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Effects of hormonal changes on the human voice: a review



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Abstract

The human voice is commonly affected by hormonal changes, both in physiological and pathological conditions. Voice changes were reported in a number of endocrine disorders, and dysphonia may be the initial sign of these disorders. A number of studies documented endocrine-related voice changes utilizing both subjective and objective voice assessment tools. The objective of the present review is to draw the phoniatrician's attention to the wide variety of typical changes, as well as the potential for hormonal imbalances that may affect the voice, in order to identify them promptly and generate proper treatment and referrals.

Keywords Voice, Vocal fold, Endocrine disorders, Thyroid, Pituitary

Background

The endocrine system is composed of glands that release hormones into the bloodstream. These hormones are carried via the blood to their target cells. The hormone's interaction with its receptor (located on the cell surface) sets off a series of biochemical events in the target cell that ultimately modify the cell's activity or function [1]. The major endocrine glands are illustrated in Fig. 1 and include:

- The hypothalamus (which releases Corticotropinreleasing hormone, Gonadotropin-releasing hormone, Thyrotropin-releasing hormone, Growth hormonereleasing hormone, Somatostatin, and Dopamine).
- The pituitary gland (the anterior pituitary releases Adrenocorticotropic hormone, Luteinizing hormone, Follicle-stimulating hormone, Thyroid-stimulating hormone, Growth hormone, and Prolactin, whereas the posterior pituitary releases Vasopressin and Oxytocin).

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- The adrenal cortex (which releases Cortisol and Aldosterone).
- The testes (which release Testosterone).
- The ovaries (which release Oestrogen and Progesterone).
- The thyroid gland (which releases Thyroid hormones and Calcitonin).
- The parathyroid glands (which release Parathyroid hormone).
- The pancreas (which releases Insulin and Glucagon) [1].

The human voice is very sensitive to endocrine changes, with various endocrine changes affecting the voice throughout the normal human life cycle [2]. The detection of androgen, oestrogen, and progesterone, in addition to thyroid hormone receptors in the human vocal fold (VF), may provide evidence to the hypothesis that variations in vocal quality are caused by hormonal influences [3–5].

The phoniatrician must be familiar with the wide variety of typical changes, as well as the potential for hormonal imbalances that may affect the voice, in order to identify them promptly and generate proper treatment and referrals [2].



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Fig. 1 Schematic representation of the location of the major hormone-producing (i.e., endocrine) organs in the body. (For the purposes of illustration, both male and female endocrine organs are presented here) [1]

Evaluation of voice disorders

It is ideal to obtain a thorough case history and conduct the following battery of tests when examining individuals with voice abnormalities in order to characterize the disorder's impact on all relevant mechanisms and dimensions: auditory perceptual assessment, laryngeal endoscopy, acoustics, aerodynamics, and patients' selfperception of how voice issues affect their daily function are among the other factors to consider [6–10].

As stated by Kotby [11], the voice evaluation protocol proceeds through three levels:

 Elementary procedures: which encompasses thorough history-taking (including vocal habits, occupation, onset, course, and duration of dysphonia), and auditory perceptual assessment (APA) of the voice with ENT and laryngeal inspection. Subjective voice assessment could also be achieved by using questionnaires such as the voice handicap index [12].

- (2) Clinical aids: this level includes videolaryngostroboscopy using either a rigid 90° laryngoscope or a flexible nasolaryngoscope. Stroboscopic examination allows the observation of the mucosal wave, amplitude of VF vibration, symmetry and periodicity, glottis closure, and ventricular folds.
 - High-fidelity voice recordings in a sound-treated room should be used for APA's documentation in order to reduce background noise. The recorded material is rated using the modified GRBAS "Grade, Roughness, Breathiness, Asthenia, Strain" scale with 4 grades from 0 "normal" to 3 "severe dysphonia" for describing the grade and character of dysphonia.

(3) Additional measures: this includes acoustic analysis (e.g., shimmer, jitter, and harmonic to noise "H/N" ratio) and aerodynamic analysis (e.g., maximum phonation time "MPT") of voice.

Effects of sex hormones on voice throughout life

Voice is regarded as a crucial secondary sexual characteristic that gives a unique impression to a person's character and personality [3]. The voice alters in response to changing sex hormones throughout life. The first and most noticeable changes occur for both sexes at puberty, when hormonal changes lead the male voice to drop by even one octave and the female voice to drop by one-third [2, 13]. At puberty, the dimensions of the male's vocal tract and larynx increase, and VFs become thicker and longer, lowering the fundamental frequency of the voice. In contrast, the female's vocal tract and VFs are shorter, resulting in their brighter voice timbre and higher voice pitch [14].

Males experience a more pronounced initial pubertal voice change but then have relatively stable circulating levels of sex hormones across their life span and undergo fewer subsequent voice changes. In females, cyclic voice and laryngeal changes occur with each menstrual cycle, and a second permanent change occurs at menopause [2].

Voice changes in the premenstrual days are common and known as dysphonia premenstrualis. The most frequent voice symptom reported by female singers was difficulty singing high notes [15]. Other commonly reported vocal symptoms were vocal fatigue, changes in voice quality, and reduced vocal range [2]. Female opera singers were excused from performance in the premenstrual and early menstrual days, a practice named "grace days" [16].

In the premenstrual period, oestrogen and progesterone work together to relax smooth muscles, which induces venodilatation. Moreover, polysaccharides break down into smaller molecules in the vocal folds (VFs) and bind water, enhancing fluid retention. These changes result in vocal fold congestion and edema [2]. Progesterone also causes mucosal dryness by increasing epithelial desquamation and decreasing mucous secretions at the vocal fold edges [13].

Some studies provided evidence of the direct effect of sex hormones on the VFs' tissue. Abitbol et al. [13] demonstrated that a woman's normal menstrual cycle resulted in noticeable alterations in both superficial laryngeal and vaginal smears. Surprisingly, at each phase of the cycle, the smears from the cervix and larynx were indistinguishable. This work has been supported by the localization of oestrogen, progesterone, and androgen receptors in both the mucosa and deeper tissues of the larynx [2, 17, 18]. Using narrow band imaging, Shoffel-Havakuk et al. [19] demonstrated increased laryngeal vascular congestion during the premenstrual period consistent with the progesterone peak (Fig. 2).

On the other hand, Chae et al. [20] and Ryan & Kenny [21] suggested the role of secondary effects of premenstrual syndrome (PMS)-related mood variations and abdominal spasms on voice. Both could result in laryngeal muscle tension or a decrease in respiratory effort, signifying that females suffering from PMS have a greater tendency for premenstrual dysphonia [19].

It is thought that the greater susceptibility of females to microvascular VF lesions, including ectasias and varices, is related to the female hormonal cycle and hormonal variations. Care should be taken as the presence of these microvascular lesions increases the risk for VF haemorrhage [22].

Pregnancy-related voice changes may be similar to those encountered premenstrually [23]. The main vocal complaints were breathiness and vocal fatigue, especially in the third trimester [24–26]. During pregnancy, the oestrogen and progesterone levels are significantly increased [27]. This leads to variable degrees of edema and congestion of the VFs' mucosa, as detected by laryngoscopy [28] and high-speed digital imaging [27]. Aggravated laryngopharyngeal reflux could also play a role [26]. Pregnancy-induced rhinitis affects about 22% of females [29], and negatively affects the voice due to the dehydrated VF mucosa subsequent to mouth breathing [30].

A significant decrease in MPT during the third trimester of pregnancy was observed in a number of studies [26, 27, 31, 32]. As explained by Cassiraga et al. [31], the diaphragm moves up during the third trimester, causing upper chest breathing. This results in poor respiratory support and a reduction of MPT. Ulkumen et al. [33] compared certain voice parameters in the third trimester with the third month postpartum and found a significant increase in MPT in the third month postpartum.

In the majority of studies of gestational voice changes [26, 32, 34], no significant changes were reported in acoustic and aerodynamic parameters during the first and second trimesters of pregnancy. On the other hand, Li and Xu [28] mentioned that the fundamental frequency of the voice is significantly decreased in some pregnant women, predominantly during the second trimester, and particularly with male fetuses. The authors found that, at 2-year follow-up, the pitch increased in most cases but did not return to normal levels and recommended further study to elucidate the cause.

In light of these findings, it would be reasonable to educate pregnant women about poor vocal habits and vocal hygiene, mainly in the third trimester. Breathing exercises



Fig. 2 Different events of the menstrual cycle and corresponding laryngeal changes as seen by narrow band imaging [19]

and professional voice support during pregnancy would be of special importance to professional voice users, particularly singers [33].

After menopause, the relative increase of androgens together with the marked drop in oestrogen and progesterone levels may cause vocal fold thickening with a slight decrease in the vocal pitch [35]. Other observable post-menopausal voice alterations include vocal fatigue, reduced vocal range, and failure to reach high notes. Professional vocalists are more attentive to these changes [36]. Most vocal abnormalities in postmenopausal females have been proven to be reversed by hormone replacement therapy (HRT) [37]. The studies of D'haeseleer et al. [38] and Hamdan et al. [39] indicated that menopausal women on HRT have a higher habitual pitch compared to those not on HRT, demonstrating the counter effect of oestrogen replacement on the VFs. It is well known that oestrogen markedly increases the glandular secretions above and below the VF edges, enhancing mucosal viscosity. It also improves VF capillary permeability, promoting better tissue oxygenation [13]. Proper counseling and cautious consideration of the risks and benefits of HRT are strongly recommended for professional voice users approaching menopause.

In males, aging is associated with a progressive thinning of the vocal folds. After the age of 30, a decline in serum testosterone levels occurs by 1% per year. The resultant sarcopenia in aged males over 65 years affects the vocalis muscle, leading to VF bowing and reduced glottic closure with perceived breathiness [40, 41]. Additionally, elevated fundamental frequency is perceived with vocal feminization [35].

Voice changes in reproductive disorders

Polycystic Ovarian Syndrome (PCOS) is the most prevalent endocrinopathy among middle-aged women [42]. Anovulation and hyperandrogenism can lead to marked virilization. Although hirsutism, oily skin, and acne are most common, androgen-mediated vocal changes can also occur [2]. Nygren et al. [43] hypothesized that high levels of androgens can result in hypertrophy of the thyroarytenoid muscle, which coincides with a lowered vocal pitch. Hannoun et al. [44] reported increased incidence of throat clearing, deepening of voice, and decreased maximum phonation time among patients with PCOS. In a group of patients with PCOS, laryngeal abnormalities such as impaired VF vibration, incomplete glottic closure configuration, and supraglottic hyperfunction (reflecting a deviant muscle tension pattern) were found to be significantly more common, according to a study by Aydin et al. [45].

Although psychogenic causes are more common, hypogonadism in males (resulting from anomalies of the gonads) can lead to puberphonia (a.k.a. incomplete mutation or mutational falsetto) [46, 47]. Failure to adopt the lower-pitched voice of adulthood is the defining feature of puberphonia. In addition to an unusually high pitch, common signs include a breathy, weak voice, reduced loudness, and pitch breaks [48, 49].

Exogenous androgens (e.g., anabolic steroids) should be avoided in females if there are any appropriate therapeutic alternatives. In clinical practice, these drugs are most frequently used for treating endometriosis or (illicitly) to improve athletic performance [50, 51], as well as in female to male transgender patients [2]. Exogenous androgens produce instability of the female voice and increased muscle mass of the VFs, leading to a drop in fundamental voice frequency and deep voice [47, 50, 52]. These changes are often irreversible, and can occur within weeks of the initiation of androgens [2].

Androgenic progesterone-containing oral contraceptives were reported to change voice character and range deleteriously after only a few months of starting therapy and may permanently masculinize the voice [53, 54]. Modern formulations of oral contraceptives use much lower hormone doses, and voice changes have not been reported [55]. In fact, current oral contraceptive formulations tend to reduce hormonal fluctuations throughout the menstrual cycle, stabilizing the singing voice [56].

Voice changes in thyroid and parathyroid gland disorders

Thyroid gland disorders can result in laryngeal dysfunction either through hormonal effects or due to local effects on the recurrent laryngeal nerves (RLNs) or surgery [2].

A) Voice changes related to hormonal disturbances

According to McIvor et al. [57], up to one-third of thyroid disease patients present with dysphonia. Thyroid hormone receptors, TR alpha and beta, have been detected in the fibrous lamina propria, cartilage, and glands of the human larynx. These receptors may contribute to the voice changes associated with thyroid dysfunction [4].

Hypothyroidism is a well-known cause of voice problems [36]. Chronic autoimmune thyroiditis, "also known as Hashimoto's disease", is the most frequent cause of primary hypothyroidism in well-developed countries (without iodine deficiency). Other less common causes are iodine deficiency, thyroidectomy, or radioiodine treatment [58]. The clinical manifestations of hypothyroidism include weight gain, persistent fatigue, cold intolerance, hair loss, skin dryness, menstrual disturbances, voice alterations, and myxedema [59].

Up to 80% of patients with hypothyroidism may have voice complaints, with the frequency of voice problems increasing with more severe hypothyroidism [60]. The voice change is sometimes the main complaint of the patient [47]. Dysphonia, vocal fatigue, softening of the voice, reduced voice range, and globus sensation may be observed even in mild hypothyroidism. The mechanism of voice changes associated with hypothyroidism is not completely understood, especially in cases of mild hypothyroidism [2]. Ritter [61] demonstrated an increased level of acid mucopolysaccharides submucosally in the vocal folds in hypothyroidism. These excess mucopolysaccharides most likely act to increase the osmolality of the lamina propria, increasing its fluid content. This results in increased vocal fold mass and decreased vibration, leading to a deep irregular voice [47]. In some cases, changes similar to Reinke's edema may be apparent [2].

In severe hypothyroidism with myxedema, these changes are more intense and may be associated with paresis of the VFs due to the thyroid gland expansion, cricothyroid muscle oedema, and vagus nerve edema [36]. Moreover, hypothyroidism may influence voice function by affecting the breathing control system, diaphragmatic muscle function, or goitre-induced obstruction of the upper airway [62]. The voice alterations reverse completely within three to six months after achieving euthyroidism [36]. As shown by Birkent et al. [63], the voice fundamental frequency of 24 hypothyroid women treated with thyroid hormone replacement therapy and brought into an euthyroid state significantly improved from a pretreatment value of "223.48 \pm 36.10" Hz to "237.64 \pm 38.31" Hz.

Thyrotoxicosis refers to the clinical condition of excessive circulating thyroid hormones, regardless of the source, while hyperthyroidism results from increased thyroid hormone production and secretion from the thyroid gland. The commonest cause of hyperthyroidism (thyrotoxicosis with hyperthyroidism) is Graves' disease, followed by toxic nodular goitre. Thyrotoxicosis (without hyperthyroidism) is most commonly caused by excess intake of the thyroid hormone medication or results from the release of preformed thyroid hormones due to thyroiditis [64]. The clinical manifestations of thyrotoxicosis comprise palpitations, fatigue, tremors, increased sweating, heat intolerance, nervousness, and weight loss [64, 65]. Mild hyperthyroidism usually does not cause voice problems. In more severe cases, muscle weakness, dehydration, and tremor all contribute to voice changes. Physiologic alterations in the laryngeal structure could also take place [2]. The most commonly reported voice changes include tremulous, breathy voice, and reduced loudness [66]. The general weakness and fatigue due to hyperthyroidism affect respiratory and laryngeal muscles, causing asthenic voice with a low H/N ratio and reduced MPT [67, 68]. The vocal behavior may reflect the anxiety element of the disease [47]. Kovacic [67] also reported that female patients with hyperthyroidism demonstrated reduced fundamental frequency and deep voice.

Voice dysfunctions in parathyroid gland diseases are caused by a general effect. Hypocalcaemia in hypoparathyroidism results in laryngospasm and other symptoms of neuromuscular irritability. On the other hand, hypercalcaemia in hyperparathyroidism leads to muscle weakness and easy fatigability [69, 70].

B) Voice changes related to local effects or surgery

The close anatomic relationship between the thyroid gland and the recurrent and superior laryngeal nerves places these nerves at risk when the thyroid gland is involved with structural or inflammatory disorders. Structural thyroid disorders, including locally infiltrating thyroid carcinoma and huge compressive goiter may affect the voice by stretching the nerves, causing vocal fold paresis or paralysis, or by invasion or compression of the larynx or trachea. Large thyroid masses can also impact voice by impairing vertical laryngeal motion. Neuropraxia of the recurrent or superior laryngeal nerves can result from inflammatory conditions of the thyroid gland, such as Hashimoto's thyroiditis [2, 71].

A hyperplastic parathyroid gland as well as a parathyroid carcinoma can lead to vocal fold paralysis due to recurrent laryngeal nerve involvement [72, 73].

Surgical Considerations

Recurrent Laryngeal Nerve (RLN) Paralysis and Paresis

Recurrent laryngeal nerve paralysis and paresis is the most feared complication of thyroid surgery, and may be unilateral or bilateral. However, such complication rarely occurs when the surgery is performed with nerve monitoring by an experienced thyroid surgeon [74, 75].

Unilateral vocal fold paralysis (UVFP) results in breathy voice quality, diplophonia, reduced loudness, short phonation duration, and a restricted pitch range [76]. Supraglottic hyperfunction is common, leading to a low-pitched, irregular voice [77].

Laryngoscopic examination in UVFP might reveal shortened and bowed VF [78]. Slight VF adduction on phonatory effort might be caused by the action of interarytenoid muscle still innervated partially from the contralateral nerve or by the intact cricothyroid muscle [79]. Arytenoid cartilage prolapse suggests intense denervation with loss of muscular support [80]. In longstanding UVFP with supraglottic hyperfunction, maneuvers such as humming permit a more in-depth evaluation of the glottic closure by causing the ventricular folds to relax [81]. On stroboscopic examination, the paralyzed VF shows wide undulating amplitudes "like the fluttering of a flag in the wind" (though amplitude could be reduced) and a loss of mucosal waves. Phase and amplitude asymmetry is obvious between the normal and paralyzed side with incomplete glottic closure [82].

External Branch of the Superior Laryngeal Nerve (EBSLN) Paralysis and Paresis

EBSLN injury causes a low pitched, weak voice that easily fatigues [57, 83–85]. Singers and professional voice users often complain of impairment in the production of high pitches and variations of speaking fundamental frequency [86].

Strobovideolaryngoscopic findings of EBSLN injury are controversial and may include VF bowing, ipsilateral glottal rotation, lack of brisk adduction and abduction, VF lag on the affected side, lower displacement of the affected VF with scissoring, and decreased amplitude and mucosal wave of the affected VF. In addition, a posterior glottic gap may be observed, although this is classically seen in concurrent RLN injury [57, 83-85]. These subtle clinical signs can be further highlighted by asking the patient to perform certain vocal maneuvers. Rubin et al. [87] claimed that VF lag (sluggishness), especially during repetitive phonatory tasks (e.g., /i/-/hi/-/i/-/hi/-/i/-/ hi/), was a hallmark of SLN injury, clarifying that paretic nerves fatigue more quickly than normal nerves. Phonation in low pitch followed by phonation in high pitch is another simple and accurate diagnostic procedure for unilateral SLN paralysis. This task results in oblique glottis caused by posterior glottis rotation to the paralytic side [83, 88]. Roy et al. [89] identified epiglottic petiole deviation to the paralytic side during high-pitched phonation as a possible diagnostic marker of unilateral EBSLN denervation (Fig. 3).

Non-neural Dysphonia

Post-operative dysphonia is sometimes seen, even in the absence of neural injury [36]. These voice changes may be related to soft tissue trauma, hemorrhage, infection, endotracheal intubation, arytenoid trauma (e.g., arytenoid dislocation), and strap muscles injury, which impairs vertical laryngeal movement or causes laryngotracheal fixation [84]. Such alterations can cause vocal fatigue,



Fig. 3 Paresis of external branch of superior laryngeal nerve (ESLN) on right side [89]: A During vocal fold abduction, petiole is in midline. B During phonation produced at normal pitch and loudness, petiole remains in midline. C During phonation elicited at highest pitch during upward glissando maneuver, epiglottic petiole deviates to right side (arrow)

difficulty with singing high notes, and reduced vocal range [90–92], and may be temporary or permanent [36].

Voice changes in pituitary gland disorders

Disorders leading to deficiency in pituitary hormones are rare, and voice changes are not always obvious. Pituitary hormone excess, resulting from functional (i.e., endocrine-active or secreting) adenomas of the pituitary, is more common. As symptoms are generally insidious in onset and often nonspecific, a smart phoniatrician may be the first to suspect and diagnose a pituitary adenoma [2].

Growth Hormone

Excess growth hormone (GH) in adults causes acromegaly. The cartilaginous larynx widens and grows; the vocal folds become thickened, dropping the fundamental voice frequency and deepening the voice. Due to the general muscular weakness associated with the disease, the voice may be weak, sometimes phonasthenic [47]. Although much of the upper respiratory compromise is due to macroglossia and hypertrophy of pharyngeal soft tissue, cases of slowly progressive laryngeal obstruction requiring tracheotomy have been reported [93]. The cricoarytenoid joints may become fixed in acromegalics due to ankylosis of the cricoarytenoid joint. This could be related to the GH activity on joint chondrocalcification [94]. Pituitary adenomectomy has been shown to normalize vocal fundamental frequency within a few weeks of surgery [93]. Other physical changes (e.g., large hands, prominent jaw) are frequently permanent [2].

Adrenocorticotropic Hormone (ACTH)

Increased pituitary ACTH production causes Cushing's disease. Voice changes are more noticeable in females than males and can range from mild changes resembling premenstrual voice changes to severe, irreversible virilization of the voice [2].

Prolactin

In females, prolactin-secreting tumors are manifested by amenorrhoea, infertility, and galactorrhea because high prolactin levels block the natural Luteinizing hormone (LH) surge that initiates ovulation. Voice changes are similar to premenstrual voice changes. Men with prolactinomas usually complain only of diminished libido, and voice changes are not noticeable [2].

Voice changes in adrenal gland disorders

Congenital adrenal hyperplasia (CAH) is an autosomal recessive disorder that results in 95% of cases from 21-hydroxylase deficiency. This causes deficient production of cortisol and often aldosterone, and overproduction of androgens [95, 96].

Nygren et al. [14] found that females with CAH showed lower voice pitch and darker voice timbre when compared with controls. The authors attributed these voice changes to the effects of androgens on the VF structure and suggested that late diagnosis or improper treatment with glucocorticosteroids after birth could be the cause.

Voice changes in diabetes

Dysphonia is encountered in 12.5% of diabetic patients, which is more than twice as common as in healthy people. Diabetes is well known to cause microvascular disease, which can lead to diabetes-related neuropathy and myopathy that affect laryngeal function [97]. Neuropathy leads to a gradual loss of fine motor control, which may be noticed early by a professional voice user. Neuropathy may also result in vocal fold paralysis in advanced cases [98]. Hamdan et al. [99] found that diabetic patients had a higher prevalence of laryngeal sensory neuropathy as compared to healthy individuals. The authors suggested that symptoms like cough and spasm could be a possible consequence of this state [58]. Xerostomia, xerophonia, and hearing loss in long-standing diabetes also contribute to voice changes in these patients [36]. Furthermore, diabetes mellitus is linked

to a higher risk of infection, generalized fatigue, edema, inadequate wound healing, and other health issues that could affect voice performance [2].

Conclusion

Hormone imbalances and dysfunction commonly affect the voice, and dysphonia may be the initial sign of a serious systemic illness. Phoniatricians must be continually alert for hormonal dysfunction in patients with voice problems. Collaboration with an expert endocrinologist interested in professional voice problems is crucial [2]. Further research is needed to extend and enrich our knowledge of hormonerelated voice changes.

Abbreviations

ACTH	Adrenocorticotropic Hormone
APA	Auditory Perceptual Assessment
CAH	Congenital Adrenal Hyperplasia
EBSLN	External Branch of the Superior Laryngeal Nerve
ENT	Ear, Nose, and Throat
GH	Growth Hormone
H/N	Harmonic to Noise
HRT	Hormone Replacement Therapy
LH	Luteinizing Hormone
MPT	Maximum Phonation Time
PCOS	Polycystic Ovarian Syndrome
PMS	Premenstrual Syndrome
RLN	Recurrent Laryngeal Nerve
SLN	Superior Laryngeal Nerve
UVFP	Unilateral Vocal Fold Paralysis
VFs	Vocal Folds

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Authors' contributions

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