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RHYTHMIC PALATAL MYOCLONUS

WITH A REPORT OF FOUR CASES*

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THE condition variously described as rhythmic palatal myoclonus, clonic spasm of the palate, and velopalatine or palatal nystagmus, is comparatively rare. It shows itself as a continuing, fine rhythmic movement of the soft palate, uvula and faucial pillars, which may be unilateral, but more commonly is bilateral, and is usually associated with a clicking noise audible both to the patient and to other people. The fact that four patients showing this curious phenomenon have been examined in one otolaryngological clinic within three years and that they were younger than the majority of those recorded hitherto, has prompted us to publish the cases and to review the literature.

Historical Review

Although during recent years this condition has received some attention mainly from neurologists, it was well known to a former generation of otolaryngologists. Politzer, in 1862, appears to have been the first to describe these movements (Gallet, 1927). In the first (German) edition of his textbook in 1878, under the heading of neuroses of the sound-conducting apparatus, he describes the condition as clonic spasm of the muscles of the eustachian tube. He states that cases had also been described by Schwartze and Brunner. In the fourth English edition (1902), three further references are given.

Spencer (1886) reported a case of a tumour of the cerebellar vermis

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occurring in a girl of 12 and exhibiting what he described as pharyngeal and laryngeal "nystagmus", i.e. rhythmical movements of the superior pharyngeal constrictor and the intrinsic muscles of the vocal cords but not involving either the soft palate or the pillars of the fauces. Some dozen or so isolated cases were reported during the next few years, notably by Bond (1896), Lack (1898) and Semon (1901).

Grunwald (1903) described the condition as being so rare as to be a curiosity. He also noted that it had occurred in diseases of the medulla, and that contractions of the tubal muscles were apt to produce a noise audible to a second person, but that sometimes it was an occupation-neurosis after excessive voice use in singers, or a tic.

Valentin (1904) reviewed the cases which had been recorded up to that date and came to the conclusion that these movements occurred in people of an emotionally unstable nature following some form of mental or nervous shock. He thought that the movements were a habit spasm, such as can occur in young children as a result of voluntary repetition and which later becomes a tic.

Further detailed reviews were made by Gallet (1927) and Leshin and Stone (1931) who analysed in detail those cases described in which associated movements had been present, and reported one further case of their own. (The majority of these cases were due to vascular accidents, occasionally to syphilis or to neurological disorders such as disseminated sclerosis or as a result of encephalitis.)

Pfeiffer (1919) reported the first traumatic case in which a shrapnel wound occurring in the left occiput produced myoclonic movements together with signs of cerebellar disturbance. Guillain and Mollaret (1931) produced a comprehensive summary of the possible sites for lesions which could be held responsible for the production of the rhythmic myoclonus characteristic of this syndrome. Riley and Brock (1933) described and commented in detail upon three further cases. Two cases with the results of post-mortem findings were described in the same year by Freeman (1933), who also noted that six cases in Leshin and Stone's review of the German and English literature contained necropsy reports. Six further occurrences of the condition were detailed by several speakers at a discussion on the foregoing papers.

During the succeeding years the condition continued to arouse interest, and experimental studies have been made with a view to establishing the anatomical sites of the causative lesion (Weinstein and Bender, 1943). An excellent summary of the knowledge of this condition to date is given by Bender, Nathanson, and Gordon (1952). These authors record a further eight cases, including the operative findings in one, and a detailed description of the post-mortem appearances in another. They also record the effects of intravenous anæsthesia employed experimentally to determine whether this would influence the condition.

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Rhythmic Palatal Myoclonus

Symptomatology

The time and mode of onset of the myoclonus are usually vague and indeterminate. It may even be present without the patient being aware of it. Most authors consider that when such a myoclonus has developed, it will persist unremittingly until death. Other writers, however, note that isolated cases of palatal spasm have been recorded in which movements have ceased following treatment applied to either the ears, nose, or naso-pharynx. In these rare cases there has been some demonstrable local focus of irritation or infection, and the patients themselves have in addition been considered to be of a neurotic temperament. It is interesting to note that one of the earliest cases described in the literature (Boeck, 1867), was considered to be of such a type, and that movements are reported to have ceased following a short course of galvanism applied to the mastoid region.

The movements are involuntary, but may, in some cases, temporarily be stopped by the application of neighbourhood stimuli, such as depression of the base of the tongue with a spatula, or the raising of the velum palati by means of a finger, also voluntary acts such as phonation and deglutition usually cause temporary inhibition. Often the movements are accompanied synchronously by a clicking sound in the patient's ear; indeed this may be the presenting symptom, and it is due to simultaneous contractions of the musculature around the eustachian orifice. Such sounds may be of considerable intensity and are usually likened to the irregular ticking of a watch or to the snapping of one thumbnail against another. Sometimes they may be clearly heard as well by other people at a distance of several feet.

Usually the myoclonus continues unremittingly during sleep, and is entirely uninfluenced by either general or local anæsthesia.

The average rate of these abnormal movements tends to be within the range of 140 to 180 oscillations per minute. Pegler (1903), however, described a curious case in which initially the rate was as low as 10 beats per minute, and which rose steadily during the period of observation to as much as 240 beats per minute. In this case no diagnosis as to the ætiology appears to have been made, but it would seem possible that it was first observed early after the occurrence of the primary lesion whatever that may have been. As a rule, however, the rate tends to remain remarkably unaltered over long periods. Occasionally sudden increases of rapidity may be observed to override what may be termed the basic rhythm of the oscillation. The rate of the movements usually bears no relation to either respirations or pulse, although cases have occurred in which synchrony with the pulse has been noted. Respiration has, however, occasionally influenced the movements. Freystadtl (1928) described a man aged 58 with continuous rhythmic contractions of palate, pharynx and larynx. The amplitude of the excursions was heightened by psychic influences, to such an extent that at times they became arrhythmic.

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Isolated bilateral myoclonus of palate and pharynx is by far the most frequently reported finding. Leshin and Stone (1931) stated that over 100 cases had been reported in the literature. Cases of unilateral palatal movements have been described much more rarely, and Bender, Nathanson, and Gordon (1952) could only find records of six such cases.

Associated myoclonic involvement of other muscle groups can occur, although this again is much rarer, Leshin and Stone having found only 26 reported cases.

The whole larynx may be involved, and there may be rhythmic movements visible in the floor of the mouth, face and neck, and even the diaphragm (Ernst, 1907; Thibonneau, 1942-3), and epigastrium (Alajounine, Thurel, and Wolfram, 1944), may exhibit oscillations. In some instances movements may originally be confined to one muscle group, gradually spreading to involve others. Ocular nystagmus has also occurred.

Case Reports

CASE 1.

G.F. No. 29771. Aged 21. Student.

25.5.50. Referred on account of attacks of dizziness which were vague in type and which appeared mainly when he was studying. These were not severe enough to prevent his working, but made him feel irritable and depressed. On two occasions he had had brief attacks of unconsciousness. After the first of these he had noticed frequent clicking noises in both ears which at times developed into a roaring noise with a feeling of pressure in his ears. Functional enquiry was otherwise negative except that he had had "influenza" in the previous term.

On examination. Both tympanic membranes were substantially normal. Hearing tests including an audiogram were normal. Nose and throat were entirely healthy, but irregular bilateral palatal movements could be seen. An irregular faint click could be heard through an auscultation tube placed in each ear in turn, and this was present whether the patient breathed through his nose or through his mouth, but could be interrupted by forcible depression of the tongue. Eustachian catheterization showed both tubes clear and the procedure failed to influence the clicking. Skiagrams of the nasal sinuses and soft tissues of the post-nasal space were clear. Examination of the central nervous system by Dr. Ritchie Russell was negative.

On 6.6.52 the appearances were unchanged, the clicking noises still continuing. Since the nature of his noises had been explained to him he had ceased to worry about them. He had been concerned before his first attendance because he could hear noises in his head and thought that this must indicate some serious cerebral disorder. When he had been told that other people could hear his noises, he was very relieved. His symptoms had become more prominent for a time when he was just about to take his final examination. Three weeks later, he returned with the observation that his examinations were over, and that during the course of a Commemoration Ball the noises had vanished.

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The irregular palatal movements and the clicks were, however, found to be quite unaltered.

The effect of infiltration of the soft palate with Nupercaine as for the carrying out of tonsillectomy under local anæsthesia, was investigated. This failed to abolish the clicking, which still remained audible although the movements became less prominent. This damping effect was thought to be due to the local swelling brought about by the infiltration. He now (March, 1953) has a teaching post in which he is happy, and is able to forget about his clicks.

CASE 2.

M.B. No. 40528. Aged 9.

23.5.52. This small girl was referred with the diagnosis "ticking larynx".

Her history was that three months previously she had wakened crying in the night and complaining of a ticking noise in her ear. At this time she was not ill nor was there any illness among the members of her family. Mumps, whooping cough and other infectious illnesses had been prevalent in the village where she lived.

On examination. Both ears showed a little wax but otherwise the appearances were normal. A clicking noise was audible at a distance of a foot or so from the child, and this could be heard more loudly *viâ* an auscultation tube inserted into the ears. Its rate was 116 per minute. The noise was louder on the left side than on the right, and was associated with respiration to the extent that it disappeared on breath-holding and on deep inspiration. There was a bilateral asymmetrical clonic contraction of the soft palate synchronous with the clicking noise. The clonus could be interrupted by stretching the palate by firm depression of the base of the tongue with a spatula. Indirect laryngoscopy showed the larynx to be unaffected. The only other abnormal clinical finding was a slight fullness in the mastoid grooves which was thought to be due to calcified lymph glands.

On 9.6.52 electromyography was carried out. At the time of this examination the audible clicking varied between 120 and 130 oscillations per minute, and the uvula could be seen moving at this rate. On insertion of the bipolar electrode, the movements ceased completely and the record showed complete silence with a normal motor interference apparent on swallowing. These findings suggested that the lesion was not in the lower motor neurone.

CASE 3.

H.J. Aged 34. Housewife.

July, 1952. This case was referred to us through the courtesy of Dr. Tow of the Department of Neurosurgery, with a diagnosis of "clicking ears".

In 1944 she had attended an E.N.T. Department in London on account of the "clicking ear" and had been treated by eustachian catheterization without any benefit. The noises appeared louder just before her monthly periods, and for the last three years had been much louder than previously, and in addition there had been at times bouts of extreme loudness. Her symptoms were more prominent on the left side, but during the past few months she had been aware of a sound which she described as being "a sort of echo" occurring in the right ear as well as the left.

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In addition she had complained of pain, which was particularly noticeable at times when she stopped to notice the ticking. This was a slight shooting pain superimposed upon a deeper throbbing sensation, and was accentuated by cold, worry and excitement. Examination showed the presence of a myoclonus affecting the mylohyoids, thyrohyoids, and the palatoglossi on both sides. There was an audible click on auscultation in the left ear and a fainter one in the right ear, synchronous with the muscular contractions. In this case there was obviously a marked psychological overlay, but the physical condition was unmistakable.

CASE 4.

G.C. No. 43733. Aged 6.

Seen on 23.1.53.

The previous night when in bed he called out to his parents that he had what he described as a "funny" feeling in his throat. There was no pain, and he was not distressed or alarmed by the sensation. His father opened his mouth and observed the uvula to be oscillating in a rhythmic fashion. Later the child, who was a good witness, said he thought the clicks started about a week before Christmas.

For the previous few days the child had suffered from coryza and hoarseness. He had been in contact with mumps, but he himself had had mumps 12 months previously. He had also had measles and chickenpox.

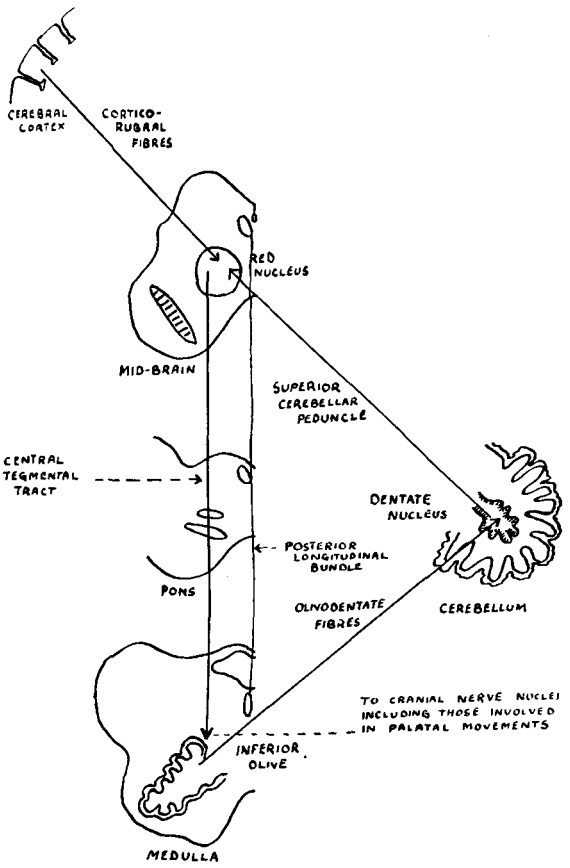
On examination. The child was above the average intelligence for his age, and was left handed. There was an audible, regular clicking sound when his mouth was open, and when first seen this was at a rate of 140 oscillations per minute. The whole soft palate and the uvula were showing a regular clonic movement which was not synchronous with the pulse. The movement was temporarily abolished by any strong neighbourhood stimulus such as firm depression of the base of the tongue with a spatula. It was re-established immediately on withdrawal of the stimulus. The sound could not be heard *viâ* an aural auscultation tube. Anæsthetization of the soft palate with 10 per cent. cocaine failed to influence the movements. The pharynx, larynx and nose showed signs of a recent upper respiratory infection, but the vocal cords moved normally. There were palpably enlarged, non-tender, cervical lymph nodes on both sides. A general physical and neurological examination showed no further abnormality. He was admitted for further investigations.

Sinus X-rays showed mucosal thickening in both maxillary antra and impaired translucency in the ethmoids and frontals, such as might be found after a recent upper respiratory infection. Chest X-ray was negative. Nasal and nasopharyngeal swabs produced pure cultures of a penicillin-sensitive *Staphylococcus aureus*. Tonsil swabs showed Gram positive cocci on Gram staining and were negative when cultured. Lumbar puncture and full blood count showed no abnormality. An electroencephalogram showed "Occasional slow spikes in the right temporo-parietal lead which on hyperpnoea became more frequent. The abnormality was not regarded as gross when his age was considered, but the authenticity of the right Sylvian fissure focus was thought to have added weight since this is the correct location for palatal epilepsy."

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It was not considered that electromyography would give any useful information (see Case 2). A Mantoux test, W.R. and Kahn were all negative.

An examination by cine-radiography showed the excursion of the soft palate to be about 1 mm.



Possible sites of lesions producing Palatal Myoclonus. This diagram illustrates the triangular area described by Guillain and Mollaret (1931), together with the suggestion that cortico-rubral fibres and the cerebral cortex itself may sometimes be involved. (Gray, 1942, states that afferent fibres connecting the red nucleus with the cortex have been variously ascribed to the frontal, temporal and parietal lobes.)

On the assumption that the condition might have been due to a virus infection a course of oral aureomycin, 150 mgms. 6-hourly was given for 6 days. There was no alteration in the palatal movements, but it was noted that they did not produce any audible sound during sleep. Sometimes the rhythm was synchronous with the pulse rate, and on one occasion after vigorous exertion it rose to 156 beats per minute. The only demonstrable effect of the aureomycin was to clear up the upper respiratory infection.

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Comment

In the four cases described above myoclonus developed before the age of 30, and in two of them before the age of 10 years. Most of the cases of palatal myoclonus previously reported in the literature have occurred in elderly subjects. Riley and Brock (1933) found 70 per cent. of the patients reviewed by them to fall into the age group 41 to 70 years, and they could find none reported as occurring in the first decade of life. Of the eight cases reported by Bender, Nathanson and Gordon (1952), six were in the sixth decade, and the youngest, aged 22, died of a reticulum-celled sarcoma in the mid-thoracic region of the spine. He had presented with signs of acute encephalitis, and post-mortem showed lesions in the right dentate nucleus and restiform body, and in the left inferior olivary nucleus.

The occurrence of reported instances of palatal myoclonus in children, is extremely rare. Spencer's (1886) description of pharyngeal and laryngeal "nystagmus" in a girl of twelve, sometimes mistakenly quoted as being the first available record of this condition, did not, in fact, as Spencer himself specifically states, exhibit any movement of either soft palate or faucial pillars. Jewell (1923) described a child with a spasm of the soft palate in whom the "click" could be heard two yards away, and which disappeared after removal of the adenoids. It is not stated, however, whether visible myoclonus continued to be present after the operation.

Another unusual feature found in one of the children was some correlation at times between the rate of the palatal excursions and the pulse rate, a finding which has been noted in only a few cases (Boeck, 1867), and which some authors even state does not occur (Bender, Nathanson, and Gordon, 1952). We are inclined to regard our findings as a coincidence, although it must be remembered that the motor connections of the palate and heart muscle in the brainstem are very close.

It is interesting to observe that, although most writers have accepted the localization of the causative lesions in this condition to be in the cerebellum and in the mesencephalometencephalic areas of the brainstem, the electro-encephalogram carried out in one of our cases suggested the possibility of damage to the right temporo-parietal cortex, the vicinity from which, it is stated, palatal epilepsy can originate. It is instructive to compare these findings with those in Jacobson and Gorman's case (1949). In that instance the electro-encephalogram demonstrated a focus of electrical abnormality in the right occipito-parietal region, and it was known that there were traumatic lesions in this area as well as in the brainstem and cerebellum on the opposite side.

Two of our cases showed involvement of muscles other than those of the palate. This association has been reported much less commonly than palatal myoclonus alone.

The clonic contractions in all four cases have shown no sign of abatement during the time they have been observed.

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Rhythmic Palatal Myoclonus

Pathological Anatomy

The earliest discoverable post-mortem examinations in cases of palatal myoclonus were two reported by Klein (1907). The first was a man aged 53, who presented with a left-sided palatal myoclonus associated with similar movements of the musculature of the floor of the mouth and of the diaphragm at a rate varying from 130-160 oscillations per minute. He later committed suicide and at autopsy the left internal capsule presented a small calcified body 12 mms. in diameter, a lesion of the left upper cerebellar hemisphere involving the dentate nucleus and another lesion in the right side of the cerebellum. The superior and middle cerebellar peduncles and the restiform body were degenerated. The contralateral red nucleus and the left cerebello-olivary tract were atrophic and there was an increase of glia in the region of the olive.

Klein's second case was that of an arteriosclerotic female of 52 who showed lesions in pons, red nucleus, right cerebello-olivary tract, and left olive.

Graeffner (1910) reported two cases. One male aged 70, had pseudo-bulbar paralysis and presented persistent clonus of uvula and larynx. At autopsy lesions involving the right cerebral hemisphere, pons, brain-stem and cerebellum were reported.

Klein (1918) reported a further case of a man aged 42 who had suffered repeated attacks of apoplexy and in whom the post-mortem examination included the finding of a hæmorrhage in the dentate nucleus. Wilson (1928) described a case of palatal nystagmus which was associated with a tumour of the corpora quadrigemina. Foix (1926) carried out a careful study of palatal myoclonus and suggested that the basic pathology of this condition was a lesion in the pons involving the central tegmental fasciculus. In all four cases which came to post-mortem this tract together with the reticulum of the metencephalic tegmentum and the olivary nuclei showed involvement. Foix also found lesions present in the dentate nucleus.

Van Bogaert and Bertrand (1928) conducted close anatomical studies and found the dentate and olivary nuclei to be frequently affected, and they suggested that for myoclonic contractions to be produced at all, it was essential for either the dentate nucleus or the inferior olivary nucleus to be involved. They propounded the theory that the myoclonus was produced as a result of a functional or anatomical release of infranuclear motor centres from the influence of higher motor centres.

This conception is very similar to that suggested by Ramsay Hunt (1933). He sought to explain cases of palatal tremor on the basis of a theory of central inhibition. He postulated the occurrence of a cell of the Golgi type which possessed a specific inhibitory influence, and which appears throughout the central nervous system. He compared the production of myoclonus with the development of chorea, and suggested that in such a disorder as encephalitis, a release of mobility at the spinal level

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resulted in myoclonus, whilst striatal lesions caused chorea. Although recognizing that such a condition as palatal nystagmus could be, and often was, due to a multiple and varied ætiology, he visualized the development of a rhythmic tremor occurring as a result of a localized encephalitic involvement of the group of these small inhibitory cells in relation to the glossopharyngeal mechanism.

Detailed correlation of the clinical, anatomical, and pathological findings was carried out by Guillain and Mollaret (1931) and Guillain, Mollaret and Bertrand (1933). These investigators described a triangular area within the midbrain lying between the red nucleus, inferior olivary and accessory nuclei and the contralateral dentate nucleus.

At post-mortem, structures lying within these boundaries, which included the central tegmental tract, the posterior longitudinal bundle, the superior cerebellar peduncle, and olivo-dentate fibres, were consistently found to be involved in some form of pathological process. They therefore held that lesions affecting these structures were responsible for the characteristic rhythmic myoclonus. The oscillatory movements were produced as a result of imbalance of the forces acting between the various nuclei and in their inter-linking pathways.

Freeman (1933a) brought forward the suggestion that the myoclonic movements were related to yawning, being part of the facio-respiratory synkesis elaborated by Wilson (1929). Later, Freeman (1933b) gave a description of a further case he had observed in which a patient suffering from illuminating gas poisoning exhibited myoclonic twitchings of his uvula on each occasion he opened his mouth, succeeded by an unmistakable yawn. The possibility that these abnormal muscular movements may be a pathological modification of yawning is also noted by Jacobson and Gorman (1949).

The similarity in character, rate and rhythm of the contractions occurring in palatal myoclonus, with the sucking movements of nurslings and the fact that in both types of movements similar muscle groups are involved, was remarked upon by Kreindler (1939).

It is therefore interesting to find that during the course of their experimental work on monkeys, Weinstein and Bender (1943) by stimulating similar areas in the brainstem to those in which post-mortem lesions have been found in cases known to have had palatal myoclonus during life, were able to initiate a variety of movements including sucking, swallowing, facio-respiratory complexes, and, when stimulating an area dorsal to the inferior olivary nuclei, rhythmic palatal contractions.

Ætiology

The majority of cases previously reported in the literature showed evidence of organic disease of the central nervous system. It has most

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commonly been observed in patients of advancing years in association with arteriosclerotic and hypertensive vascular lesions. Additional ætiological agents which have been incriminated include trauma, epidemic cerebrospinal meningitis, chronic epidemic encephalitis, syphilis, disseminated sclerosis, carbon monoxide poisoning, new growths, aneurysm of the vertebral artery, acute rheumatism (McKenzie, 1923), electric shock (Baruk, Osianik, and Borenstein, 1945), and hysteria. In some cases no obvious causative factor has been found. Cases 1, 2, and 4, of our series occurred in December, January and February, times of the year when virus infections are rife. It might not be unreasonable to suggest that possibly these, like other cases of "idiopathic" type, had a viral invasion of the brainstem in the region of the inferior olive.

Summary and Conclusions

A clinical record of four cases of rhythmic palatal myoclonus is given. These present an unusual age incidence for this condition and exhibit several uncommon features.

A review of the relevant literature is given and a consideration of the pathological anatomy suggests that there is usually a lesion of the brainstem, more particularly in the region of the inferior olive.

Despite the previously reported preponderance of this disorder in the elderly, palatal myoclonus may occur at any age. It may be localized to the soft palate or there may be more widespread associated muscular contractions.

The ætiology is frequently obscure and no form of treatment yet attempted is of any value in causing cessation of these rhythmic movements.

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