About the voice production mechanism of cat purring – a critical appraisal of Remmers & Gautier, 1972

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Introduction

Most mammals produce their vocalizations according to the MyoElastic-AeroDynamic (MEAD) principle [4, 25, 13], through passive, self-sustaining oscillation of the vocal folds. The MEAD principle suggests that the emerging fundamental frequency (f_o) correlates roughly with vocal fold length [18, 29, 11]. Considering the data reported by Dunn *et al.* for a number of mammals [8, Fig. 4B], this relation can by expressed with a simple linear model as $f_o = 10^{(-1.278 log_{10}(L)+3.757)}$. Assuming a vocal fold length of $L = 0.75$ cm in an adult domestic cat, this would predict a f_o of about 435 Hz, effects of vocal fold tension and subglottal pressure notwithstanding. This value is well within the f_o range of all known cat call types [30] – with the exception of purring, which is an order of magnitude lower in frequency [10, 21] and can therefore not be readily explained by this simple model.

A number of non-MEAD production principles have been suggested for cat purrs, including refuted hemodynamic and diaphragmatic/thoracic mechanisms [10, 21]. Based on a single electromyographic (EMG) study [17], the current scientific belief is that cat purrs are created by recurrent contraction and relaxation of intrinsic laryngeal muscles, executed every glottal cycle. This *Active Muscle Contraction* (AMC) mechanism is fundamentally different from the MEAD principle, because both the actual sound generation and the control of f_o are established neurally, rather than through the physical principle of passive, self-sustained oscillation based on aerodynamics and laryngeal tissue biomechanics.

Surprisingly, only the work by Remmers & Gautier considered empirical evidence of the actual voice production phenomenon on a deeper biomechanical and aerodynamic level [17], while other previous

contributions were predominantly concerned with the system's output, i.e., the acoustic signal. For that reason, the work by Remmers & Gautier is the key evidence for the current physiological and physical understanding of the purring mechanism. While it has been quoted in later studies, the proposed voice production explanation has never been challenged or critically appraised. There are, however, a number of potential issues with that study. These are discussed here as follows:

1 Potential data artifacts

1.1 Potential issues of electromyographic (EMG) data:

The debate of AMC vs. MEAD is not new. In the 1950s, Husson proposed the so-called neurochronaxic theory of voice production [14], according to which voice production in human speech and singing would be created through AMC. This theory was rightly refuted in favor of MEAD [32, 20], foremost by van den Berg's excised larynx experiments [4]. Husson and coworkers crucially based their argumentation on EMG evidence, which prompted van den Berg and Dedo & Dunker to investigate and document potential pitfalls when acquiring EMG signals [7]. These latter authors showed that mechanical movements of the investigated structures can, without neural stimulation, develop artifactual EMG signals up to 100 μV. In particular, Dedo & Dunker identified signals which "*were caused by mechanical vocal-fold jarring of the electrode rather than by active contraction of the muscle fibres*" [7, p. 309]. Given that Remmers & Gautier only provide uncalibrated EMG data [17] (see Figure 1 for a graphical reproduction of these data), similar artifactual phenomena can not be ruled out in their study. In this context, their statement that posterior laryngeal EMG "*displayed activity which was entirely similar to that seen in the anterior laryngeal recording*" can be interpreted towards the potentially spurious nature of their data, because the mechanical vocal fold motion could have potentially caused artifacts in both the "posterior" and "anterior" EMG data.

It is also possible that the periodic EMG signal that Remmers & Gautier recorded reflects not periodic central stimulation (as they assume), but a peripheral, reflex activation of the muscle due to deformation of stretch receptors (e.g. muscle spindles or Golgi tendon organs). Direct evidence supporting this possibility comes from van den Berg [31], who found a "laryngeal microphonic" signal, caused by deformation of the intrinsic laryngeal musculature (and not centrally generated). Less direct evidence supporting an afferent origin of these periodic EMG signals comes from Kirkwood et al [15], who recorded periodic EMG signals from the cat intercostal muscles during purring, but found that these were only transitorily present, and could be rendered asymmetric with either postural adjustment or by cutting the sensory nerves of these muscles. These results argue against the idea that the periodic EMG signals result from central nervous stimulation, and instead suggest that respiratory gating by the glottis propagates pressure pulses back into the lungs (Frazer-Sissom), eliciting EMG signals due to passive deformation of the stretch receptors in the intercostal muscles by the acoustically generated signal.

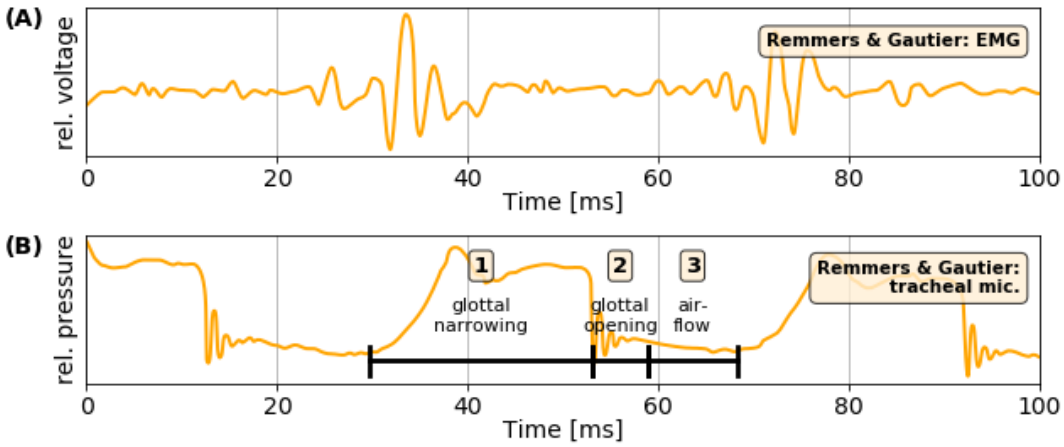

Figure 1: Reproduction of Figure 6 by Remmers & Gautier [17], data for egressive purring. (A) anterior larygeal (cricothyroid muscle) EMG; (B) tracheal pressure. Remmers & Gautier's interpretation of their data with respect to glottal narrowing (1), glottal opening and sound production (2), and airflow (3) is superimposed upon the tracheal pressure signal.

1.2 Potential issues of subglottal (tracheal) pressure measurement:

Remmers & Gautier built their model for AMC sound generation upon the acquired subglottal pressure signal. They state that their subglottal pressure sensor was inserted as reported by Scotto & Naitove [23]. According to those latter authors, the tracheal tube was inserted upwards obliquely, facing away from the tracheal air stream [23, Fig. 1]. However, if the pressure probe is not inserted precisely perpendicular to the exhalatory airstream, the acquired signal will likely contain spurious information introduced by the exhalatory air stream [2]. Remmers & Gautier's pressure signal should thus be interpreted with care. Improper pressure probe placement could potentially explain the apparent discrepancies between the subglottal pressure signal from Remmers & Gautier and those from authors documenting (human) voice production according to MEAD [6, 22, 24, 25, 16]. According to these latter sources and in contrast to the evidence from Remmers & Gautier there is no buildup of subglottal pressure during the closed phase until the vocal fold separate. Rather, there is a short negative pressure peak at the incident of glottal closure (i.e., the termination of the glottal air flow at the end of the open phase, constituting the main acoustic excitation event according to MEAD), which introduces damped acoustic oscillations in the subglottal tract caused by subglottal cavity resonances.

2 Issues concerning the physical sound generation principle

Remmers & Gautier based their model of physical sound production mostly on their interpretation of the tracheal pressure data, because no time-synchronized visual evidence of the glottis during purring was produced. They claim that "*the purr results from active glottal closure, consequent development of trans-glottal pressure, and then its dissipation by sudden separation of the vocal cords.*" They further state that the "*sound burst begins coincidentally with the abrupt pressure wave (fig. 8). Such*

a rapid pressure transient suggests an explosive dissipation of trans-glottal pressure, presumably the result of a sudden separation of the vocal cords ..." [17]

This puts the glottal excitation event in their model at the incident of glottal opening (see Figures 6, 7, and 8 in [17]). We believe that this might possibly be a mis-interpretation of the data. The subglottal pressure signals in Figures 6, 7, and 8 of [17] clearly show an exponentially decaying sinusoid immediately after the acoustic excitation event, during what they termed "phase 2 (glottal opening)" of the oscillation – see Figure 1B. Interpolating from the indicated time scale, this sinusoid has a frequency of about 830 Hz.

The trachea from the *carina* to the vocal folds can be modeled as a quarter wave resonator [27] whose resonances are approximately found at frequencies of

$$f_n = \frac{1}{2n-1} \times \frac{c}{4L} \; [Hz] \tag{1}$$

For the first resonance ($n = 1$), this equation reduces to $f = \frac{c}{4L}$. Solving for L gives $L = \frac{c}{4f}$. Assuming a speed of sound of $c = 340 \; \frac{m}{s}$ and setting f to 830 Hz, the estimated tracheal length is L = 102 mm, which is on the lower end of the values indicated for adult female domestic cats by Zimmermann et al. [33]. It is therefore conceivable that the decaying sinusoid found in the subglottal pressure data produced by Remmers & Gautier (recall Figure 1B) is actually brought about by the lowest resonance of the subglottal (tracheal) vocal tract. However, this assumption would only hold for a closed glottis with vocal folds in contact, because (a) a closed end is needed at the glottis to establish a quarter wave resonator with that particular length (an open glottis would change the effective length of the resonator into the supraglottal vocal tract [3]). Consequently, the glottis was most likely closed during the occurrence of the exponentially decaying oscillation at 830 Hz. Furthermore, (b) with an open glottis the acoustic loading of the resonance would dissipate much quicker, because the sound would be lost in the supraglottal vocal tract. This suggests that the acoustic excitation pulse shown Figures 6, 7, and 8 of [17] actually occurs at the incident of glottal closure, just as predicted by standard MEAD theory, and not at the incident of glottal opening (as suggested by Remmers & Gautier).

Glottal opening is likely facilitated by tracheal driving pressures and the respective volumetric air flow, just as Remmers & Gautier surmise. Morevoer, it is known from the MyoElastic-AeroDynamic (MEAD) theory that glottal closure is facilitated by the following phenomena: (a) passive recoil forces found within the vocal fold tissue; (b) a negative intraglottal pressure caused by a divergent vocal fold shape profile during the closing phase; (c) Bernoulli forces; and (d) potentially an inert supraglottal air column that helps to create an asymmetric forcing function [4, 26, 28, 13]. It is thus unclear why, on top of these four different physical force phenomena, glottal closure would have to be facilitated through muscular activity occurring every glottal cycle.

According to Remmers & Gautier, the EMG activity does not occur at the moment of acoustic excitation (see Figures 6, 7, and 8 in [17]). Therefore, crucially, active muscular contraction is not directly causal to sound generation even in their model. When following the argumentation presented here, i.e., that Remmers & Gautier mis-intepreted their subglottal pressure signals, and that the acoustic

excitation event *in vivo* actually occurs at the moment of glottal closure (and thus cessation of air flow, just as predicted by the MEAD theory), it is thus unclear why the EMG activity is necessary to establish the vocalization phenomenon.

3 Issues concerning voice production physiology

3.1 The cricothyroid (CT) muscle does not primarily affect vocal fold adduction:

Remmers & Gautier build their case on their main bulk of evidence from EMG probe insertion into the cricothyroid (CT) muscle, and partly on additional EMG probe insertion into unspecified muscles of the "posterior larynx" in two cats.[1] In particular, all EMG traces shown in [17] (with the exception of Figure 4) depicted the "anterior laryngeal EMG", and thus the data coming from the probe inserted into the CT muscle. However, considering the functional similarity found within mammalian larynges [12], the CT muscle is not primarily an adductor of the vocal folds. Rather, the CT muscle is a tensor of the vocal folds, in MEAD mainly responsible for elongating the vocal folds [5] and thus controlling the frequency of vocal fold vibration [27]. As a secondary function at least in humans and dogs, the CT has been found to have a partially adductive function (to max. 40 – 50 % of full adduction) [1], but not resulting in glottal closure without the simultaneous contraction of the LCA and/or IA muscles. Rather, full contraction of the CT is likely to abduct the vocal folds (thus opening the glottis) [5, 1]. Activity in the CT muscle alone would therefore certainly not facilitate glottal closure, as is implicitly assumed by Remmers & Gautier, who do not distinguish between the individual intrinsic laryngeal muscles in their discussion.

3.2 Innervation issues:

Remmers & Gautier found that purring does not stop if the superior laryngeal nerve is severed. Thus, the CT muscle (from which they derive their main EMG evidence) is not needed for purring. However, impairment of the superior laryngeal nerve also does not prevent voice production according to the MEAD theory, as can be seen in human patients with superior laryngeal nerve paralysis [9]. Therefore, the notion that purring does not stop when the superior laryngeal nerve is severed does not necessarily support the AMC principle.

Furthermore, Remmers & Gautier found that purring stops if the recurrent laryngeal nerve is severed. The recurrent laryngeal nerve supplies both the vocal fold adductors (lateral cricoarytenoid muscle, LCA, and the interarytenoid muscles, IA) and the vocal fold abductors (posterior cricoarytenoid muscles, PCA). In humans (and thus MEAD-based voice production), impairment of the recurrent laryngeal nerve can also lead to dysphonia, because then the larynx can not be properly re-configured from

[1] "*In all cats, electrodes were implanted under direct vision in the cricothyroid muscle*" and "*In addition, electrodes were attached to the posterior larynx at the cranial margin of the cricoid cartilage in two cats.*" Remmers and Gautier [17, p.352]

the breathing configuration to the voice production configuration [19]. For this reason, also Remmers & Gautier's argument that disruption of the recurrent laryngeal nerve stops the purring production is not a compelling argument for the AMC production principle, because the same effect could have happened if the purring were driven by MEAD.

Conclusion

Based on the argumentation presented here, there is reason to believe that the physical and physiological aspects of the cat purring mechanism might be different than what has previously been proposed by Remmers & Gautier [17]. Further research *in vivo* is required, in order to establish an empirically anchored model that describes the causal phenomena and physical forces which induce glottal closure, glottal opening, and actual sound generation.

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