Thickness and Histologic and Histochemical Properties of the Superior Pharyngeal Constrictor Muscle in Velocardiofacial Syndrome

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Background: Velocardiofacial syndrome (VCFS) is one of the most common multiple anomaly syndromes in humans. Pharyngeal hypotonia, one of the most common findings in VCFS, contributes to hypernasal speech, which occurs in approximately 75% of individuals with VCFS.

Objective: To evaluate the thickness and histologic and histochemical properties of the superior pharyngeal constrictor (SPC) muscle in patients with VCFS to determine whether a muscle abnormality exists that might contribute to the hypotonia seen in these patients.

Subjects: The SPC muscle thickness in 26 VCFS patients (18 male and 8 female; age range, 3-29 years) was compared with SPC muscle thickness in age- and sex-matched controls using magnetic resonance images. The histologic and histochemical properties of the SPC muscle in 9 VCFS patients (6 male and 3 female; age range, 4-12 years) were compared with SPC muscle in 3 adult cadavers without VCFS (all male; age range, 80-86 years) using specimens obtained during pharyngeal flap surgery.

Results: The thickness of the SPC muscle was significantly less in patients with VCFS (2.03 mm) than in patients without VCFS (2.85 mm). The SPC muscle contained a significantly greater proportion of type 1 fibers in patients with VCFS (27.7%) than in adults without VCFS (17.9%), and the diameter of the type 1 fibers was significantly smaller in patients with VCFS (21.6 µm) than in adults without VCFS (26.6 µm).

Conclusions: Differences in the thickness and histologic and histochemical properties of the SPC muscle found in patients with VCFS compared with individuals without VCFS may offer insight into the cause of pharyngeal hypotonia and hypernasal speech seen in these patients.

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vical vertebra inferiorly. Motor innervation to the SPC muscle is derived from the pharyngeal plexus (vagus and glossopharyngeal nerves). Golding-Kushner demonstrated a decrease in the thickness of the posterior pharyngeal wall on plain radiographs in patients with VCFS. However, further characterization of the SPC muscle in humans has received little attention. The purpose of this study is to evaluate the thickness, histology, and histochemistry of the SPC muscle in patients with VCFS to determine whether a muscle irregularity exists that might contribute to the hypotonia seen in these patients.

METHODS

MAGNETIC RESONANCE IMAGING

The thickness of the SPC muscle in 26 patients with VCFS (18 male and 8 female; age range, 3-29 years) and 26 patients without VCFS (18 male and 8 female; age range, 3-27 years) was determined using T1- and T2-weighted cervical spine and brain magnetic resonance images (MRIs). Patients without VCFS did not have a primary myopathy. Using computer software (eFilm 1.5 Medical Imaging System; eFilm Medical Inc, Milwaukee, Wis), measurements were taken in the midsagittal plane at the following 3 levels for each patient: the level of the first cervical vertebra, the midpoint of the second cervical vertebra, and the inferior aspect of the second cervical vertebra (Figure 1). The average of the 3 measurements was taken as the thickness of the muscle.

MUSCLE SPECIMENS

A biopsy specimen of the SPC muscle at the midline of the posterior pharyngeal wall was obtained during routine superior pharyngeal flap surgery in 9 children (3 female and 6 male; age range, 4-12 years) with VCFS. All patients provided informed consent to undergo biopsies, which were approved by the Institutional Review Board at the State University of New York Upstate Medical University, New York. Biopsy specimens were also obtained from 3 adult cadavers (all male; age range, 80-86 years) within 24 hours post mortem before embalming. The biopsy specimens were oriented for serial transverse sectioning, mounted on slices of cork in OTC compound (optimal temperature cutting compound) (Tissue Tek; Miles Laboratories, Naperville, Ill), and frozen in liquid methyl-butane chilled with liquid nitrogen. The specimens were stored at −70°C until sectioning, at which time they were cut into 5-µm sections in a cryostat at −20°C.

General histologic analysis was performed after staining the specimens with modified Gomori trichrome, described as follows. The specimens were placed in hematoxylin at room temperature for 5 minutes, rinsed in distilled water, stained in modified Gomori trichrome at room temperature for 10 minutes, rinsed in 0.2% acetic acid, dehydrated with alcohol, cleared with xylene, and mounted. After mounting, the sections were examined using an optical microscope magnified ×40.
Oxidative activity was demonstrated by staining the specimens with reduced nicotinamide adenine dinucleotide (NADH), described as follows. The specimens were incubated for 30 minutes in a solution containing NADH, nitro blue tetrazolium, and trizma base–hydrochloric acid buffer at 37°C. They were then rinsed in distilled water, 30% acetone, 60% acetone, 30% acetone again, and distilled water again, dehydrated with alcohol, cleared with xylene, and mounted. After mounting, the sections were examined using an optical microscope magnified at ×40.

Enzyme-histochemical analysis was performed after staining the specimens for demonstration of myofibrillar adenosine triphosphatase (ATPase) activity, described as follows. For each patient, some sections were preincubated in calcium chloride–sodium barbital buffer at pH 9.4 for 15 minutes at room temperature, and other sections were preincubated in sodium acetate–acetic acid buffer at pH 4.3 for 5 minutes at room temperature. All stained sections were then incubated for 10 minutes at 37°C in a solution with pH 9.4 containing 0.1M sodium barbital, 0.18M calcium chloride, distilled water, and adenosine triphosphate. The sections were then rinsed in calcium chloride, placed in cobalt chloride for 3 minutes, rinsed in distilled water, placed in 1% ammonium sulfide for 3 minutes, rinsed in distilled water again, dehydrated in alcohol, cleared with xylene, and mounted. After mounting, sections were examined using an optical microscope magnified ×40.

Individual muscle fibers were identified and differentiated based on their staining intensity for myofibrillar ATPase under different pH conditions. Two major fiber types (types 1 and 2) can be distinguished in human muscles. Type 1 fibers, which have a large amount of oxidative enzymes, are lightly stained at pH 9.4 and darkly stained at pH 4.3. Type 2 fibers, which have a large amount of ATPase, are darkly stained at pH 9.4 and lightly stained at pH 4.3. (Figure 2 and Figure 3). Percent distribution of type 1 vs type 2 fibers was determined. Finally, measurements of muscle fiber diameter were performed using an optical microscope magnified ×40 with a micrometer scale contained in one eyepiece. For each patient or cadaver, 64 to 113 muscle fibers were counted.

**STATISTICAL ANALYSIS**

Muscle thickness data (MRI) were submitted to a 3-factor, mixed-model analysis of variance (ANOVA), crossing location of measurement (first cervical vertebra, midpoint of the second cervical vertebra, and the inferior aspect of the second cervical vertebra) with sex (male and female) and conditions (VCFS and without VCFS). Given no significant interaction terms involving location of measurement, the 3 measurements were averaged to create a single muscle thickness out-

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**Figure 2.** Cross sections from the superior pharyngeal constrictor muscle of a patient with velocardiofacial syndrome stained for myofibrillar adenosine triphosphatase (ATPase) at pH 4.3 (A), where light-stained muscle fibers correspond to type 2 and dark-stained muscle fibers to type 1, and pH 9.4 (B), where light-stained muscle fibers correspond to type 1 and dark-stained muscle fibers to type 2 (original magnification ×20).

**Figure 3.** Cross sections from the superior pharyngeal constrictor muscle of an adult cadaver without velocardiofacial syndrome stained for myofibrillar adenosine triphosphatase (ATPase) at pH 4.3 (A), where light-stained muscle fibers correspond to type 2 and dark-stained muscle fibers to type 1, and pH 9.4 (B), where light-stained muscle fibers correspond to type 1 and dark-stained muscle fibers to type 2 (original magnification ×20).
come variable for subsequent analyses. Pearson correlation coefficients were used to quantify the relationship between age and average muscle thickness.

Type 1 and type 2 average muscle fiber diameters were submitted to a 2-factor, mixed-model ANOVA, crossing fiber type (type 1 and type 2) with condition (VCFS and without VCFS). Given a significant condition by fiber type interaction term, subsequent 1-way analyses compared type 1 fiber diameters in VCFS patients with type 1 fiber diameters in adult cadavers without VCFS and type 2 fiber diameters in VCFS patients with type 2 fiber diameters in adult cadavers without VCFS. Furthermore, the SDs of the muscle fiber diameters were submitted to a similar ANOVA that crossed fiber type (type 1 and type 2) with condition (VCFS and without VCFS). However, no interaction terms were significant and therefore no additional statistical comparisons were conducted. Percent distribution of type 1 fiber data was analyzed with a 1-way ANOVA comparing patients with VCFS with patients without VCFS.

RESULTS

MAGNETIC RESONANCE IMAGING

Table 1 gives the average thickness of the SPC muscle in patients with and without VCFS, respectively. The overall average muscle thickness was 2.03 mm (SD, 0.49 mm; range, 1.0-3.0 mm) in patients with VCFS and 2.85 mm (SD, 0.30 mm; range, 2.0-3.3 mm) in patients without VCFS. This difference in muscle thickness is statistically significant (P<.001). The analysis revealed a significant linear trend contrast (P<.003), indicating that the muscle was thicker superiorly (at the level of the first cervical vertebra) and thinner inferiorly (at the inferior aspect of the second cervical vertebra), but this trend was similar between patients with and without VCFS (Figure 4). Muscle thickness was positively correlated with age in patients with (P<.001) and without (P=.02) VCFS. No difference in muscle thickness was noted between males and females in patients with or without VCFS (Figure 5).

MUSCLE SPECIMENS

Considerable variability in the diameter and shape of the SPC muscle fibers was noted in all VCFS patients and all adult cadavers without VCFS. The fibers had a rounded rather than polygonal configuration and were relatively loosely packed. Compared with adult cadavers without VCFS, VCFS patients had a greater amount of endomysial space between muscle fibers, and the fibers were more loosely packed. Neither adults without VCFS nor VCFS patients demonstrated any peculiar morphologic features, such as ragged red fibers, central nuclei, fiber splitting, nemaline rods, hyaline fibers, or degenerating fibers. In both VCFS patients and adult cadavers without VCFS, type 1 and 2 muscle fibers showed fairly intense staining for NADH such that it was often difficult to distinguish between the 2 fiber types. In general, type 1 fibers stained slightly more intensely.

Table 2 gives the average muscle fiber diameter and distribution of fiber types in patients with and without VCFS. Respectively. The overall average frequency of type 1 fibers was 27.7% (SD, 2.01%; range, 23.9%-31.3%) in patients with VCFS and 17.9% (SD, 2.15%; range, 15.9%-20.2%) in adult cadavers without VCFS (Figure 6). Conversely, the overall average frequency of type 2 fibers was 72.3% (SD, 1.99%; range, 68.8%-76.1%) in VCFS patients and 82.1% (SD, 2.16%; range, 79.8%-84.1%) in adult cadavers without VCFS.
This difference in distribution of muscle fiber types was statistically significant \((P<.001)\). The average overall diameter of type 1 fibers was 21.6 \(\mu m\) (SD, 2.09 \(\mu m\); range, 18.7-25.4 \(\mu m\)) in VCFS patients and 26.6 \(\mu m\) (SD, 1.41 \(\mu m\); range, 25.3-28.1 \(\mu m\)) in adult cadavers without VCFS. The average overall diameter of type 2 fibers was 26.4 \(\mu m\) (SD, 2.03 \(\mu m\); range, 24.3-29.7 \(\mu m\)) in VCFS patients and 26.7 \(\mu m\) (SD, 0.90 \(\mu m\); range, 25.8-27.6 \(\mu m\)) in adult cadavers without VCFS. The 2-factor ANOVA revealed a significant fiber type by condition interaction \((P<.001)\) term, indicating that VCFS patients and adult cadavers without VCFS differ in type 1 vs type 2 fiber diameter. Subsequent analysis revealed that the difference in the average diameter of type 1 muscle fibers between VCFS patients and adult cadavers without VCFS was statistically significant \((P<.001)\). In patients with VCFS, type 1 fibers were significantly smaller than type 2 fibers (21.6 vs 26.4 \(\mu m\), respectively). There was no difference in the average diameter between type 1 and type 2 fibers in adult cadavers without VCFS (26.6 and 26.7 \(\mu m\), respectively). The average type 2 fiber diameter in VCFS patients was similar to the average type 2 fiber diameter in adult cadavers without VCFS (26.4 and 26.7 \(\mu m\), respectively). Because age and sex were inherent confounders in this study, namely, all VCFS patients were children and all cadavers without VCFS were male adults, no conclusions could be drawn regarding the relationship of age and sex on muscle fiber diameter and distribution of muscle fiber type. Unfortunately, these confounders were necessary due to the limited availability of adult cadavers and the unavailability of child cadavers.

![Figure 4. Average thickness of the superior pharyngeal constrictor muscle measured on magnetic resonance image with respect to location. C1 represents the level of the first cervical vertebra, mid-C2 represents the midpoint of the second cervical vertebra, and low-C2 represents the inferior aspect of the second cervical vertebra. The difference in the muscle thickness between patients with velocardiofacial syndrome (VCFS) and patients without VCFS is statistically significant \((P<.001)\).](image1)

![Figure 5. Average thickness of the superior pharyngeal constrictor muscle measured on magnetic resonance image with respect to sex. There is no statistically significant difference in the muscle thickness between males and females in patients with VCFS and without VCFS.](image2)

This study analyzes the thickness, histology, and histochemistry of the SPC muscle in patients with VCFS. The significant findings include the following: the decreased thickness of the SPC muscle compared with age- and sex-matched controls; the abundance of endomysial space between muscle fibers in VCFS patients; the increased proportion of type 1 muscle fibers in VCFS patients compared with adults without VCFS; and the smaller diameter of the type 1 muscle fibers in VCFS patients compared with adults without VCFS. These results may be significant in elucidating the etiology of the velopharyngeal hypotonia and subsequent hypernasality frequently seen in patients with VCFS. Velopharyngeal competence, which is largely dependent on adequate function of the soft palate muscles and the SPC muscle, is critical in the development of normal speech. Therefore, a deficiency or an irregularity in any of these muscles might contribute to velopharyngeal hypotonia with subsequent incompetence and hypernasality.

Although no specific abnormalities in SPC muscle fiber morphologic features were present in either VCFS patients or adults without VCFS, VCFS patients exhibited significantly more endomyssial space between muscle fibers, resulting in exceedingly loosely packed muscle fibers compared with adults without VCFS. This histologic finding and the atrophy of the type 1 muscle fibers may correspond to the significantly decreased SPC muscle thickness in VCFS patients seen on MRIs.

It is well known that the fiber composition of a muscle strongly influences its functional properties. A high proportion of type 1 fibers typically indicates slow contraction with high resistance to fatigue, whereas a high proportion of type 2 fibers typically indicates fast contraction with low resistance to fatigue.\(^2\) In this study, the SPC muscle was found to contain a much higher proportion of type 2 muscle fibers than type 1 fibers in patients with VCFS and adult cadavers without VCFS. This finding suggests that the SPC muscle possesses fast contraction properties with large, high threshold motor units not well outfitted for finely graded movements. The lit-
literature contains a paucity of information regarding the histology and histochemistry of the soft palate muscles and the SPC muscle in humans. Leese and Hopwood found that the mean percentage of type 1 muscle fibers for the pharyngeal constrictor was 32.5% and that the fiber diameter was approximately 30 µm, but they did not specify which pharyngeal constrictor muscle was examined. They also found that there was little difference in fiber type distribution or fiber diameter with respect to age or sex. Stal and Lindman characterized the muscles of the soft palate in 5 healthy adults and found that the palatopharyngeus and uvula contained some of the highest proportions of type 2 fibers ever reported for human muscles (86.2% and 87.2%, respectively), whereas type 1 fibers predominated in the levator and tensor veli palatini muscles (74.9% and 57.0%, respectively). Moon and colleagues determined that the levator veli palatini muscle in 12 healthy adults contained an average of 59.8% type 1 muscle fibers. Lindman and colleagues compared the levator veli palatini muscle in 5 healthy adults with 8 infants with cleft palate. They discovered that infants with cleft palate had a smaller mean fiber diameter, a larger variability in fiber size and form, and a higher proportion of type 2 fibers.

In this study, patients with VCFS were found to have a significantly higher proportion of type 1 muscle fibers than adults without VCFS. Previous studies report relatively stable fiber-type distributions across ages ranging from young adulthood to 100 years. Thus, this differ-

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**Table 2. Average Muscle Fiber Diameter and Distribution of Fiber Type in the Superior Pharyngeal Constrictor Muscle in Patients With and Adult Cadavers Without Velocardiofacial Syndrome**

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Type 1 Muscle Fibers</th>
<th>Type 2 Muscle Fibers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% (SD) Diameter, µm</td>
<td>% (SD) Diameter, µm</td>
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<tr>
<td></td>
<td></td>
<td></td>
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<tr>
<td><strong>With Velocardiofacial Syndrome</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/F/5</td>
<td>23.9</td>
<td>22.0 (2.9)</td>
</tr>
<tr>
<td>2/M/7</td>
<td>27.2</td>
<td>18.7 (4.9)</td>
</tr>
<tr>
<td>3/F/6</td>
<td>28.6</td>
<td>21.0 (7.7)</td>
</tr>
<tr>
<td>4/M/7</td>
<td>27.4</td>
<td>22.2 (3.4)</td>
</tr>
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<td>5/M/4</td>
<td>26.5</td>
<td>22.6 (4.3)</td>
</tr>
<tr>
<td>6/M/5</td>
<td>27.2</td>
<td>19.5 (3.1)</td>
</tr>
<tr>
<td>7/F/12</td>
<td>28.7</td>
<td>25.4 (5.4)</td>
</tr>
<tr>
<td>8/M/8</td>
<td>28.8</td>
<td>23.2 (4.7)</td>
</tr>
<tr>
<td>9/M/6</td>
<td>31.3</td>
<td>19.8 (2.7)</td>
</tr>
<tr>
<td><strong>Overall average</strong></td>
<td>6.6</td>
<td>27.7</td>
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<tr>
<td><strong>Without Velocardiofacial Syndrome</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10/M/86</td>
<td>17.7</td>
<td>28.1 (4.0)</td>
</tr>
<tr>
<td>11/M/80</td>
<td>15.9</td>
<td>25.3 (4.6)</td>
</tr>
<tr>
<td>12/M/81</td>
<td>20.2</td>
<td>26.4 (4.1)</td>
</tr>
<tr>
<td><strong>Overall average</strong></td>
<td>82.3</td>
<td>17.9</td>
</tr>
</tbody>
</table>

**Figure 6.** Muscle fiber-type distribution. The difference in the distribution of muscle fiber type between patients with velocardiofacial syndrome (VCFS) and adult cadavers without VCFS is statistically significant ($P < .001$).

**Figure 7.** Average muscle fiber-type diameter. The difference in the diameter between type 1 fibers and type 2 fibers in patients with velocardiofacial syndrome (VCFS) is statistically significant ($P < .001$). The difference in the diameter of type 1 fibers between patients with VCFS and adult cadavers without VCFS is statistically significant ($P < .001$). There is no statistically significant difference in the diameter between type 1 fibers and type 2 fibers in adult cadavers without VCFS. There is no statistically significant difference in the diameter of type 2 muscle fibers between patients with VCFS and adult cadavers without VCFS.
ence in fiber-type distribution is not likely related to pheno-
typic modification of the SPC muscle due to shifting
functional demands during growth and maturation. One
possibility is that this difference is a consequence of al-
tered functional activity of the SPC muscle as a result of
pharyngeal hypotonia, since it is well known that modi-
fications in muscle activity may cause changes in the fi-
ber type and other characteristics. On the other hand,
an intrinsic irregularity in the muscle may be respon-
sible for the altered fiber-type distribution. Velocar-
diofacial syndrome is caused by a microdeletion of chro-
mosome 22q11.1. A novel clathrin heavy chain gene, which
is not identical to the ubiquitously expressed clathrin
heavy chain gene, has been isolated from this area of mi-
crodeletion. This novel clathrin heavy chain gene has
its maximal level of expression in skeletal muscle. More
research has suggested that TBX1, also isolated from this
area of microdeletion, is a major candidate gene in the
development of the VCFS phenotype. The differences
found in this study might be direct or downstream ef-
fects due to haploinsufficiency of the clathrin and/or TBX1
genes, an avenue for future research (Bernice E. Mor-
row, PhD, oral communication, May 2002).

In this study, the type 1 muscle fibers in patients
with VCFS had a significantly smaller diameter than the
type 1 muscle fibers in adults without VCFS. The type 1
fibers in VCFS patients were also smaller than the type
2 fibers in VCFS patients, which were essentially the same
size as both the type 1 and type 2 fibers in adults with-
out VCFS. Lindman and colleagues found that the di-
ameter of the muscle fibers of levator veli palatini muscle
in infants with cleft palate was significantly smaller than
in adults without cleft palate. They concluded that the
ey early developmental stage, rather than a cleft-associa-
ted abnormality, was responsible for this finding. They cited
Tomoda and colleagues, who found differences in muscle
fiber diameter between infant and adult levator veli palat-
tini muscle samples. The VCFS patients in this study were
older and more developed than infants, so the smaller
diameter of the type 1 fibers in these patients was not
necessarily due to an early developmental stage. Fur-
thermore, the diameter of the type 2 fibers in VCFS pa-
tients was equivalent to the diameter of the type 2 fibers
in adults without VCFS. Muscle fiber size is related to
stimulation by its motor nerve such that frequent stimu-
lation will increase the size of the muscle fibers it innerv-
ates and vice versa. Therefore, one possibility to ex-
plain the finding in VCFS patients is a lack of frequent
stimulation of the type 1 fibers. On the other hand, an
intrinsic abnormality of the type 1 muscle fibers, possi-
ibly related to haploinsufficiency of the novel clathrin
heavy chain gene and/or the TBX1 gene mentioned in the
previous paragraph, might be responsible for the smaller
type 1 fiber diameter seen in VCFS patients.

The SPC muscle fibers in this study had pheno-
typic characteristics that are different than those of limb
muscles but similar to those of other facial and soft pal-
ate muscles. In limb muscles, type 1 fibers usually stain
noticeably darker with NADH than type 2 fibers, be-
cause they are oxidative and therefore have more mito-
chondria. In this study, type 2 fibers generally stained
almost as strong or as strong as type 1 fibers. Stal and
Lindman and Lindman et al found similar results in
soft palate muscle fibers and concluded that this high mi-
tochondrial activity of type 2 fibers indicates a high ca-
pacity for aerobic metabolism and a high fatigue resis-
tance, which reflects a high demand for blood supply in
velopharyngeal movements. Facial and extraocular
muscles have also been found to have a higher oxidative
capacity than in limb muscles. Diameter of both type
1 and 2 fibers in human limb muscles generally in-
creases with age to a normal of 40 to 80 µm. In this
study, the type 2 muscle fiber diameter was similar be-
tween the children with VCFS and adults without VCFS,
and the average diameter of the adult type 1 and type
2 muscle fibers was 26.6 and 26.7 µm, respectively. Fur-
thermore, there was great variability in diameter and shape
of the SPC muscle compared with limb muscles. This con-
curs with Stal and Lindman, who found significant indi-
vidual and intramuscular variability in the diameter and
shape of the soft palate muscle fibers. This variability is
most likely related to anatomic and functional differ-
ences between the velopharyngeal muscles and the limb
muscles. For example, muscle fiber growth may not be
as necessary under the minimal load conditions in the
pharynx. Finally, muscle development in the head and
neck may be genetically regulated differently than in limb
muscles.

The histologic arm of this study is limited by the
small size of the VCFS group (9 children) and the re-
stricted control group (3 adult cadavers, all male). The
study will be continued and will include a larger group
of VCFS patients and an expanded control group con-
sisting of younger cadavers and non-VCFS patients un-
dergoing surgery.

In conclusion, significant differences in the thick-
ness, histology, and histochemistry of the SPC muscle
were found in patients with VCFS compared with sub-
jects without VCFS. These differences may offer insight
into the cause of pharyngeal hypotonia and hypernasal
speech seen in these patients. Furthermore, character-
ization of the SPC muscle revealed a phenotype differ-
tent than that of limb muscles but similar to that of other
facial and soft palate muscles. These findings most likely
parallel differences in muscle development and func-
tional demands. A more detailed study of the fiber-type
distribution and fiber diameter using muscle tissue ob-
tained from both adults and children is necessary to clarify
the characterization of this muscle and to determine the
differences between children with and without VCFS and
between children without VCFS and adults without VCFS.

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REFERENCES


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