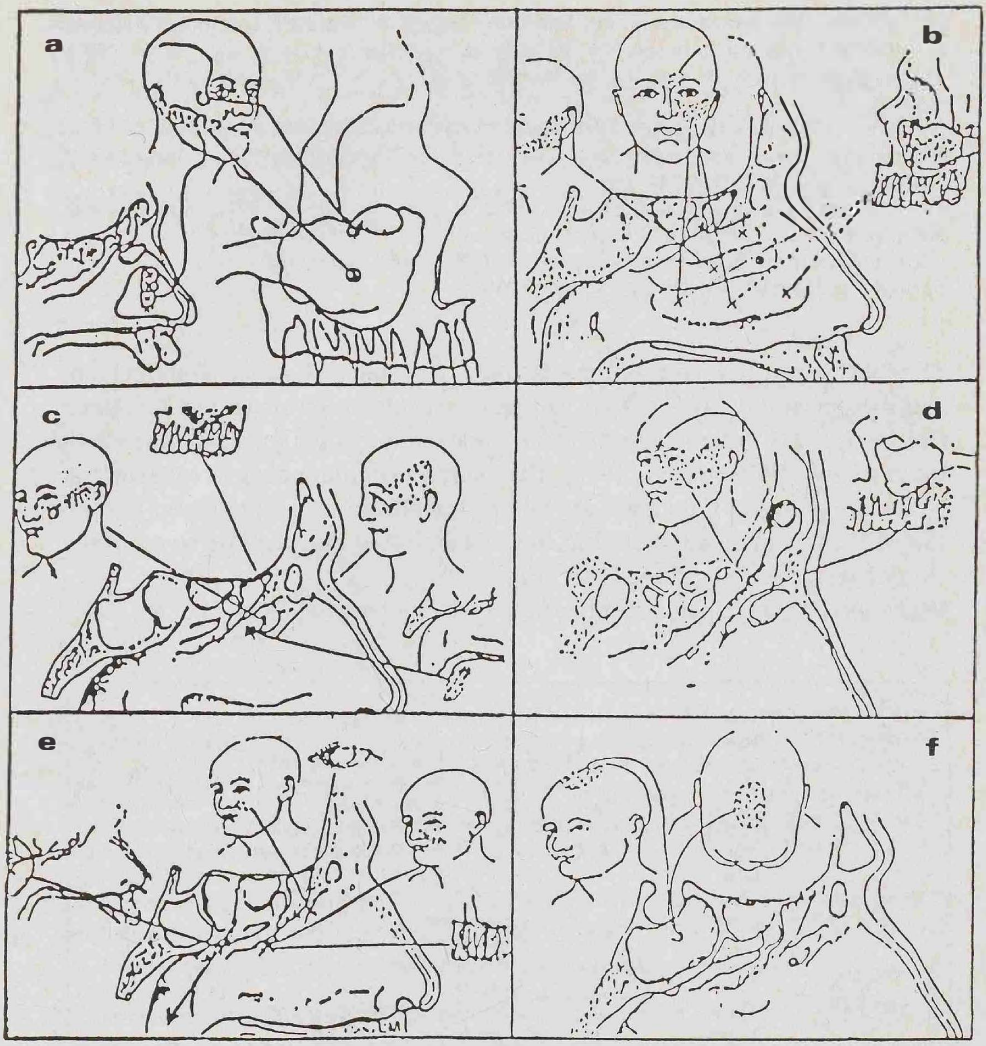


Figure 7. Various areas stimulated on the nasal septum, turbinates, sinus ostia and the corresponding anatomical locations of the referred pain.
A. The points stimulated on the septum are shown by crosses and on the lateral wall of the maxillary sinus as cross hatched circles. The areas in which pain of 1 plus to 2 plus intensity was felt are indicated by crosses within an outline on the small head above. Note that widely separated stimuli cause pain to be felt in the same areas.
B. The points stimulated on the turbinates are indicated by crosses, from which lines lead to the indicated areas in which pain of 4 plus to 6 plus intensity was felt.
C. Large crosses indicate stimulation of the ostium of the maxillary sinus. Lines lead to the areas, indicated by small crosses in which pain of 6 plus to 9 plus intensity was felt. A dotted circle over the zygoma indicates the area of erythema and hyperalgesia which long outlasted stimulation of the ostium.

D. Lines lead from the points stimulated in the nasofrontal duct to the areas in which pain of 5 plus to 7 plus intensity was felt. On stimulation of the inner wall of the frontal sinus minimal pain of no more than 1/2 plus intensity was felt only in the area indicated directly over the sinus.
E. Crosses indicate the points of pressure against the walls of the superior nasal cavity in the region of the sphenoid and ethmoid sinuses, with the indicated areas in which pain of 5 plus to 6 plus intensity was felt.
F. The area is indicated in which pain of 1 plus to 2 plus intensity was felt on faradic stimulation of the mucosal lining of a sphenoid sinus by Dr. Bromson Ray.
(Reprinted with permission from The Nose, T.H. Holmes et al. eds.
Springfield, Illinois: Charles C. Thomas, 1950)

The mechanism triggering the headache is believed to be that of an antigen-antibody reaction centrally that will result in release of histamine, serotonin (Herberg, 1973), noradrenaline and neurotransmitters from the degranulation of the mast cells (Wilson et al., 1980). The neurotransmitters, such as substance P, Neurokinins A and Calcitonin are potent smooth muscle contractors (Barnes, 1986). Various mediators released from an activated mast cell involved in the allergic response (Normans, 1985) (Figure 8).
Immunoreactive (somatostatin like) cells may be targeted (Arnold et al., 1986)

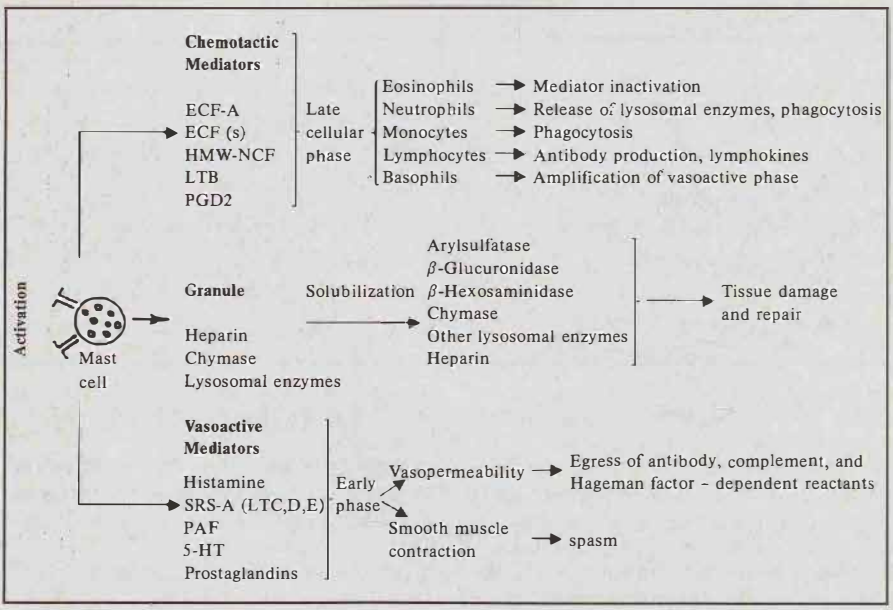


Figure 8. Various mediators released from an activated mast cell involved in the allergic response.
(Reprinted with permission from S. Norman. Antihistamines. Newsletter Syntex Lab no 10, 1985)

where by adrenergic and cholinergic neurotransmission in smooth muscle is inhibited (Cohen et al., 1978) (Figure 9).

All the precipitants of the headaches in our sample were thought to cause this initial allergic-inflammatory reaction and hence cause spasm of smooth muscles in various distant shocked organs. As any sympathetic reaction it is more generalised, the parasympathics in different locations will counteract this (Anggard, 1977; Johnson, 1978; Graham, 1982).

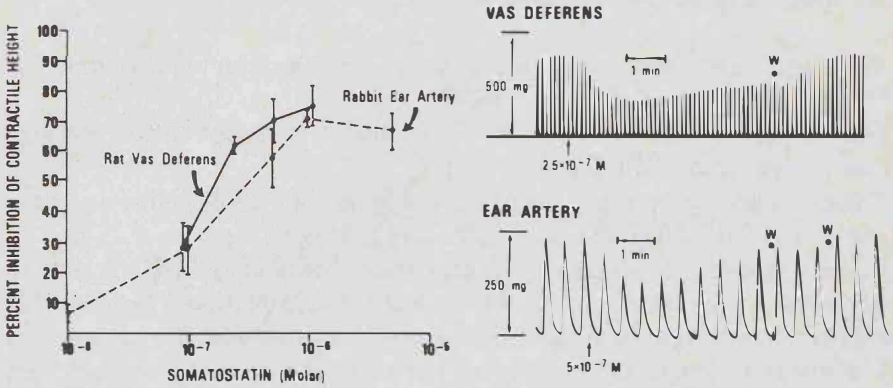


Figure 9. Concentration response curves for the inhibition of field stimulated contractions of rat vas deferens and rabbit ear artery strips by somatostatin (left). Points on graph represent mean values ± S.E. for 4-12 tissues at each point. Rat vas deferens were field stimulated at 40 v, 1.0 msec duration and .12 to .15 Hz. Rabbit ear artery strips received a 5 Hz train of pulses for 5 sec every 30 sec (40 v, 0.7 msec duration). Actual dynograph recording of the tissue concentration of somatostatin (right). Change of bathing solution to drug free Kreb's buffer is indicated by wash (w).
(Reprinted with permission from M.C. Cohen et al.: Somatostatin inhibits adrenerge and cholinerge neurotransmission in smooth muscle. Life Science 1978; 23: 1659-1664)

Spasm of the arterioles in the head will cause the aura (Johnson, 1978; Blau, 1978). In the nose the initial vasoconstriction has to be followed by the reactive vasodilatation and hence engorgement and swelling of the nasal mucosa and turbinates and hence the initiation of the headache. This accounts for the immediate allergic reaction. The nasal mucosa can be involved in the delayed reaction by the fact that chemotactic mediators will cause aggregation of granulocytes, eosinophils etc. The cell membranes permeability is affected and can cause disruption of the surface and hence the hyperresponsiveness and pain stimulation (Figure 8). Some of our patients described two varieties of headache, one they called the sinus headache and the other the migraine, the migraine is the one that throbs and can remain a day or more and may have an aura, while the sinus headache is accompanied by pressure and no throbbing. The immediate allergic reaction with

its vasoconstriction followed by the vasodilatation is believed to cause the engorgement of the turbinates and the migraines as reported by our patients in Figures 5 and 6.
The delayed reaction alone targeting the nasal and/or sinus mucosa may produce the headaches of the sinus variety. However, some delayed mediators may enhance the vasoactive phase and we may get mixed varieties.
Thermograms done on patients with all kinds of headaches show the hottest areas to be the inner canthi (where the nasal turbinates are); when there is an acute sinusitis, the cheeks and/or the forehead show hot areas.

The allergic reaction affecting other areas will cause the symptoms referred to as "Acephalgic Migraine" (Kunkel, 1986).
Smooth muscle contraction causes the vasospasm of variant angina and Reynaulds phenomena (Miller et al., 1981).
Histamine release with increase in the cell membrane permeability cause the whelps and itch of urticaria (McAuliffe et al., 1943).
Increase in gastric glands secretion causes "gastro-rhea!" or hyperacidity and gastroduedenal ulcer (Ekbom, 1970). Abdominal colic and diarrhea (Graham, 1982) are again caused by smooth muscle contraction. "Current evidence suggests that Somatostatin from immunoreactive cells exerts its effects locally" (Arnold et al., 1986). It inhibits smooth muscle adrenergic and cholinergic neurotransmission (Cohen et al., 1978) (Figure 9). Its presence has been demonstrated by immunohistochemistry in different regions of the brains and spinal cord (Hockfelt et al., 1975).
The pain never follows the exact distribution of the cranial nerves (List, 1968). Horton (1956) succesfully desensitized patients with histamine. These patients had headaches that failed to be relieved with either nerve section or alcohol injection of the nerves with release of serotonin for the parasympathetic reactive vasodilatation there will be stimulaton of the VM and SON hypothalami this produces hypersomnia and hypothermia with vasopressin (ADH) secretion and as reported by our patients when they were provoked by the foods that showed high IgE with the RAST method (Figure 4). Many reported feeling sleepy (Hoover, 1985) or feeling cold and some had swelling of the fingers, making their rings feel tight on their fingers.
Cellular protein and glucose levels according to the individuals neutrition, stress, trauma will stimulate the VMH to increase the levels of growth hormones within minutes from 3 nanograms/mm to 50 (Guyton, 1982).
Somatostatin was found to affect pituitary, extra pituitary endocrine, neurocrine and non-endocrine targets. Its presence has been demonstrated in the brain (Hockfelt, 1975).
The onset of the reactions with provocation of our patients took place within

15-30 seconds, indicating the central, humoral and hormonal involvement in these reactions (Jacob, 1982) (Figure 4).

The so-called "migraine personality" (Herberg, 1973) gives an additional indication of the central involvement of the allergic reaction, targeting either the serotogenic or the adrenergic neuronal junctions. This compromises the balance of serotonin and noradrenaline in the hypothalamic and autonomic central control systems (Figure 10).

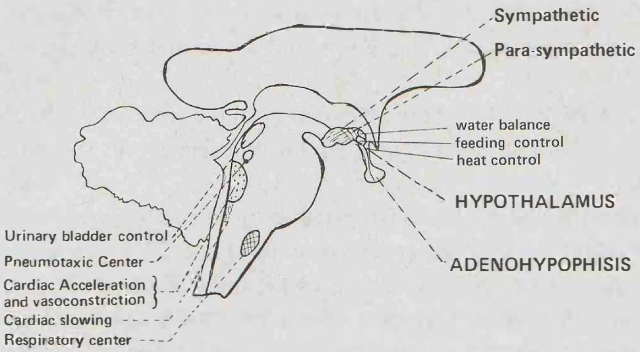


Figure 10. Autonomic control centers of the brain.

The immediate reaction of the antigen-antibody takes place in 15-30 seconds. Wilson and Sharma (1979), with micro-electrode techniques measured a drop of 30 MV of action potentials in the intradermal skin allergic reaction and likewise in the buccal mucosa on food contact. Negative potentials of over 100 MV may be generated locally within 15-30 seconds of application of the diluted antigen and severe central reactions occur; e.g. sleep, headache etc. Further dilutions will clear these symptoms (Willoughby, 1965; Miller, 1972). This reaction can be central as "It is easier for food antigens to gain access to the hypothalamic area due to the absence of a blood brain barrier" (Wilson and Brodie, 1961, Hemings, 1975). Richman et al. (1981) showed that macrophage enriched cells from Peyer's patches were capable of presenting antigen to primed T-cells following antigen feeding in vivo. Also "interruption of the stomach mucosa due to a reaction to one food makes it easier for other molecules to gain entry" (Lessof, 1983). According to Wilson et al. (1980) synthesised nerve cells react to antigen-antibody reaction and will stimulate other nerve cells. Hence we see evidence of distant peripheral disturbances in autonomic control functions, e.g. bronchospasm, tacycardia or bradycardia, tearing of eyes (Figure 4).

The symptoms reported by our patients on provocation with the foods strongly support the central effect that is passed to peripheral target organs by neurotransmitters stimulating their autonomic nerves. This reaction takes place in 15-30 seconds.

The mechanism involved possibly being interference with the intracellular calcium as a result of the propogated stimulus (Tasaka, 1986), or again somatostatin effect on ion transporrt (Guandalini, 1980). And this compromises the cell membrane in the shocked organ and hence the symptom in that organ.

Serotonin metabolises to 5HT in the brain. And we know that Reserpine releases Serotonin (5HT) and depletes arterioles from noradrenaline has a similar effect to those allergic reactions that target the serotonin neural functions, it produces sleep, myosis, bradycardia and peripheral vasodilatation, nasal congestion and rhinorrhea, increases gastric secretion and intestinal motility and headache (Bruyn, 1980).

A close look at all the different pharmacological preparations used for treatment of headaches and migraines supports this etiological theory. These drugs fall into four groups.

1. Those that will prevent the constriction of the smooth muscle of the arterioles and are given for prophylaxes and treatment e.g. Ca Blockers (Meyer et al., 1984; Bonuso, 1986; Tasaka, 1986), or Lithium (Kudrow, 1982).
2. Those that will reverse the state of vasodilatation again site of action, the smooth muscles of the arterioles; e.g. Cortisone (James, 1975; Graham, 1982), Ca Blockers (Bonuso, 1986; Tasaka, 1986), Ergotamine (Bruyn, 1980) and Caffeine (Johnson, 1978), Beta Blockers (Malm, 1977; Kunkel, 1982) prevent vasodilatation and are naturally contraindicated in asthmatics.
3. Those that interfere with the allergic-inflammatory process peripherally, stabilizing the cell membranes, their prostaglandins and calcium transport, e.g. Aspirin, Indomethesin (Bruyn, 1980) and Cortisone. Acitaminophen inhibits prostaglandin synthetase, also exerts an antipyretic action through the hypothalamic temperature control center, like aspirin.
4. Those that are serotonin antagonists, e.g. methylesergide (Friedman, 1963) or serotonin and norepinephrine antagonists in varying degrees e.g. the tricyclic antidepressants e.g. amytriptaline (Richelson, 1984). These drugs will stabilize the hypothalamus and central autonomic control neural junctions and those in turn will stabilize the shocked peripheral organs.

   None of the above drugs are safe for long term treatment and will not give complete control of the headaches.

In our eighty patients the headache varied in intensity and character from excruciating to dull, and pulsating or contineous. Mapping of the areas affected in the head and neck as seen in our patients, is shown in Figure 11. In our sample we had classical migraine, common migraine, muscle contraction headaches, tension headaches, clusters and chronic clusters. All were treated in the same way. Hence we prefer the term "chronic recurrent headaches" that includes all the above named varieties (Sluder, 1927; Vail, 1932; Horton, 1956; Ekbom, 1970; Kudrow, 1982). An interesting finding is that 51.3% of our patients had occipital pain that

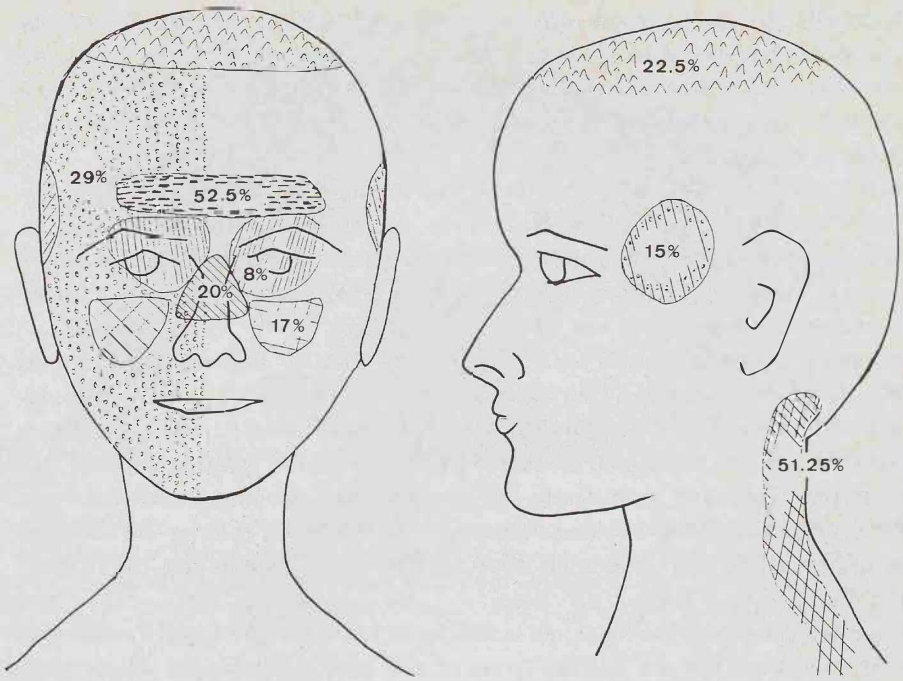


Figure 11. Distribution of the pain in the head and neck areas of the patients reported. Some patients had pain in more than one area, others' pain starts in one area and moves to other areas. 29% had migraines.

was uni- or bilateral, in some it was the only area affected. Repeated relapses were caused by foods and/or infection of the sinuses. Kerr worked out the pain of nasal origin and how it is referred to the occipital area by convergence of the axons of cervical nerves 1, 2 and 3, into the neurons of the nucleus of the fifth nerve (Kerr, 1961, 1972; List, 1968).

The severity of the headache will depend on whether one or two or all the three precipitants take place at the same time. For example in such a predisposed individual, if she/he has a deviated nasal septum, and chronic rhinosinusites she/he will have headaches. If that individual also has inhalent allergy she/he may get clusters in the fall or spring. If she/he also develops food allergy, she/he will develop chronic clusters. Migraines take place as the infection is often one-sided as untreated repeated sinusitis leads to pathologically thickened mucosa that will hamper their drainage. And more so because the allergic reaction is notorious for targetting one spot or organ in the same manner every time that particular antigen-antibody reaction takes place; examples are patients that get tearing from one eye only in ragweed or grass season, those that get the same red itchy skin lesion in the same spot each and every time they eat pork etc. Allergists have

hundreds of such cases and why those reactions stick to that pattern nobody knows yet. Subjects with deviated nasal septa, and those that choose to sleep on one side seem to lateralise their infection due to faulty drainage. Figures 5 and 6 show the incidence of the factors and foods that repeatedly triggered the headaches in these patients.

In this study treatment was directed towards elimination of the cause and prevention of the recurrence. The first step taken was complete control of the infection in the nose and sinuses. Initially this was done with various antibiotics and mucolytics followed by surgical removal of polyps and re-establishing good drainage windows in the sinuses if and when was indicated.

Secondly, complete control of the allergy after accurate diagnosis of its extent. We did this by doing RAST on the inhalents in the area and the foods. Provocation with the foods that showed high IgE with the RAST singled out the foods that induced headaches; the patient then followed a four day rotating diet and this identified yet one or more foods. All the headache inducing foods were completely eliminated for 4-8 weeks period or indefinitely if their re-introduction gives headaches. Immunotherapy was started for both inhalents and foods to gain the phenomena of tolerance.

Figures 5 and 6 show the foods and factors inducing the headaches, it was thought that only foods that are high in tyramine will produce headache (Hanington, 1983; Hyman, 1986). However, our list showed that non-tyramine foods act through their antigen-antibody reaction, releasing histamine etc., and this will cause the immediate reactions (Bruyn, 1980; Lessof, 1983). When the delayed reactions take place incorporating the chemotactic mediators released from the degranulation of the mast cells, these will attract neutrophils, monocytes, lymphocytes and basophils to the targeted area which is the mucosal lining of the nasal turbinates and septum and they will remain swollen thus maintaining the head pain for many hours. The reactive vasodilatations of the initially constricted vascular tissue of the turbinates will be a definite additional factor that increase the bulk of those turbinates (Norman, 1985) (Figure 8). Individuals with headache induced by inhalent allergy used cromolyn nasal, as it prevents antigen induced histamine release from the mast cell.

Thirdly, from the Caldwell view of the X-rays of the sinuses (Figures 1 and 2) plus the clinical examination of the nose, a decision was made to surgically correct impacted turbinates with a deviated nasal septum or spur (Ryan, 1976; Ryan and Kern, 1978; Koch-Henriksen, 1984; Novak, 1984). This is particulary important in cases of "continuous" headaches ("chronic clusters") also the "daily" headache patient. Reducing the bulk of the erectile tissue of the turbinates is believed to be the key for the success of this surgical interference. The nasal turbinates will swell reflexly with increase of pressure in the paranasal sinuses (McAuliffe, 1943), therefore polyps or mucopus had to be surgically cleared when necessary. Hol-

Figure 12a. Biopsy from the nasal mucous membrane of a 36 year old male with chronic vasomotor rhinitis before and after interview engendering nasal hyperfunction and conflict. Biopsy of the left lower turbinate obtained before interview when nasal function was within average limits. Section shows low grade chronic inflammation, relatively undilated vascular and lymphatic channels and compact, quiescent mucous glands. There is no edema of the stroma.

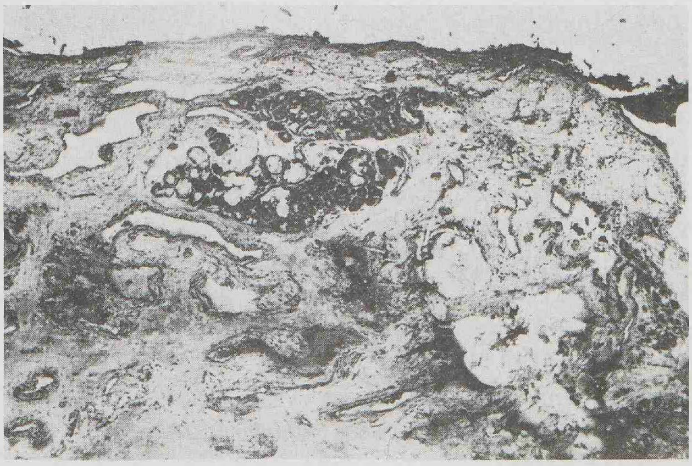


Figure 12b. Biopsy of the right lower turbinate obtained one hour later, at the point of maximal nasal hyperfunction accompanying intense conflict and 'verge of tears'. Section shows prominent, dilated vascular and lymphatic channels, active mucous glands filled with secretion. Lighter value of the stroma indicates edema.
(magnification x200, Masson's Trichome Stain)
(Reprinted with permission from The Nose, T.H. Holmes et al. eds.
Springfield, Illinois: Charles C. Thomas, 1950)

mes (1950) demonstrated engorgement of the nasal mucosa of the turbinates with frustration, humiliation and anxiety (Figure 12a, b). The patients in our sample that had surgical reduction of the turbinates, had relapses from stress only in the presence of infection of the nose and sinuses. Local vasomotor changes in the erectile tissues of the nose as accompaniments of stress, exhaustion, anxiety, sexual excitement have been observed by Fenton (1941). "Ordinarily such variations are not associated with symptoms, but if there be inflammation due to infection these changes enter awareness." (McAuliffe et al., 1943).

CONCLUSION

Eighty patients with chronic recurrent headaches are presented and eight controls. Treatment was directed towards controlling and preventing the allergic inflammatory stimulus, and correcting nasal turbinate and septal impaction. Support from the literature as to the nasal origin of the pain, its propagation and of the action of pharmaceuticals used for headache is presented.

RÉSUMÉ

Cette présentation comprend l'étude de 80 patients souffrant de maux de tête chroniques et répétitifs, y compris de type migraineux. Leurs problèmes ont été traités de la façon suivante:

1. Contrôle des phénomènes infectieux du nez et des sinus.
2. Contrôle et immuno-thérapie des allergènes alimentaires et respiratoires.
3. Correction chirurgicale des cornets hypertrophiés et de la cloison nasale si indiquée.

Ces différentes thérapies ont donné comme resultat une disparition permanente des maux de tête sur une période prolongée de 1 à 35 ans. Le texte qui suit présente une revue de la littérature qui traite de l'origine nasale des différentes formes de céphalées. Le mécanisme délenchant consisterait en une reaction centrale antigènes-anticorps menant à la production d'histamine et de substances appelées neuro-transmettrices. Quand ces substances atteignent la muqueuse nasale, cette dernière devient oedémateuse et déclenche un stimulus douloureux au niveau du cou et de la tête. Il est à noter que l'intensité du phénomène douloureux et plus marquée si la muqueuse nasale était le siège préexistant de phénomènes inflammatoires et de déviation de la cloison. Si les substances neuro-transmettrices ont pour cibles des organes autres que le nez, nous observons un phénomène appellé "symptôme migraineuz non céphalique". Un désequilibre central entre le niveau de serotonine et de noradrénaline est considéré comme étant le facteur responsable du type de personnalité dite "migraineuse" et du malfonctionnement du contrôle autonome central. Nous montrerons que les médicaments utilisés dans le traitement des maux de tête ont pour effet de prevenir ou bien de renverser le mécanisme allergie-inflammation et ses

séquelles: c.à.d. un spasme des muscles lisses des artérioles provoquant une vaso-constriction suivie d'une vaso-dilatation réactionelle.

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