Metabolic Mechanisms of Vocal Fatigue

***Chayadevie Nanjundeswaran, †‡Jessie VanSwearingen, and §‖¶#**Katherine Verdolini Abbott,** **Johnson City, Tennessee, and †‡§‖¶#**Pittsburgh, Pennsylvania*

Summary: Objective. This study aimed to identify potential metabolic mechanisms including (1) neuromuscular inefficiency, (2) cardiovascular recovery deficits, or (3) both, in individuals with complaints of vocal fatigue.
Study Design. Within- and between-subjects group design was used in this study.
Methods. Three groups of women participated in the study, including (1) individuals with complaints of vocal fatigue; (2) vocally healthy sedentary individuals; and (3) vocally healthy, cardiovascularly conditioned individuals. Group assignment was based on results from the Vocal Fatigue Index, laryngeal examination, and self-report regarding exercise regimens. Metabolic profiles were obtained using gas exchange measures monitored during vocal task performance (reading) at two different loudness levels, and during recovery from reading.
Results. Statistical analyses did not reveal reliable group differences in metabolic cost for or recovery from vocal tasks. However, descriptive review of oxygen uptake and recovery kinetics revealed patterns indicating reliance on differential energy resources for the vocal task in individuals with vocal fatigue compared with cardiovascularly trained, vocally healthy individuals in particular. Slow oxygen uptake kinetics at task onset was a characteristic of the vocal fatigue group, indicating a general reliance on anaerobic resources to meet the demands of the vocal task, pointing to possible neuromuscular inefficiency. Individuals with vocal fatigue also demonstrated an increase in oxygen consumption following vocal task compared with cardiovascularly trained individuals, suggesting possible cardiovascular recovery deficits.
Conclusion. This study provides initial data relevant to possible metabolic mechanisms of vocal fatigue and the potential relevance of aerobic conditioning in individuals with such fatigue.
Key Words: Vocal fatigue–Gas exchange–Neuromuscular inefficiency–Aerobic conditioning–Oxygen uptake kinetics.

INTRODUCTION

Vocal fatigue is a frequently occurring, often debilitating condition, affecting many individuals with voice problems. Professional and other voice users may be susceptible to this fatigue, which may affect social and occupational functioning.[1–3] Prior research has attempted to identify mechanisms underlying vocal fatigue and possible treatment options for it. However, interpretation of results has been complicated by the lack of a consistent definition of vocal fatigue itself and by the use of different study methodologies including (1) study of individuals with and without vocal complaints,[1,4–6] (2) study of voice changes following prolonged voice use,[7–11] (3) study of treatment outcomes for individuals with complaints of vocal fatigue,[2,12–14] and (4) study of recovery from vocal loading in healthy individuals.[15] Moreover, existing data have failed to reveal a robust relationship across self-reported and instrumented measures of vocal fatigue, thereby providing minimal information about mechanisms that may underlie this phenomenon. In sum, mixed and inconclusive results in the vocal fatigue literature are attributable to uncertainties about the definition of vocal fatigue, variations in the choice of subject populations across studies, the use of varying instrumented and self-report measures that may not be sensitive to capture this variegated phenomenon, and the use of different loading tasks that may or may not elicit vocal fatigue.[16] Given the debilitating nature and prevalence of this condition,[2,3] it is imperative to conduct further probes to understand the underlying mechanisms and identify effective treatment options.

In contrast to the situation in the literature *on vocal* fatigue, research on fatigue in the exercise physiology domain has been more extensive. Fatigue has been investigated under three schools of thought: schools that emphasize peripheral fatigue, those that emphasize central fatigue, and those that emphasize a combination of peripheral and central mechanisms. In each school of thought, fatigue has been variably defined and different causes of fatigue have been proposed.[17–25] Full discussion is beyond the scope of this paper. However, in brief, peripheral fatigue is thought to result from factors relating to the neuromuscular junction and muscle function itself, including factors pertaining to the intracellular environment and muscle fibers.[17,21,22] In contrast, central fatigue is associated with reduced recruitment of motor units, poor coordination of recruited motor units, and reduced central drive due to increased inhibitory input to the motor cortex.[25,26] The third school of thought emphasizes the interplay between central and peripheral mechanisms.[18,24,27] Among hypotheses, a common proposal is that in both peripheral and central fatigue, the oxygen supply to the muscle and the brain is inadequate for the task demands.[28] In sum, oxygen is needed to meet the energy demands of active tissues during physical activity or task

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From the *Department of Audiology and Speech-Language Pathology, East Tennessee State University, Johnson City, Tennessee; †Department of Physical Therapy, University of Pittsburgh, Pittsburgh, Pennsylvania; ‡School of Nursing, University of Pittsburgh, Pittsburgh, Pennsylvania; §Department of Communication Sciences and Disorders, University of Pittsburgh, Pittsburgh, Pennsylvania; ‖Department of Otolaryngology, University of Pittsburgh, Pittsburgh, Pennsylvania; ¶McGowan Institute for Regenerative Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania; #Center for the Neural Basis of Cognition, Carnegie-Mellon University, Pittsburgh, Pennsylvania; and the **Center for the Neural Basis of Cognition, University of Pittsburgh, Pittsburgh, Pennsylvania.
Address correspondence and reprint requests to Chayadevie Nanjundeswaran, Department of Audiology and Speech-Language Pathology, College of Clinical and Rehabilitative Health Sciences, East Tennessee State University, Lamb Hall, Box 70643, Johnson City, TN 37614. E-mail: nanjundeswar@etsu.edu
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TABLE 1.
Oxygen Supply Issues in Fatigue

- Oxygen supply may be altered or inadequate due to inadequate delivery of oxygen to the working muscles (related to cardiac output and circulation issues), and extraction of oxygen from blood delivered to the working muscle may be inadequate to support the physical activity—the opposite of endurance,[25]
- There can be increased reliance on anaerobic resources during task performance due to a delay in the timing of the oxygen supply to meet task demands. The result is a depletion of the local (muscle) anaerobic energy resources (PCr, glycogen) followed by excessive metabolite accumulation (eg, lactic acid, hydrogen ions, and calcium ions), the by-products of anaerobic metabolism. The accumulation of these metabolites impacts both muscle physiology and neuromuscular function, which in turn can contribute to a decline in force production or inefficient patterns of muscle recruitment for a task.[25,26] (Note that in the presence of oxygen, some of the accumulated metabolites can be used as fuel in aerobic metabolism; for information about the Krebs cycle, see Ref. 33),
- Inadequate supply of oxygen for the energy demand influences inhibitory feedback to the CNS and may contribute to the perception of effort in task performance,[23]
- Endurance exercise, which is known to enhance oxygen supply (due to improved cardiac output and oxygen extraction), has been shown to delay the onset of fatigue, presumably due to the provision of adequate oxygen to meet task demands earlier in the onset of performance, and thus the reduction or prevention of the accumulation of excessive metabolites.[27,28]

CNS, central nervous system; PCr, creatine phosphate.

performance. The desired energy source for this activity is aerobic metabolism, which is dependent on an adequate and consistent supply of oxygen to the active tissues. With fatigue there may be a lag in availability or poor extraction of oxygen to meet task demands. When oxygen supply is insufficient, task-related fatigue—defined as performance decrements over time—may ensue.

Despite the desired use of aerobic metabolism to meet task-related energy demands, at the onset of physical activity, there is an initial reliance on anaerobic metabolism as an energy source. Specifically at the onset of activity, reliance is on local tissue stores of creatine phosphate and glycogen in the muscle to meet task demands. These fuel sources can independently support performance for a few minutes only. Continued performance is sustained by aerobic metabolism, which is dependent on an adequate supply of oxygen to the tissues, as oxygen is the fuel needed for aerobic metabolism (ie, aerobic resources). Mismatch between oxygen supply to the active tissues and energy demands can lead to performance decrements, that is, fatigue[23,29–32] A summary of mechanisms related to oxygen supply in fatigue is provided in Table 1.

Relative to oxygen supply, two specific potential mechanisms may explain fatigue: (1) neuromuscular inefficiency, and (2) cardiovascular recovery deficits. Neuromuscular efficiency refers to the "how" of performance, specifically the degree to which a task is performed using a minimal amount of muscle activation. Optimal efficiency occurs when only those muscles necessary for task completion are used, with appropriate timing and coordination across muscles.[19,34] Inefficiency, involving the recruitment of more muscles than needed for task performance or "incorrect pattern" of muscle activation, will lead to greater energy demands for task performance. On the other hand, cardiovascular recovery following physical activity refers to the extent and the time course within which physiological functions return to the baseline homeostatic state. This recovery is generally enhanced in aerobically trained individuals.[35] Specifically, aerobic (endurance exercise) training is associated with a rapid regulation of postexercise metabolism and the recovery to homeostasis.[35–37] In addition, training can also improve delivery of oxygen to the active tissues, thus a reduction in the "lag" in aerobic resources at activity onset, which subsequently impacts the magnitude of postexercise recovery.

Appreciation of these two possible mechanisms in fatigue is enhanced by understanding of oxygen uptake kinetics and recovery kinetics. The dynamic behavior of oxygen uptake in the transition from rest to exercise is termed oxygen kinetics and is dependent on both central circulation (i.e., delivery of oxygen to working muscles) and peripheral extraction (i.e., ability of working muscles to use the available oxygen). Two further relevant concepts have to do with (1) oxygen deficit and (2) excess postexercise oxygen consumption (EPOC).[38,39] At the onset of any task, anaerobic resources in the muscle are broken down until aerobic resources come into play, observed as an achievement of steady state in oxygen consumption to meet task demands (Figure 1). The state that precedes the achievement of oxygen steady state, when there is a lag in the rate of oxygen consumption (i.e., inadequate oxygen supply) relative to task demands, is termed the state of oxygen deficit.[33,40] This deficit is greater in aerobically untrained individuals than in aerobically fit individuals, in part because untrained individuals do not have efficient oxygen delivery and extraction to meet task demands.[36] In turn, greater oxygen deficit is correlated with the increased reliance on anaerobic resources for task performance. Anaerobic resources are by nature limited and thus may be a factor in fatigue, understood as a decline in the ability to sustain an activity. Additionally, and related to our previous discussion of neuromuscular efficiency, the activation of muscles irrelevant to task performance, or activation of muscles at a level that exceeds task demands, may further contribute to fatigue. The slower oxygen uptake at the onset of task delays the achievement of oxygen steady state, in turn contributing to an early onset of fatigue.[41–43] This partial or total dependence on anaerobic resources