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Application of nonlinear dynamics theory to understanding normal and pathologic voices in humans

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The theory of nonlinear dynamics was introduced to voice science in the 1990s and revolutionized our understanding of human voice production mechanisms. This theory elegantly explains highly complex phenomena in the human voice, such as subharmonic and rough-sounding voice, register breaks, and intermittent aphonic breaks. These phenomena occur not only in pathologic, dysphonic voices but are also explored for artistic purposes, such as contemporary singing. The theory reveals that sudden changes in vocal fold vibratory patterns and fundamental frequency can result from subtle alterations in vocal fold geometry, mechanical properties, adduction, symmetry or lung pressure. Furthermore, these changes can be influenced by interactions with supraglottal tract and subglottal tract resonances. Crucially, the eigenmodes (modes of vibration) of the vocal folds play a significant role in these phenomena. Understanding how the left and right vocal fold eigenmodes interact and entrain with each other, as well as their interplay with supraglottal tissues, glottal airflow and acoustic resonances, is essential for more sophisticated diagnosis and targeted treatment of voice disorders in the future. Additionally, this knowledge can be helpful in modern vocal pedagogy. This article reviews the concepts of nonlinear dynamics that are important for understanding normal and pathologic voice production in humans.

This article is part of the theme issue 'Nonlinear phenomena in vertebrate vocalizations: mechanisms and communicative functions'.

1. Introduction: vocal apparatus as a system of coupled oscillators

Human phonatory apparatus is anatomically located in the breathing airways and consists of (i) the lungs and the subglottal tract delivering airflow, functioning as the power supply, and also as an acoustic resonator (cavities of the lungs, bronchi, trachea), (ii) oscillating tissues located mostly in the larynx (mainly the vocal folds) that modulate the airflow to produce the sound source, and (iii) the supraglottal vocal tract (the complex of epilaryngeal, pharyngeal, oral and nasal cavities) serving as an acoustic resonator and sound modifier ([1–3]; figure 1, top). As trivial as it seems to uninformed listeners, human phonation results from highly complex neural, muscular, aerodynamic, biomechanical and acoustic processes. For brevity, in this article, we will focus mainly on the nonlinear processes of fluid–structure–acoustic interactions in the larynx and the subglottal and supraglottal tracts. The high-level neuromuscular motor control and somatosensory feedback, albeit highly important [4–8], will not be discussed here.

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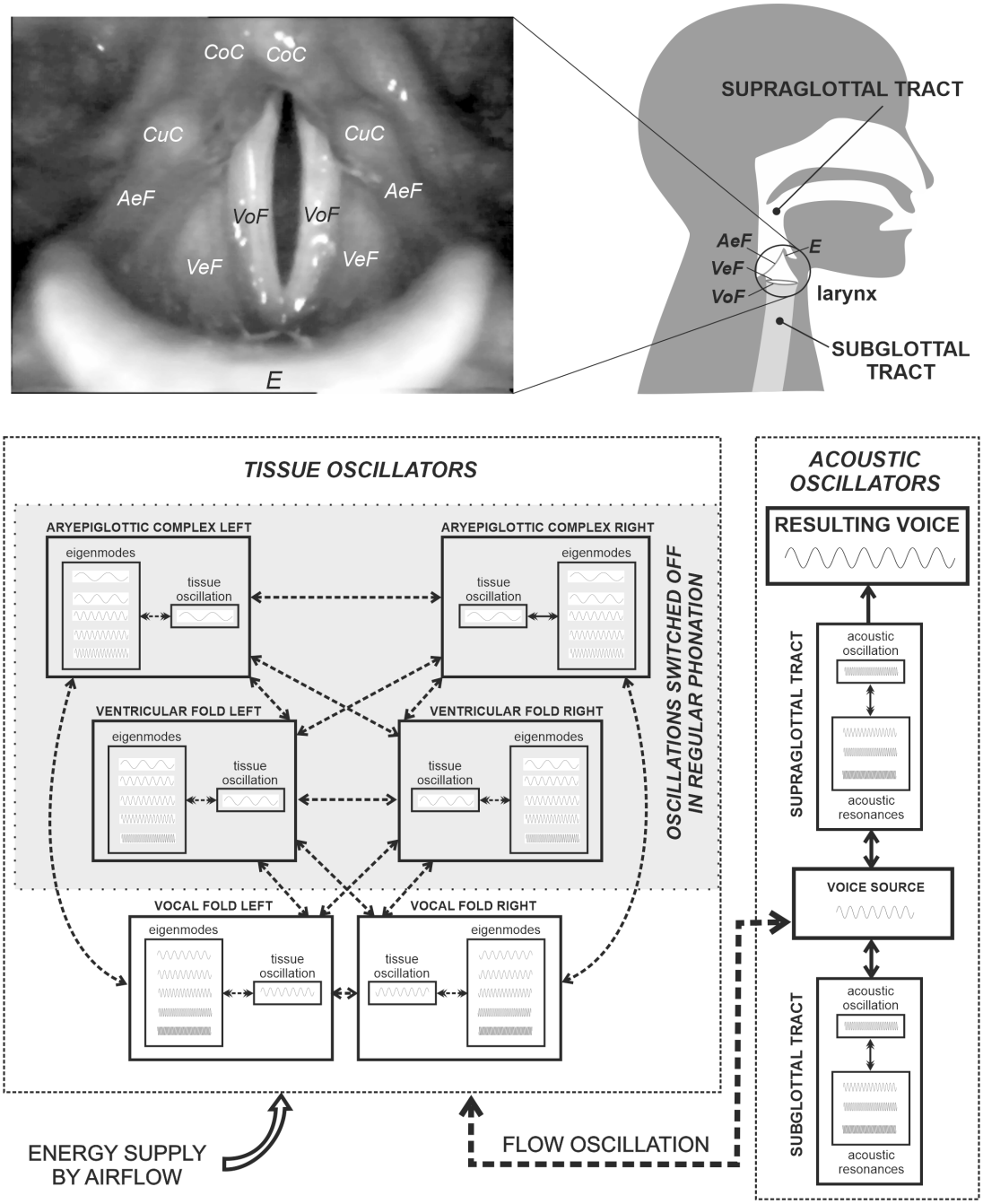

Figure 1. The phonatory apparatus in humans. Top: endoscopic view of the larynx with the tissues that can oscillate during voice production (left) and their anatomic location in the breathing airways, which act as acoustic oscillators (right). Bottom: the phonatory apparatus schematically represented as a system of multiple coupled oscillators. The double-headed arrows represent mutual interactions between the different oscillators. VoF, vocal fold; VeF, ventricular fold; AeF, aryepiglottic fold; E, epiglottis; CuC, cuneiform cartilage; CoC, corniculate cartilage placed on the top of the arytenoid cartilage. Together, E and the pairs of CoC, CuC and AeF form the tissues of the aryepiglottic complex, also termed the 'laryngeal collar'.

Principally, the phonatory apparatus can be understood as a nonlinear dynamic system of many coupled oscillators interacting with each other. They can be divided into tissue and acoustic oscillators (figure 1, bottom). The most crucial tissue oscillators are the two elastic vocal folds, which are excited by airflow delivered from the lungs. The vocal folds can vibrate in many ways and exhibit different vibratory regimes, depending on the eigenmodes of the vocal folds being excited by the fluid–tissue–acoustic interaction. These will be extensively covered in the following sections.

Besides the vocal folds, some supraglottal tissues may also exhibit flow-induced oscillations during phonation. These include the two ventricular folds—also known as false vocal folds—which are located immediately above the vocal folds, as well as the tissues of the aryepiglottic complex forming the laryngeal collar. The aryepiglottic complex encompasses the apexes of the arytenoid cartilages, the two aryepiglottic folds with embedded cuneiform and corniculate cartilages, and the epiglottis (figure 1). In regular phonatory cases, these supraglottal tissues do not oscillate. However, they can be brought into oscillations and interfere with the vocal fold oscillations [9,10]. The involuntary oscillation and participation of the ventricular folds and tissues of the aryepiglottic complex during phonation is often considered pathologic and diagnosed as ventricular dysphonia or muscle tension dysphonia [11–15]. On the other hand, voluntary oscillation of these supraglottal tissues is explored in some languages for phonetic purposes [16,17], and in some ethnic and contemporary singing styles, such as hard rock, metal or alternative

singing, for artistic effects [18–22]. In these cases, such voice production is not recognized as pathologic [23]. Importantly, these supraglottal tissues may help to create a substitute voice source in patients with dysfunctional or missing vocal folds [24,25]. Like the vocal folds, the supraglottal tissues can exhibit different vibratory regimes, depending on the eigenmodes involved in their resulting vibration.

During phonation, the tissue oscillators interact with the air flowing from the lungs and are induced into self-sustained oscillations in a complex process involving flow–tissue–acoustic interactions. This process is described by the 'myoelastic–aerodynamic (MEAD) theory' of voice production [26–29]. The laryngeal tissue vibration modulates the glottal (laryngeal) airflow, which is the main voice source [2,30–32]. This sound source excites two acoustic resonators: the subglottal tract, i.e. the cavities below the larynx, and the supraglottal tract, also called the vocal tract, formed by the cavities above the larynx (figure 1). These cavities act as acoustic filters and modify the sound of the source at the glottis. This acoustic process has classically been described by the 'source-filter theory' of voice production [32,33]. The subglottal and supraglottal tracts have many acoustic resonances, which can be excited depending on the sound source spectrum. The supraglottal tract's excited resonances appear as 'formants' in the resulting voice spectra [1,32,33]. The formants of the supraglottal tract not only play a major role in determining the acoustic quality of the produced speech sound, particularly for vowels, but also contribute to the timbre of the singing voice [34–36]. The formants of the subglottal tract remain mainly inside the body and are usually neglected in the traditional linear source–filter theory of voice production. However, recent findings extend the linear source–filter theory by incorporating the nonlinear source–filter interaction effects, through which the acoustic oscillations of both the subglottal and supraglottal tracts can influence the voice source by altering glottal airflow (level 1 interaction) or even laryngeal tissue oscillations (level 2 interaction) [37]. The source–filter and MEAD theories explain the voice production of humans, as well as the sound production of other vertebrates, such as mammals, birds and amphibians [38–45]. Therefore, principally similar voice phenomena occur in animals and humans, only reflecting the differences in anatomy, physiology and control systems of the different animals [46,47].

2. The internal oscillators within the oscillating tissues—eigenmodes

The tissue oscillators in the human vocal apparatus, such as the vocal folds, ventricular folds, and the tissues of the aryepiglottic complex, can vibrate in different ways and at different frequencies, depending on the eigenmodes involved in the resulting oscillation. The role of the eigenmodes is crucial for explaining many vocal phenomena. However, its understanding is far from complete. In general, eigenmodes, or modes of vibration, describe fundamental patterns of movement that are allowed by physical laws and boundary constraints [48–50]. Each eigenmode has a specific frequency (eigenfrequency) at which all parts of the system move either in phase or 180° out of phase. Similarly, eigenmodes also exist for acoustic propagation in the sub- and supraglottal tracts and occur as acoustic resonances [51,52].

The eigenmodes can be viewed as building blocks for constructing more complex vibratory patterns, such as wave-like motions in which the different parts of the system vibrate with different phases. This occurs, for example, in the vocal folds during phonation. For the vocal system, tissue eigenmodes can be considered for the vocal folds alone without their interaction with the airflow. In this case, the eigenmodes are referred to as *in vacuo* eigenmodes. The eigenfrequencies and spatial movement pattern of the *in vacuo* eigenmodes depend on the geometric and mechanical properties of the tissue oscillators. Thus, vocal fold eigenmodes and their eigenfrequencies often vary among larynges, with muscular adjustments, or owing to pathology or ageing. The eigenmodes can be calculated mathematically [53–56] or observed experimentally by exciting the vocal apparatus with external vibrations without airflow [57]. When the vocal folds and their interaction are considered as a whole, the eigenmodes and their eigenfrequencies will vary with the airflow, as described further below in section 4a.

The *in vacuo* eigenmodes of the most crucial vocal oscillator—vocal folds—were first theoretically described in 1975 by Titze & Strong [53] and first visualized laryngoscopically *in vivo* in 2000 by Švec *et al*. [57]. While each eigenmode exhibits movement in all three dimensions, vibration in the medial–lateral direction directly modulates airflow and is thus more relevant to voice production. Examples of some of the vocal fold oscillatory movements along the medial–lateral direction for a few *in vacuo* eigenmodes are shown in figure 2. Theoretically, each vocal fold has an infinite number of *in vacuo* eigenmodes [53,54]. However, usually, only a few eigenmodes are strongly excited. In normal voice, the combination of 10 and 11 modes (figure 2), which describe the in-phase and out-of-phase medial–lateral movement, respectively, has been found to be essential for the observed vocal fold motion [27,58–61]. The higher-order modes, such as the 20, 21 or 30 modes (figure 2), were found to play a role in subharmonic, biphonic and irregular vocal fold oscillations [60,62–67]. Similarly to the vocal folds, the ventricular and aryepiglottic folds also have eigenmodes of oscillations that determine their oscillatory behaviour when excited. All these eigenmodes of the laryngeal tissues and the sub- and supraglottal tracts act as coupled oscillators within the phonatory apparatus (recall figure 1).

3. Nonlinear dynamic phenomena in voice—attractors and bifurcations

Since many coupled oscillators are involved in the voice production process, the phonatory system exhibits nonlinear dynamic phenomena similar to those known from other systems of coupled oscillators in physics. This became apparent in the 1990s when the theory of nonlinear dynamics was discovered as a novel tool for explaining voice phenomena [68–72]. Crucial insights into the nonlinear dynamics of voice were provided in 1993 by Titze, Baken and Herzel in their landmark article [73]. To

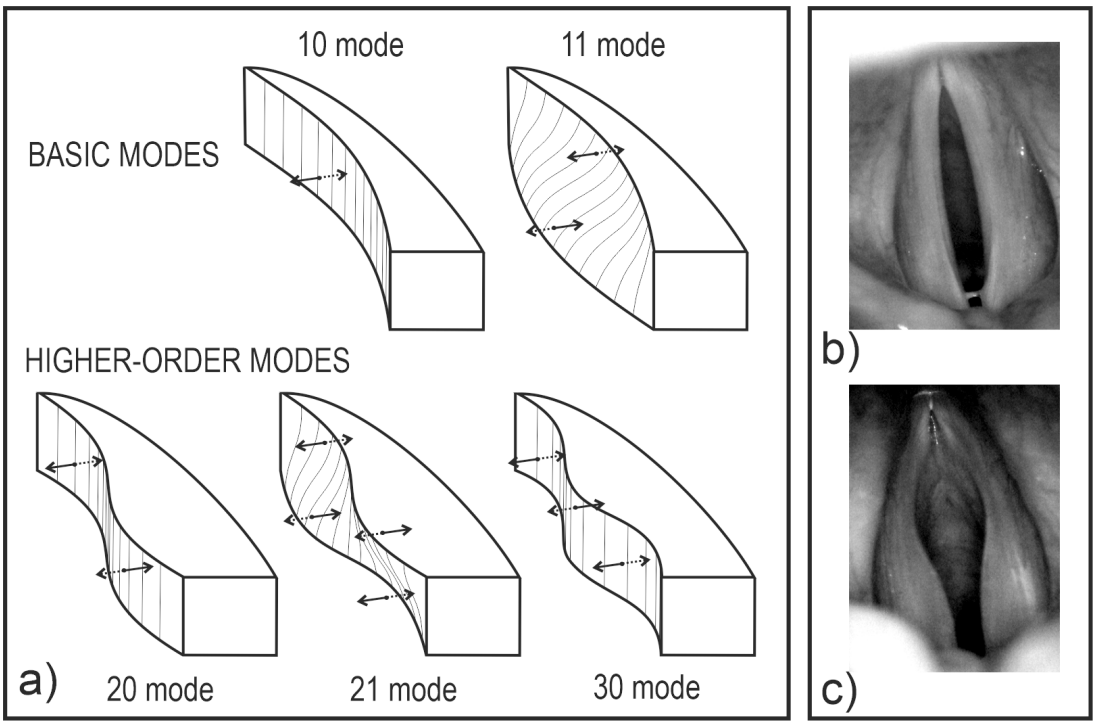

Figure 2. (a) Schematic view of the medial–lateral movement of selected *in vacuo* eigenmodes of the right vocal fold. The basic modes (10 and 11) are expected to be excited in normal voice, whereas the higher-order modes (20,21,30, etc.) play a role in subharmonic and irregular vocal fold oscillations. The two digits labelling the modes indicate the number of half-wavelengths across the vocal fold length and thickness, respectively. Inspired by Titze, 2000 [1]. (b) High-speed videolaryngoscopic view of normal phonation in which the vibratory pattern of the vocal folds is formed mainly by the basic modes (i.e. 10 and 11 modes). (c) High-speed videolaryngoscopic view of a rough subharmonic phonation. The deflection shape of the oscillating vocal folds indicates a strong presence of the 21 mode here.

explain the basic principles, let us consider two oscillators with natural oscillating frequencies f_1 and f_2, coupled with a coupling strength K. These two oscillators could represent, e.g. two asymmetric vocal folds, co-vibrating vocal and ventricular folds, two tissue eigenmodes, or acoustic resonances of the sub- and supraglottal tract.

Depending on the frequency ratio f_2/f_1 and the coupling strength K, the oscillators will show different patterns of oscillations (figure 3). For small coupling strength, the two oscillators will influence each other very little and will vibrate desynchronously at frequencies equal to or close to their respective natural frequencies f_1 and f_2. With increasing coupling strength, the two oscillators will interact more, and their frequencies will tend to get synchronized (entrained) at an integer ratio, i.e. 1 : 1, 1 : 2, 2 : 3, etc. In figure 3, the synchronization regions are recognized as 'Arnold tongues' [74,75]. The tongues correspond to different 'attractors' in a phase space and relate to simple as well as to complex subharmonic vibration regimes [46,73]. As seen in figure 3, increasing coupling strength broadens the f_2/f_1 regions for synchronized behaviour, but when the coupling strength becomes too high, the different tongues start to overlap and the two oscillators will tend to behave chaotically. When more than two oscillators get involved, their synchronization patterns become more complex and the subharmonic and chaotic behaviour becomes more likely.

In general, it is helpful to classify four basic attractor types as they appear in phase space [46,73]:

(1) Stationary point, characterizing non-oscillatory behaviour;
(2) Limit cycle, characterizing (pseudo-)periodic oscillations. The limit cycle can be simple, typical for healthy voice (relates to 1 : 1 synchronization pattern), or complex, typical for subharmonic phonations (relates to entrainment ratios of 1 : 2, 1 : 3, 2 : 3, 3 : 4, etc.). The subharmonic oscillations are typically perceived as rough voices with low pitch, or as phonations with two simultaneous pitches related by the musical interval of a fifth, fourth, major or minor third, etc., and fall under the phonations known as diplophonia [76,77].
(3) Torus, characterizing relatively independent, desynchronized oscillations with unrelated frequencies, typical for biphonic voices [76,78]. They are perceived as rough voices with two independent audible pitches. Tori (biphonation), and folded, complex limit cycles (subharmonic phonations) are often not distinguished from each other in clinical practice and are recognized under the more general term 'diplophonia' [79,80].
(4) Strange attractor, characterizing irregular, chaotic oscillations, typical for heavily rough voices with perceptually unrecognizable pitch. Together with breathiness, roughness is an essential component of voice hoarseness [81,82].

There have been many research efforts toward adapting such classification for both research and clinical purposes. More details can be found, e.g. in the papers of Titze [83], Jiang *et al*. [84], Zhang *et al*. [85] or Sprecher *et al*. [86].

When crossing the boundaries between the different attractor regions, the oscillatory behaviour can change from one to another attractor suddenly. Such sudden behaviour changes between different attractors are called 'bifurcations' [73,87]. It has been found that a smooth change of a single phonatory parameter can trigger sudden changes in phonatory behaviour.

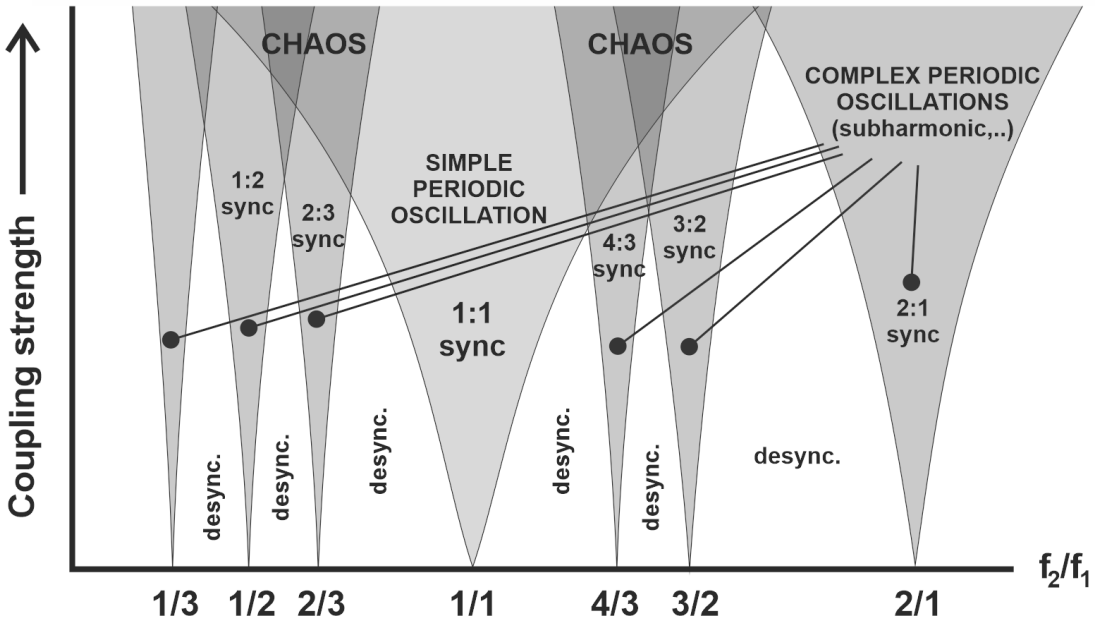

Figure 3. Schematic bifurcation diagram of the behaviour of two coupled oscillators with different ratios of their natural frequencies f_2/f_1. The resulting behaviour shows either simple or complex oscillations based on the frequency ratio f_2/f_1 and the coupling strength. Such resulting oscillations are also observed in voice: simple oscillations (1 : 1 synchronization) are typical for normal voice; complex periodic oscillations (synchronizations 1 : 2, 2 : 3, 4 : 3, etc.) occur in subharmonic phonations; two unrelated frequencies (desynchronization) are found in biphonation; and chaotic oscillations are found in irregular, heavily rough voices. Inspired by Titze, Baken and Herzel, 1993 [73].

This parameter can be airflow, subglottal pressure, vocal fold tension or stiffness, vocal fold adduction, vocal fold asymmetry, ventricular fold adduction or asymmetry, vocal fold thickness, formant adjustments, etc., as discussed below.

4. The coupling strength: three coupling mechanisms in phonation

There are at least three coupling mechanisms during phonation: coupling by airflow, by contact, and by source–filter interaction. The coupling of the two vocal folds by the intraglottal airflow is the most essential coupling mechanism for the self-sustained vocal fold oscillation. Most notably, it allows the synchronization of eigenmodes in vocal fold tissues, enabling phonation onset. Furthermore, coupling by airflow may also excite the supralaryngeal tissues into vibration and contribute to their synchronization pattern with the vocal folds. Once the vocal folds are excited into vibration and come into contact, the vocal fold contact functions as an important coupling mechanism, which may stabilize vocal fold vibration but, when excessively strong, can also lead to subharmonic or chaotic vocal fold vibration. Source–filter interaction may also impact glottal flow and even vocal fold vibration. In the following, we briefly discuss these three major coupling mechanisms and how the coupling strengths can be manipulated in human phonation. While there have been many research efforts on this topic, e.g. [88–90], the following focuses on insights gained from studies using computational continuum models of phonation [59,91–94], which allow discussion of vocal control through manipulation of realistic properties of the vocal system.

(a) Phonation onset: eigenmode synchronization by airflow

Coupling by airflow plays a role in various voice phenomena, including, e.g., entrainment of the oscillations of the two vocal folds and of the supraglottal tissues, but also synchronization of the eigenmodes in the tissues. Here, we describe one such synchronization effect enabling the onset of phonation. Phonation results from a complex interaction between the vocal folds and the glottal flow, which transfers energy from the airflow into the vocal folds to initiate and sustain vibration. While there are many potential mechanisms for energy transfer from airflow to vocal folds (see recent reviews in [2] and [29]), an important mechanism is related to the vocal fold eigenmodes and their coupling by airflow. The lowest subglottal pressure that enables phonation is recognized as the 'phonation threshold pressure' [95]. When the subglottal pressure reaches the threshold value, phonation starts through a Hopf bifurcation [73,90,96], in which one eigenmode becomes excited in a mode-merging process and its amplitude grows with time until the final limit cycle is reached [91,97].

One example of such mode-merging process is presented in figure 4, which shows the imaginary (frequency) and real (growth rate) parts of the first three eigenvalues of the combined vocal-fold-airflow system as a function of increasing subglottal pressure. The leftmost condition in the figure represents the state at rest when no airflow is applied, at which the eigenvalues reduce to those of the *in vacuo* vocal fold eigenmodes, i.e., the vocal fold resonance frequencies. In this case, the first *in vacuo* eigenmode exhibits movement mainly in the vertical direction, whereas the second and third eigenmodes exhibit largely medial–lateral in-phase and out-of-phase motion (corresponding to modes 10 and 11 in figure 2), respectively. The *in vacuo* eigenvalues have a zero growth rate, indicating that any disturbance will not grow in amplitude with time. As the subglottal

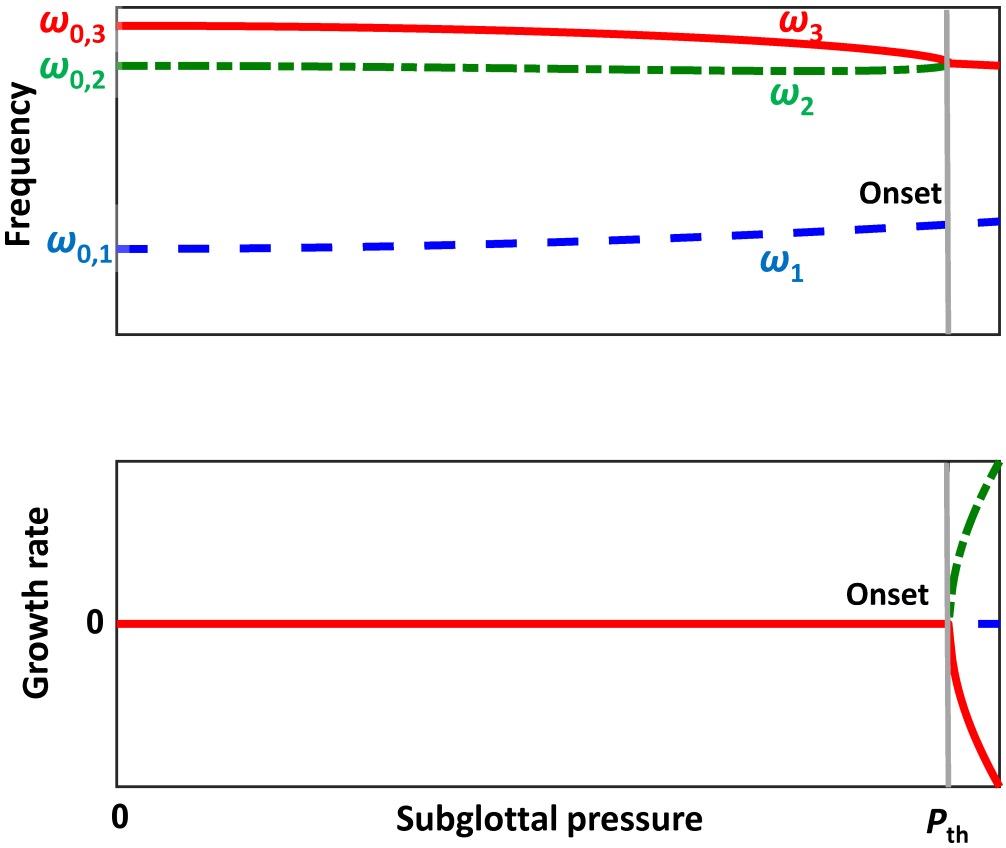

Figure 4. The imaginary (frequency) and real (growth rate) of the first three eigenmodes of the coupled vocal fold-airflow system. With increasing subglottal pressure, the frequencies of the second and third eigenmodes gradually approach each other. Phonation onset occurs as a Hopf bifurcation as the second and third eigenmodes are synchronized to the same frequency, at which the second eigenmode becomes excited with a positive growth rate. Data from a computational continuum model of vocal folds, adapted from [2,91].

pressure increases, the interaction between the vocal folds and airflow modifies the eigenfrequencies, the shapes of deformation, and the vibration patterns associated with the eigenmodes. In figure 4, the airflow causes the second and third modes to gradually approach each other in frequency, eventually merging and synchronizing at the same frequency. This synchronization combines the second and third *in vacuo* modes of the vocal folds and forms a complex conjugate pair of eigenmodes. In one of the eigenmodes (the second mode in figure 4), the two *in vacuo* eigenmodes are synchronized in a favourable phase relation that allows net energy transfer from airflow into the vocal folds, as indicated by the positive growth rate. This indicates that the amplitude of this mode will grow over time, enabling phonation. In contrast, for the third eigenmode in figure 4, the *in vacuo* eigenmodes are synchronized in a way that transfers energy away from the vocal folds. As a result, the third eigenmode has a negative growth rate and will not be excited. The interested reader is referred to [91] for the mathematical details of this mode-merging synchronization process.

Because phonation onset results from the interaction of eigenmodes, the resulting vocal fold vibration often includes contributions from the first few *in vacuo* eigenmodes of the vocal folds, particularly the ones heavily involved in the synchronization (the second and third *in vacuo* eigenmodes in figure 4). These eigenmodes are synchronized at the same frequency but in a favourable phase relationship, often leading to a vertical phase difference in vocal fold motion along the surface, also known as a mucosal wave [98]. This vertical phase difference allows the intraglottal pressure waveform to be at least partially in phase with vocal fold velocity, thus transferring energy into the vocal folds to initiate and sustain phonation (see [91] for a detailed analysis). Note that flow-induced vibration owing to eigenmode synchronization is a common phenomenon in coupled fluid-structure systems [99,100].

For simplicity, only the first three eigenmodes are shown in figure 4 to illustrate the concept of eigenmode synchronization. The vocal folds have an infinite number of eigenmodes, and eigenmode synchronization may occur between many different pairs or groups of eigenmodes. The group of eigenmodes that requires the least subglottal pressure to synchronize will eventually dominate and emerge at phonation onset. For example, in figure 4, although the first and second eigenmodes also slightly approach each other in frequency, it is the second and third eigenmodes that are synchronized at a lower subglottal pressure and eventually emerge at phonation onset.

The amount of subglottal pressure required to initiate phonation onset P_{th} depends on the frequency spacing of the two *in vacuo* eigenmodes of the vocal folds that are being synchronized, and their coupling strength β. For the example given in figure 4, in which the second and third eigenmodes (with *in vacuo* frequencies $\omega_{0,2}$ and $\omega_{0,3}$) are synchronized, the phonation threshold pressure is provided by the formula below [92]:

$$P_{\text{th}} = \frac{\omega_{0,3}^2 - \omega_{0,2}^2}{\beta}.$$

Thus, if the two *in vacuo* eigenmodes are far apart in frequency or not coupled well, a high subglottal pressure is required to initialize phonation. Zhang provided an analytic equation for the coupling strength β associated with airflow–vocal fold interaction [92]. In general, the coupling is stronger for smaller gaps, particularly when the glottal gap is small with respect to the tracheal size [92]. Thus, medialization to bring the vocal folds together often lowers the phonation threshold pressure. The medial surface vertical thickness also has a moderate effect, with the phonation threshold pressure lowest at an intermediate thickness and much higher for vocal folds that are either too thin or too thick [93]. The movement patterns in the two *in vacuo* eigenmodes that are being synchronized are also important. The coupling is strong if the motion in one mode is spatially similar to the intraglottal pressure induced by the other mode. In other words, the coupling is strong if one mode creates an intraglottal pressure profile similar to vocal fold motion in the other mode, allowing efficient energy transfer from one mode to the other. This is why an in-phase mode is often more easily synchronized with an out-of-phase mode, and why phonation onset in figure 4 occurs between the second and third eigenmodes instead of between the first and second eigenmodes.

To facilitate synchronization, one could reduce the frequency spacing between eigenmodes, which can be achieved by muscular adjustments that manipulate vocal fold stiffness and geometry [2]. One could also increase coupling strength by increasing vocal fold approximation, as mentioned earlier, or maintaining an intermediate vertical thickness. Finally, one may also increase the subglottal pressure. Theory predicts that relatively small changes in the eigenfrequency spacing, coupling strength or subglottal pressure could cause sudden changes from no voicing to voicing, which relates to, e.g. the intermittent aphonic breaks observed in pathologic voices.

Figure 4 shows phonation onset that occurs with a gradual increase in the amplitude of the second eigenmode, also known as a supercritical Hopf bifurcation. Phonation onset may also occur through a subcritical Hopf bifurcation, in which small gradual increase in subglottal pressure over threshold leads to an abrupt increase in the vocal fold vibration amplitude. In such a case, hysteresis of the phonation threshold pressure is observed, i.e. different threshold values for phonation onset and offset [101–103]. More details can be found in [90] and [104].

(b) Coupling by vocal fold contact

Another important coupling mechanism is vocal fold contact. Indeed, sufficient vocal fold contact is essential to producing normal-sounding voice quality. Insufficient vocal fold contact may potentially lead to desynchronization of the left and right vocal folds, causing biphonation or diplophonia [105]. Improving vocal fold contact has been one of the most essential physiological goals of voice therapy for people with breathy and weak voices. Improved vocal fold contact can be achieved through vocal exercises or through surgical techniques. Some of the current possibilities for surgically manipulating coupling strength through vocal fold contact are discussed in §7 of this article, focusing on patients with left–right vocal fold asymmetry due to unilateral vocal fold paralysis.

While vocal fold contact is important for normal voice production, too strong vocal fold contact is not considered beneficial, as it strongly enhances cross-eigenmode interaction due to the highly nonlinear nature of contact mechanics [94]. One consequence of such enhanced interaction is that many eigenmodes are excited by excessive vocal fold contact, and they tend to entrain simultaneously at various synchronization ratios (recall figure 3). For example, creaky phonations often arise through such enhanced contact-related coupling. Increasing coupling strength due to stronger vocal fold contact often leads to a cascade of changes in vocal fold vibration, from subharmonics to chaos [94]. The contact-related coupling strength depends on the spatial extent and depth of vocal fold contact, and is controlled primarily by the vertical thickness of the medial surface, glottal gap, vocal fold stiffness in the transverse plane and the subglottal pressure. In general, vocal instabilities such as subharmonics or chaos are more likely to occur in thick folds with low transverse stiffness when tightly approximated, often through the actions of the thyroarytenoid muscles, and when subjected to high pressure [94].

In cases of organic pathologies, such as lesions placed around the mid-membranous portion of the vocal folds (e.g. nodules, polyps or cysts), the contact is localized to only limited portion of the vocal fold. This creates a node that hinders tissue oscillations at that location and supports the excitation of higher-order anterior–posterior modes, entraining at frequency ratios different from 1 : 1, which causes subharmonic and irregular vocal fold oscillations observed in dysphonia [66,106,107]. Overall, the contact-related coupling in phonation is a highly complex phenomenon and its understanding is rather limited.

(c) Coupling by source–filter interaction

While the classical acoustic source–filter theory of voice production assumed the voice source to be independent from the filter, i.e., from the vocal tract [32], the current understanding is that the voice source can be altered through the interaction with the subglottal and supraglottal tracts' acoustics [3,37,108]. The voice source modification happens through two levels of acoustic interaction [37]. The first level (level 1) was described by Rothenberg in 1970s and was found to be based on the flow–acoustic interaction owing to vocal tract inertance [109,110]. It causes changes (skewing) in the glottal flow pulse [30,109–111]. This modifies the sound intensity and sound spectrum. It supports voice production when the voice fundamental frequency is lower than any resonance frequency of the vocal tract, and the input acoustic impedance of the vocal tract is inertive [37,108,111]. With a strong coupling to the glottal airflow, acoustic interaction may also have a noticeable effect on the oscillations of the vocal folds as well as those of the supraglottal tissues. Titze [37] labelled this as level 2 interaction. It has been found to cause changes in the fundamental frequency (f_o) of voice, as well as in the vocal fold oscillation amplitudes and the dynamic glottal closure pattern [37]. The effects of the vocal tract acoustic resonances on the voice f_o were first observed by Weiss in 1932 during singing experiments with artificially lengthened vocal tracts [112,113], but were later found also to occur in realistic *in vivo* conditions

[114–117]. Extreme vocal tract constriction, as in the case of voiced consonants, may also reduce the transglottal pressure—the driving force of vocal fold vibration—thus reducing the amplitudes of both vocal fold vibration and glottal flow.

The coupling strength between the source and the vocal tract was found to be increased by increasing the vocal tract inertance through narrowing the epilaryngeal space [37,118,119]. Manipulating source–filter interaction by changing the vocal tract shape is often explored in voice therapy, particularly in semioccluded voice therapy exercises, and is considered helpful for making the resulting voice more efficient [120–123]. However, there are still some contradicting issues making our understanding of the interaction effects far from complete [124].

The source–filter coupling strength also critically depends on the laryngeal configurations. For example, while vocal tract inertance often skews the glottal flow waveform to the right, this effect becomes negligible when the vocal folds vibrate with incomplete glottal closure [124,125]. Recent studies also showed that when allowed to move freely in the vertical dimension, the vocal folds are more easily entrained to vocal tract resonances, and restraining the vertical motion of the vocal folds reduces the degree of source–filter interaction [124,126,127].

The strongest source–filter interaction effects occur when the fundamental frequency f_o of phonation approaches the resonance frequency of the supraglottal tract, as in the case of soprano singing [116,128–130]. In such conditions, synchronization may occur between vocal fold eigenmodes and one of the acoustic resonances. As a result, the vocal folds often vibrate at a frequency close to the acoustic resonance being synchronized [131,132]. However, voice instabilities may occur when the voice f_o is crossing the resonance frequencies of the vocal tract [114–116,133–136]. For speech, the f_o is often sufficiently lower than any acoustic resonance, but when the f_o is close to a rational ratio with one of the acoustic resonances, a non-1 : 1 synchronization between f_o and the acoustic resonance may still occur, which may lead to subharmonics or chaos [37]. For more general conditions, source–filter interaction is weak and its impact on the voice source is small [124]. However, source–filter interaction can still interfere with the eigenmode synchronization pattern at the larynx and thus modify the bifurcation boundary, which may either suppress laryngeal instabilities or introduce new vocal instabilities [94]. Owing to the complexity involved, currently it is not possible to predict whether source–filter interaction suppresses or facilitates vocal instabilities at a specific voice condition, except for a few special conditions such as when f_o approaches a vocal tract resonance.

The role of the subglottal tract for voice production has been investigated much less than that of the supraglottal tract. The subglottal resonances are much less apparent in the resulting voice signal, although they are not negligible [137]. Theoretical and experimental studies reveal that, like the vocal tract, the subglottal tract shows both level 1 and level 2 interactions with the voice source [37,108,115,131,138,139]. Similarly, interactions with subglottal tract were found to trigger voice instabilities [115,131]. The importance of the influence of subglottal acoustics on voice instabilities is still a matter of debate—Lehoux *et al*. [140] reported that similar frequency jumps occur in human larynges with or without subglottal resonances, and the subglottal resonances caused only minor modifications to these. Importantly and unquestionably, the shape of the subglottal tract is much more rigid than the shape of the vocal tract and it therefore shows rather fixed resonance frequencies. Titze suggested that these subglottal resonances could be responsible for the occurrence of the rather fixed transition ranges between chest, head, middle and head registers recognized in singing pedagogy [1,141]; more research is required in this area, however.

5. Register breaks: Competition between eigenmode synchronizations

As mentioned earlier, each vocal fold has an infinite number of *in vacuo* eigenmodes. This means that synchronization can occur between different groups of eigenmodes. These different groups of eigenmodes may compete with each other for dominance. This competition for dominance offers an explanation to perhaps the most controversial problem in voice science and singing voice pedagogy, i.e., the discontinuity between the chest and falsetto registers in human voice. The chest and falsetto registers correspond to different vibratory regimes of the vocal folds and are also known as the two laryngeal vibratory mechanisms M1 and M2, respectively [142,143]. The M1–M2 discontinuity is known particularly as involuntary voice breaks in boys during puberty [144,145], but presents also a challenge for singing voice in adults. In western operatic tradition, singers put extensive effort into avoiding and smoothing this register discontinuity; on the other hand, register jumps are voluntarily explored for artistic purposes in some singing styles, such as in yodelling [146,147]. In 1998, Berry *et al*. [148] hypothesized that dominance of different groups of vocal fold eigenmodes is responsible for the occurrence of chest and falsetto registers in voice. Such shift in mode dominance was later observed in voice registers simulated by mathematical models of the vocal folds by Tokuda *et al*. [149], Zhang [94,150] and Geng *et al*. [151]. The studies show that, at certain critical conditions, a slight change in the control parameters of the vocal system may tip the balance between different eigenmode groups and cause another group of eigenmodes to become dominant. When this happens, it often leads to a frequency jump or a qualitatively different vibratory pattern. Such sudden change in vocal fold oscillation has been related to the so-called ‘saddle node bifurcation’ [118,152,153].

Figure 5 shows an example of a simulated break from chest to falsetto register, triggered by smooth increase of subglottal pressure, reported by Zhang in 2018 [94]. In chest-like vibration, the vocal folds vibrate with a long duration of glottal closure, whereas in falsetto-like vibration pattern, the vocal folds vibrate with a much-reduced glottal closure (figure 5b) and increased fundamental frequency. Figure 5c shows that this sudden change in vocal fold oscillation is accompanied by a dramatic change in eigenmode excitation. Mainly the first *in vacuo* vocal fold eigenmode contributes to the vibratory pattern in the falsetto-like vibration, whereas both the first two *in vacuo* eigenmodes are strongly excited in the chest-like vibration. Whereas in figure 5 this jump was triggered by a change in subglottal pressure, similar bifurcations have also been observed in many experimental and computational studies to be triggered by a slight change in vocal fold tension, stiffness, or geometry [89,140,149–151,154–157], as well as by changes in the resonances of the subglottal and vocal tract [37,115,118,131,136].

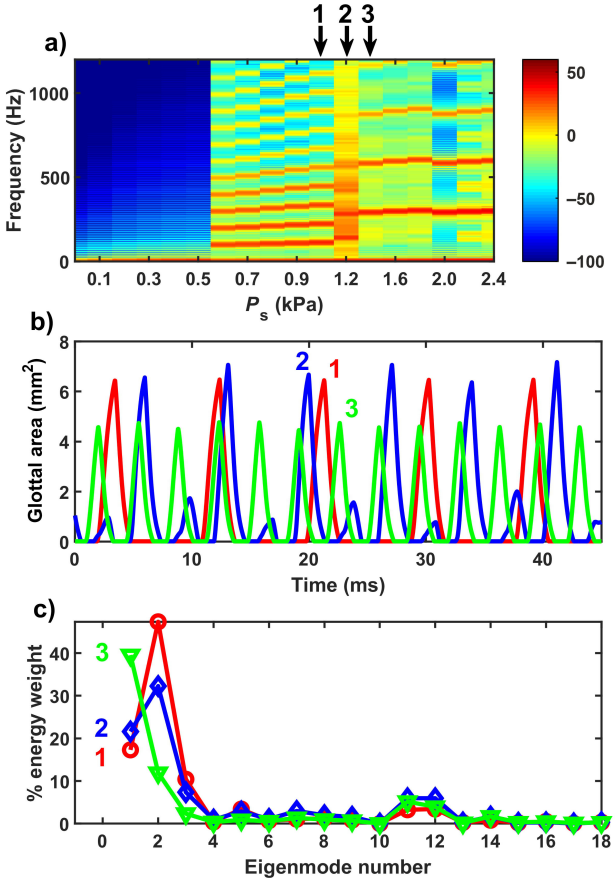

Figure 5. A chest-falsetto register jump induced by a slight increase in the subglottal pressure P_S. (a) A narrow band spectrogram; (b) glottal area waveforms from three instances of the experiment—before (1), during (2) and after (3) the transition, as indicated in a); (c) percentage energy contribution of the different *in vacuo* vocal fold eigenmodes to the resulting vocal fold oscillation before (1), during (2) and after (3) transition. The register jump is accompanied by an increase in fundamental frequency from 129 to 298 Hz, a significant decrease in the closed quotient from 0.67 to 0.30, and energy redistribution among the first few vocal fold eigenmodes. Data from a computational continuum model of vocal folds, adapted from [94].

In vivo experiments with chest-falsetto register breaks showed that different people exhibit different spontaneous leap intervals in f_o [155,158,159]. Different spontaneous leap intervals were also found among different excised human larynges, revealing that individual laryngeal anatomy largely influences the magnitude of the chest-falsetto breaks [140,156]. Differences in laryngeal anatomy have also recently been related to differences in leap intervals observed in animal vocalization [160]. In human singing voice, smaller f_o leap intervals between the registers are expected to make it easier to achieve the hypothetical 1 : 1 entrainment (mix) of the registers, allowing the smooth transition of the registers without noticeable pitch jumps or quality change [153,155,161]. Women tend to show smaller leap intervals than men [142,155,158], which could explain the common experience that women singers have less difficulty with smoothening and hiding the register transition. The chest-falsetto leap intervals increase with voice intensity and decrease with increased pitch [159,162,163]. The inter-individual differences in chest-falsetto leap intervals most likely reflect the different eigenfrequencies of the modes involved in the vocal fold oscillation within the chest and falsetto registers, which are sensitive to individual mechanical properties of the vocal folds. We expect the smoothing of the chest-falsetto transitions to be related to entrainment of the eigenmodes involved in both chest and falsetto registers to identical eigenfrequencies. Laryngeal, vocal tract, as well as pressure adjustments can, in principle, be used for this purpose; the exact mechanisms to achieve this are still the subject of research, however.

6. Subharmonic and chaotic oscillation

As discussed above, subharmonic and chaotic oscillations of the vocal folds can be induced by excessively strong coupling strength by airflow, contact or vocal tract interaction. The critical pressure and flow causing irregular vocal fold oscillations by flow coupling are recognized as the 'phonation instability pressure' and 'phonation instability flow', respectively [164]. An *in vivo* case study by Švec & Pešák [162] indicated a link between subharmonic phonations and chest-falsetto register discontinuities: they showed that chest-falsetto jumps with 3 : 2 f_o ratio, evoked by increasing airflow at intermediate and high pitches, transform into subharmonic phonations corresponding to the 3 : 2 attractor (recall figure 3) at low pitches. A similar link between chest-falsetto leap intervals and subharmonic attractors has been observed in spectrograms at intermediate states between chest-falsetto jumps [149,155]. An example of such intermediate subharmonic state during chest-falsetto transition can be observed in figure 5. This suggests that such phenomena stem from the same origin, most likely related to the excitation of the same vocal fold eigenmodes in these phonations [161,165]. In principle, since vocal fold eigenmodes behave as coupled

oscillators within the vocal fold tissues, their entrainment and desynchronization can lead to various types of subharmonic and irregular vocal fold oscillations, as illustrated in figure 3.

Subharmonic and chaotic oscillations may alternatively occur also owing to the interaction of the vocal folds with supraglottal structures, i.e. the ventricular and aryepiglottic folds. A recent simulation study of Inoue *et al*. [166] has nicely demonstrated how the ventricular folds can be engaged to vibrate together with the vocal folds by increasing the coupling strength through a smooth increase in phonatory airflow, and how they produce a bifurcation cascade from nearly-regular to subharmonic and chaotic oscillations. The natural oscillatory frequencies of the ventricular and aryepiglottic folds differ and are typically considerably lower than those of the vocal folds [57,167]. Therefore, when they are brought into oscillation together with the vocal folds, their coupled oscillations will entrain at frequency ratios different from 1 : 1 (recall figure 3). *In vivo* investigations reveal that the ventricular-vocal oscillations go often to a 1 : 2 synchronization: the ventricular fold completes one vibratory cycle while the vocal folds complete two. This 1 : 2 entrainment has been explored, for example, in kargyraa throat singing [19,20]. Also 1 : 3 entrainment and chaotic behaviour have been observed between the ventricular and vocal folds *in vivo* [20,168–170]. Entrainment ratios of 1 : 4 to 1 : 7, along with chaotic oscillations, were reported between the tissues of the aryepiglottic complex and the vocal folds in metal singing [22].

7. Clinical manipulation of coupling strength in left–right laryngeal asymmetry

Human larynges are usually more or less asymmetric in structure [171–173]. Therefore, the left and right vocal folds usually have slightly different *in vacuo* eigenfrequencies. However, because the degree of this natural left–right asymmetry is normally small, the two vocal folds are often entrained to vibrate at the same fundamental frequency owing to the coupling, mostly by airflow but also by contact when present [174,175] (recall the 1:1 attractor in figure 3). The voice often sounds normal, but the natural asymmetry may manifest as slight left–right oscillatory amplitude and phase differences [176–179].

Larger degrees of asymmetry between the two folds may arise owing to pathology such as unilateral paresis or paralysis. Such conditions often lead to large left–right differences in vocal fold tension, stiffness and geometry, accompanied by limited ability to bring the vocal folds together, which reduces the flow coupling strength (see a detailed discussion in Zhang & Luu [180]). As a result, higher subglottal pressure is often required to initiate phonation. With reduced coupling, the two vocal folds may vibrate at different frequencies, resulting in biphonation (unsynchronized vibrations) or subharmonic voice, corresponding to, for example, 2 : 3 or 3 : 4 entrainment of the left–right oscillatory frequencies [62,105,179,181]. In large left–right asymmetry, the paralysed vocal fold may also vibrate with large amplitude, whereas the normal fold is barely excited [174,180].

Clinically, interventions in patients suffering from unilateral vocal fold paresis or paralysis mainly aim to improve coupling between the vocal folds. Voice therapy can help in some cases [182–184]. Further improvement in coupling can be achieved surgically by either injection laryngoplasty or medialization laryngoplasty, in which injectables or implants are used to medialize the affected vocal fold toward glottal midline [185–188]. When a posterior glottal gap is present, arytenoid adduction procedures through sutures may also be performed to close the posterior glottal gap [189–194]. These procedures often partially restore coupling between the two vocal folds by airflow and contact, so that the 1 : 1 entrainment can be achieved, and significantly improve voice outcomes after surgery. Unilateral paralysis, however, often results in the affected side being thinner and softer than the normal side, which often limits post-intervention voice improvement [195]. Thus, intervention may also aim to increase the vertical thickness in the affected fold by, for example, directing the laryngoplasty implant toward the inferior portion of the vocal fold medial surface during implant insertion [196]. This often further improves vocal fold contact and coupling strength between the two vocal folds, and significantly improves the voice outcomes in patients whose voice otherwise would remain unsatisfactory or suboptimal after initial intervention [197].

8. Conclusion

The most crucial insight gained from the theory of nonlinear dynamics is that small changes in phonatory adjustments can cause big changes in the vibratory behaviour of the vocal apparatus and in the produced voice characteristics. Such bifurcations may present themselves as, e.g., jumps from regular oscillations to subharmonic and irregular oscillations, register breaks, or intermittent aphonic breaks [87]. Clinically, it suggests that a relatively small pathological change (e.g. tissue infections, lesions, neural impairments, etc.) can have a profound influence on the phonatory function. It is worth noting that the nonlinear factors can be beneficial as well as detrimental to the voice.

While the nonlinear dynamics theory provides an elegant theoretical framework for understanding different voice phenomena, our current understanding of the nonlinear dynamic mechanisms still remains qualitative, largely based on insights learned from simplified lumped-element models of the vocal folds, which are often not easily translatable to human phonation. This has limited the application of the nonlinear dynamics theory to practical problems, particularly in the clinic. Considering the crucial role played by vocal fold eigenmodes and their synchronization, future studies should aim to establish a more quantitative understanding of the eigenmode synchronization process, including interactions with sub- and supra-glottal acoustics, as well as its manipulation by muscular activities or clinical intervention in more realistic phonation models. Such an improved, quantitative understanding may facilitate translation to practical applications and improve clinical management of voice disorders, as well as help to design more targeted singing voice exercises in voice pedagogy.

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Authors' contributions. J.G.Š.: conceptualization, formal analysis, funding acquisition, investigation, methodology, project administration, resources, visualization, writing—original draft, writing—review and editing; Z.Z.: conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, resources, software, validation, visualization, writing—review and editing.

Both authors gave final approval for publication and agreed to be held accountable for the work performed therein.

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