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Biological Basis and Geometry in Cleft Palate Repair

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Congenital cleft palate is one of the most common birth defects in the craniofacial region. Patients with cleft palate may experience maxillary growth retardation as well as dysfunctions in speech and feeding. Over the past centuries, management of cleft palate has evolved from simple closure of the cleft, although now surgical reconstruction of the palatal structures is one of the most important treatment steps. The optimal palatoplasty should achieve velopharyngeal competence (VPC) in order to develop normal speech, while causing the least growth disturbance to the maxilla and occlusion. This requires not only the closure of the cleft, but also restoration of palatal functions. It is a widely accepted concept that functional reconstruction of the intravelar muscles, particularly the levator veli palatini muscle (LVP), is an essential component in palatoplasty. The levator muscle plays an important role in raising the soft palate, which is critical to achieving adequate velopharyngeal closure. In patients with cleft palate, the origins of the levator muscle on the cranial base are approximately normal, but its course and palatal insertions are abnormal. The levator muscle fibers do not intermingle with fibers from the opposite side to form a sling; by contrast, they course anteriorly within the velum, merge with the other velopharyngeal muscles, and attach to the posterior edge of the palatine bone. Accordingly, modern palatoplasty emphasizes retro-positioning and reconstruction of the levator sling to optimize palatal functions after surgery. Meanwhile, it is necessary to minimize or even eliminate the relaxing incisions and denuded bone surface to avoid excessive scar formation and interference with maxillofacial growth after surgery.

13.1 Exploit the Maximum Muscular Potential in Velopharyngeal Closure

The levator veli palatini muscle plays a primary role in elevating the soft palate that contributes to velopharyngeal closure. Structural abnormalities and functional incompetence of this muscle result in velopharyngeal inadequacy (VPI) in individuals with cleft palate, sometimes even in children with repaired palate. A number of magnetic resonance imaging (MRI) investigations carried out at our center in collaboration with the University of Maryland provide insights into the abnormalities related to VPI [1-3].

We compared velopharyngeal structures including the LVP muscle using high-resolution MRI scans in three matched groups of young children: 1) children with repaired cleft palate and post-operative VPI; 2) children with repaired cleft palate and adequate velopharyngeal closure; and 3) children without cleft palate [1] (Figs. 13.1 – 13.4). It was noted that the children with VPI have less retro-positioning of the levator muscle, and therefore a longer distance for the soft palate to travel in order to reach the posterior pharyngeal wall during velopharyngeal closure (Figs. 13.5 – 13.6). Nonetheless, there are few differences in structural parameters between the two groups with statistical significance. Greater differences are found between the children with and without cleft palate (Fig. 13.5, 13.7 – 13.8). This result suggests that primary palatoplasty cannot restore the levator muscle and velopharyngeal anatomy to "normal", but the better restoration is associated with better velopharyngeal function.

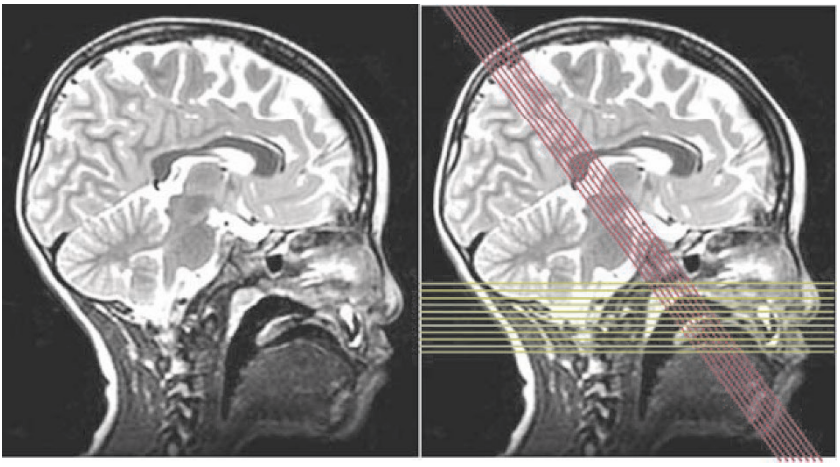

Fig. 13.1 Planes of MRI scans. The image on the left is a midsagittal slice of a 6-year-old boy with right complete cleft lip and palate and VPI following palatal repair. In addition to a multislice sagittal scan covering the whole head, there were a transverse scan covering the whole velopharyngeal mechanism and an oblique scan covering the entire course of the levator muscle sling. The orientations of the transverse and oblique scans are demonstrated in green and red lines in the image on the right

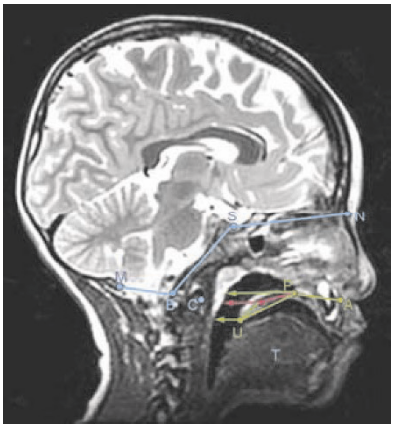

Fig. 13.2 Measurements on the sagittal plane. This graph demonstrates the landmarks and measurements on the sagittal plane of the same child as in Fig.13. 1. The red dot in the middle of the velum stands for the center of the palatal muscles (M). The distance between the posterior nasal spine (P) and M is the effective velar length, whereas that between the uvular tip and M is the posterior velar length. The red arrow stands for the muscle pharyngeal depth. The two other green arrows are the pharyngeal depth and uvular pharyngeal depth. N: nasion, S: sella, B: basion, M: magnum, C_1: anterior border of the first cervical vertebral arch, A: anterior nasal spine (anterior border of the hard palate), P: posterior nasal spine (posterior border of the hard palate), T: tongue

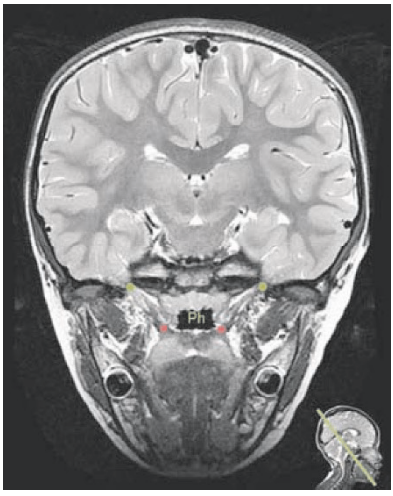

Fig. 13.3 Measurements on the levator veli palatini muscles. This image presents the landmarks used to measure the levator veli palatini muscles (LVP). The orientation of the scan is indicated by the green line at the lower right corner. The two green dots mark the origin of the LVPs, while the two red dots represents where the LVPs insert into the velum. The distance between the green and red dots on either side is the length of the extravelar portion of the LVP. Ph: pharynx

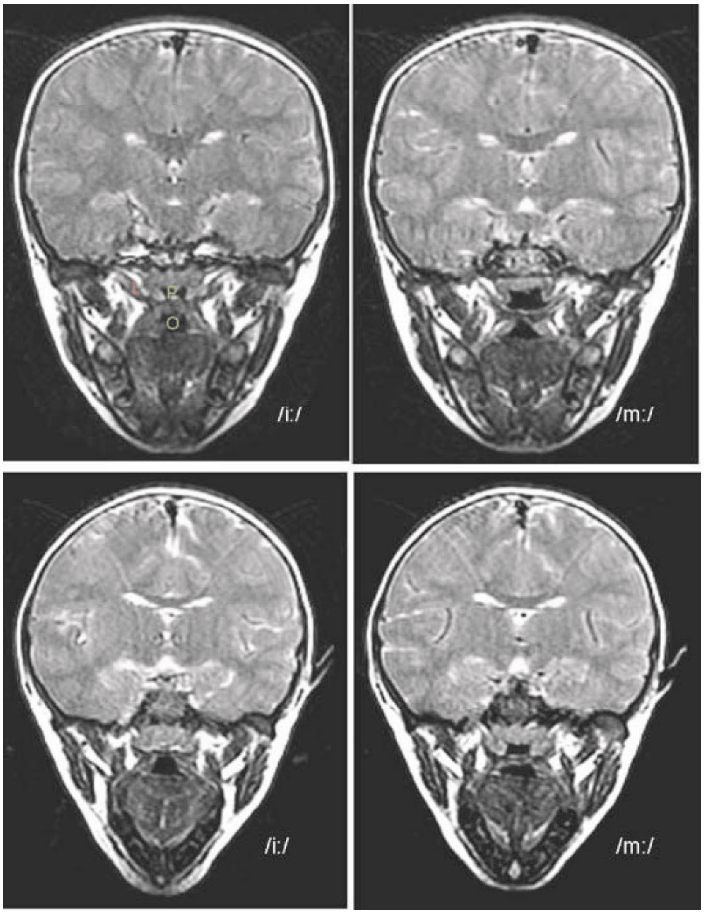

Fig. 13.4 Levator veli palatini muscles during sustained phonation. The four oblique coronal slices above show the contraction of the levator veli palatini muscles (LVPs) during sustained phonation. The levator muscles are reconstructed by the radical dissection technique and form a continuous sling (hypointense area marked by "L"). The upper two images come from a 7-year-old girl with right complete cleft lip and palate and VPI. The lower two images are from her twin brother with left complete cleft lip and palate and adequate velopharyngeal closure. In both cases, there is still a velopharyngeal gap during phonation of /m:/, whereas the adequate closure is demonstrated in the lower left image during phonation of /i:/, but not in the upper left image. L: levator veli palatini muscle, P: pharynx, O: oral cavity

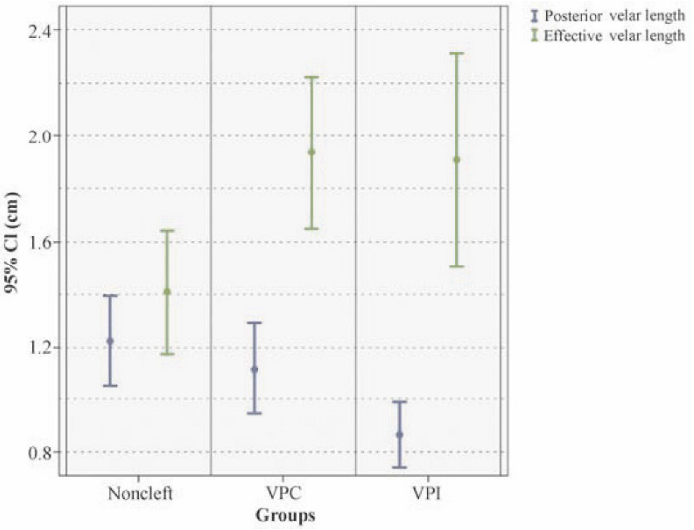

Fig. 13.5 Effective and posterior velar length in the three groups. The two groups with cleft palate have significantly longer anterior (effective) velum than the Noncleft group due to anteriorly located posterior border of the hard palate, which implies more work load in order to pull the velum toward the posterior pharyngeal wall during speech. The posterior velar length is the shortest in the VPI group, followed by the VPC group and Noncleft group

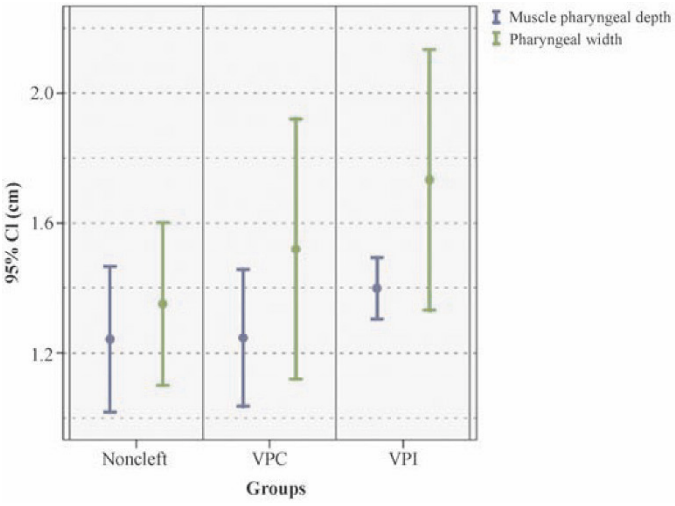

Fig. 13.6 Functional pharyngeal dimensions in the children with repaired cleft palate. The VPC group tends to have narrower and shallower pharynx than the VPI group, although the overall differences between these two groups are not significant (p>0.05)

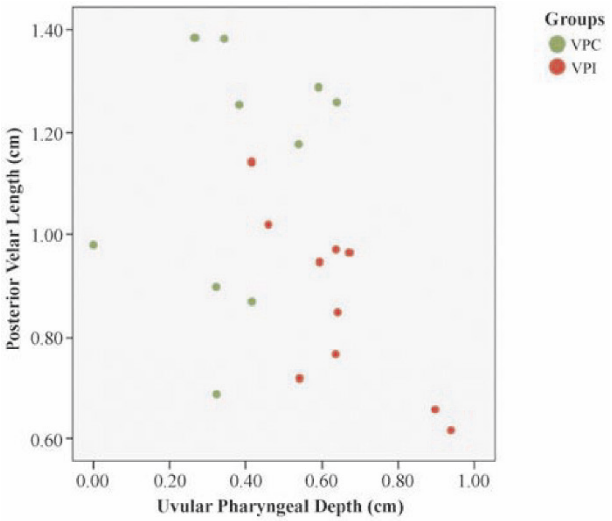

Fig. 13.7 Scatter plot of the uvular pharyngeal depth and posterior velar length in the children with repaired cleft palate. The VPC group has significantly shorter posterior velum and longer uvular pharyngeal depth compared to the VPI group. However, there is no clear distinction between these two groups

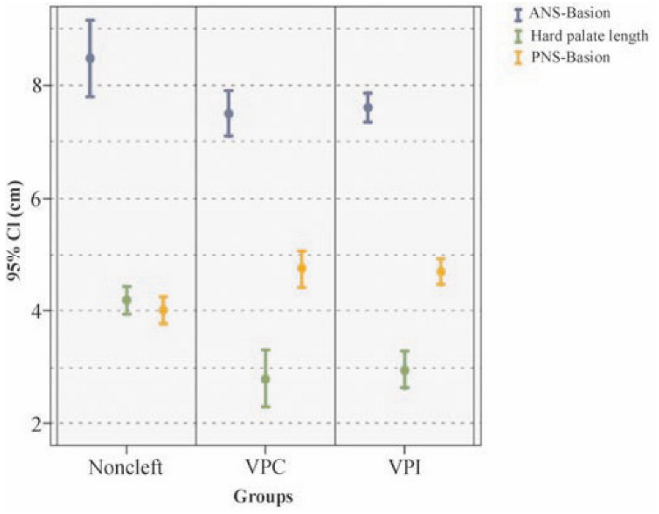

Fig. 13.8 Cranial base depth, hard palate length, and pharyngeal depth in the three groups. The two groups with cleft palate have significantly shorter hard palate and cranial base (ANS-Basion) than the Noncleft group because of the retruded and underdeveloped maxilla in the presence of cleft palate. As a result, the posterior border of the hard palate is more anteriorly located in the two groups with cleft palate compared to that in the Noncleft group as evidenced by the significantly longer pharyngeal depth (PNS-Basion)

Further comparison was made to investigate the extent of maximum velopharyngeal movement and levator muscle shortening during sustained phonation of non-nasal sounds in the three groups [2]. The VPI group had the least maximum velar stretch, lowest maximum velar height, smallest maximum pharyngeal constriction, lowest maximum velopharyngeal ratio (anterior velar length/pharyngeal depth), and least pharyngeal constriction ratio followed by the VPC group (Figs. 13.9 – 13.12). The effective velopharyngeal ratio at rest was found to have a strong positive correlation with the extent of maximum velopharyngeal closure in the antero-posterior dimension during sustained phonation (Fig. 13.13), which suggests a predictive value of the structural ratio between the anterior soft palate length and the pharyngeal depth for VPC. The anterior soft palate originates from the posterior border of the hard palate and ends at the center of the palatal muscles. Its length is determined, in part, by retro-positioning of the LVP muscle in primary palatoplasty.

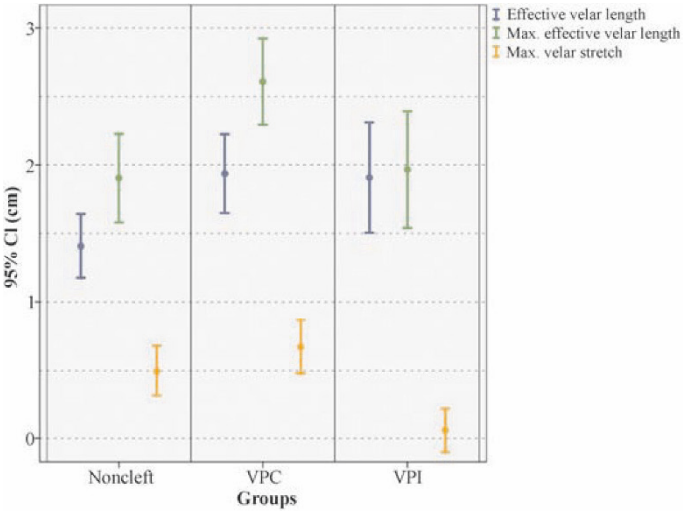

Fig. 13.9 Effective velar lengths at rest and during sustained phonation. The effective velar length at rest is similar in the VPI and VPC group, whereas the velum of the VPC group is stretched significantly more than that of the VPI group during sustained phonation ($p<0.0001$). As a result, the VPC group has significantly greater maximal effective velar length ($p=0.041$) than the VPI group. The Noncleft group has the second greatest velar stretch, whereas the VPI group demonstrates almost no velar stretch on average. The central circle stands for the mean value, whereas the line represents the 95% confidential interval of the variable

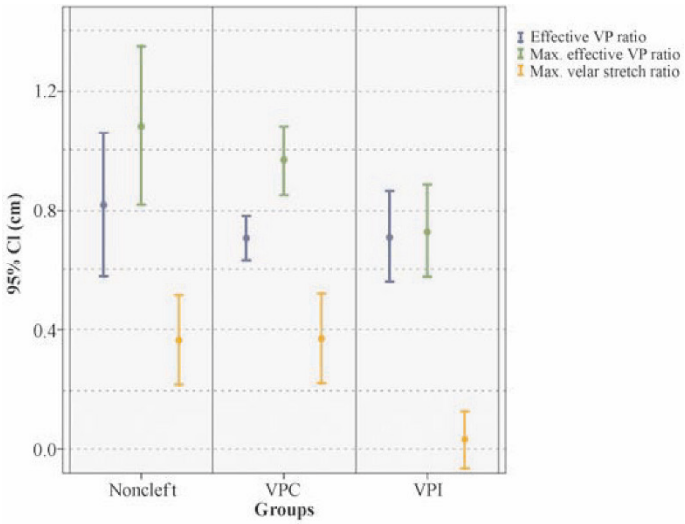

Fig. 13.10 Effective velo pharyngeal (VP) ratios at rest and during sustained phonation. The effective VP ratio at rest is similar among three groups, but the Noncleft group has the greatest maximal effective VP ratio, followed by the VPC and VPI groups respectively. This can be explained by the fact that the Noncleft and VPC groups having significantly greater maximal velar stretch ratio than the VPI group. The central circle stands for the mean value, whereas the line represents the 95% confidential interval of the variable

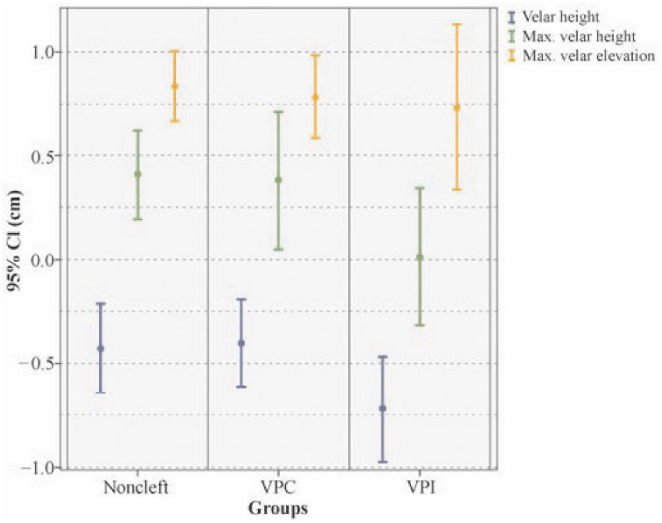

Fig. 13.11 Velar heights at rest and during sustained phonation. The VPI group's palatal muscle center is located the lowest at rest and at its maximal height, although it elevates the similar amount during sustained phonation to those of the Noncleft and VPC groups. Despite the consistent trend, the difference between groups is not statistically significant. The central circle stands for the mean value, whereas the line represents the 95% confidential interval of the variable

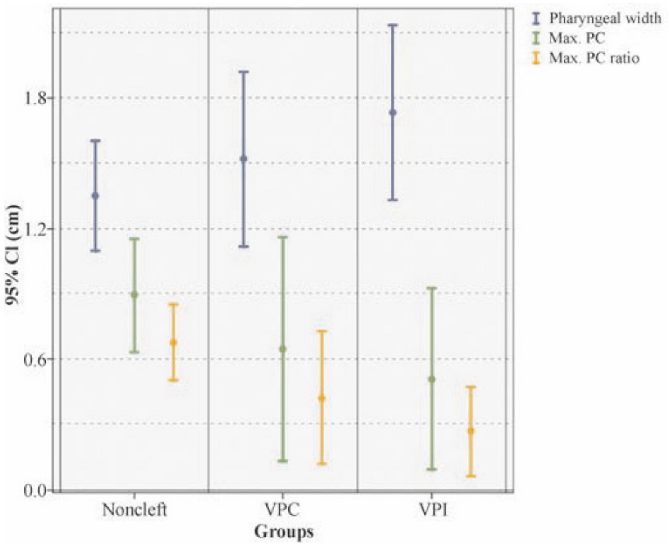

Fig. 13.12 Pharyngeal widths at rest and during sustained phonation. The VPI group has the widest pharynx at rest, but the least maximal pharyngeal constriction (Max.PC) and maximal pharyngeal constriction ratio (Max.PC ratio). The difference between the Noncleft and VPI groups is significant for the maximal pharyngeal constriction ratio (p =0.009). The width measurements are in the unit of centimeter. The central circle stands for the mean value, whereas the line represents the 95% confidential interval of the variable. All width measurements are in the unit of centimeter

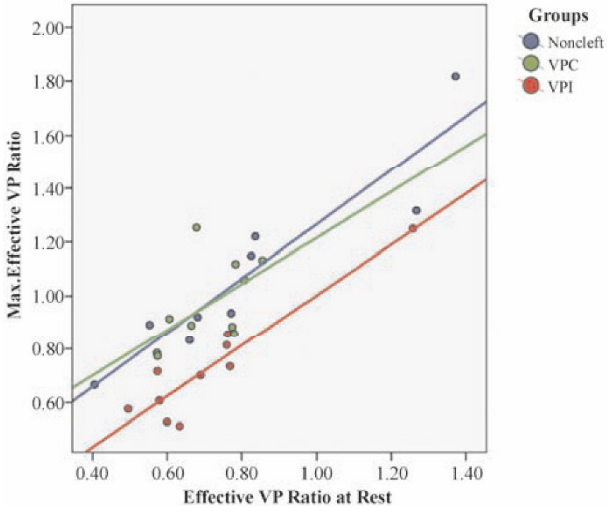

Fig. 13.13 Correlation between the effective VP ratios at rest and during sustained phonation. Regardless of the cleft condition and velopharyngeal function, the maximal effective VP ratio is strongly and significantly correlated to the effective VP ratio at rest (*Spearman*ρ =0.709 – 0.798, p<0.001 – 0.05). The three colored oblique lines stand for different correlation coefficients for the three groups

Interestingly, a strong linear correlation is found between the maximum velar stretch and the maximum pharyngeal constriction ratio in the VPI group (Fig. 13.14), which may imply that velopharyngeal closure movements in the antero-posterior and medial-lateral directions are correlated. It is a matter for further investigation and clarification as to whether the procedures enhancing velar stretch may also improve pharyngeal constriction.

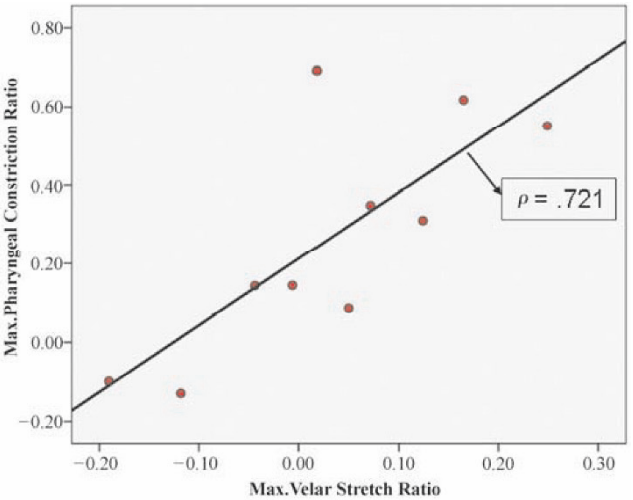

Fig. 13.14 Correlation between the maximal velar stretch and pharyngeal constriction ratio in the children with repaired cleft palate and VPI. In the VPI group, there is a strong and significant correlation between the maximal velar stretch ratio and the maximal pharyngeal constriction ratio (*Spearmanρ* =0.721, *p* =0.019). Although not presented in this graph, the maximal velar stretch is also strongly correlated to the maximal pharyngeal constriction in this group (*Spearmanρ* =0.709, *p* = 0.022). The slope of the oblique line represents the correlation coefficient

Contrary to the previous hypothesis that different length and orientation/angulation of the LVP muscle is associated with cleft palate and VPI, our studies did not find any significant differences between the three groups. Neither did we detect any significant difference in levator muscle shortening ratio during phonation. These results suggest that the primary cause of VPI is not the morphology or contractibility of the levator muscle. Rather, retro-positioning of the levator muscle helps to improve biomechanic competence of the velopharyngeal system. Our results in turn highlight the critical value of thoroughly reconstructing the mal-positioned levator muscle in primary palatoplasty.

13.2 Histological Changes of the Levator Veli Palatini Muscle Caused by Reconstruction in Primary Palatoplasty

Different surgeons may have different understanding of the extent to which the

LVP should be dissected. Some suggest that the LVP should not be dissected too extensively; otherwise muscle function will be compromised by the damaged blood supply resulting from dissection. However, this is merely a theoretical inference and has not been proven by scientific experimentation or is supported with strong evidence. Few researchers to date have studied whether the LVP dissection would lead to muscular fibrosis or whether there would be a strong correlation between extent of dissection and muscular injury.

Animal experimentation was conducted (on cats) to investigate the histological changes of the LVP after different extents of dissection [4]. Extending from the midline to the pterygoideus hamulus, one half of the soft palate was divided equally into three parts: inner, middle and outer part. Based on the different extent of muscular dissection, the subjects were divided into three groups: LVP dissected within the inner part in group 1) LVP dissected within the inner and middle part in group 2) and LVP dissected throughout the entire width of the soft palate in group 3) The LVP muscle in the opposite half palate was left intact to serve as the control. The results were as follows.

13.2.1 Histological Changes of the LVP Following Muscle Dissection

It is widely agreed that the LVP should be dissected and reconstructed in primary palatoplasty. However, the concern that LVP reconstruction might damage the muscle by destroying its blood supply still prevails. If this concern were true, the function of the LVP would be weakened by muscular fibrosis. Our study [4] found that there was no histological change of the dissected LVP muscle in group 1. The arrangement of muscle fibers was orderly and cross-striations were observed one and three months post-operatively. No hyperplasia of fibrous connective tissue was present in this group (Figs. 13.15 – 13.17). These findings confirm that mild muscular dissection does not cause structural changes to the muscle.

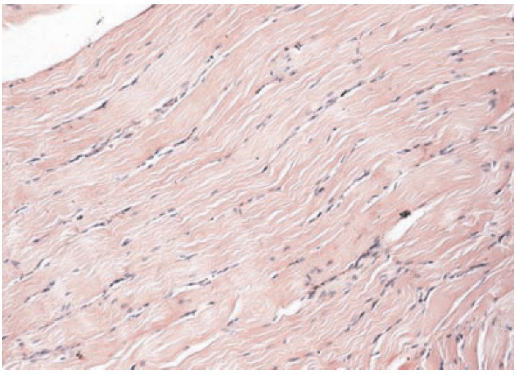

Fig. 13.15 Group normal: The muscle fibers are orderly and tightly arranged. The cross striations are clear (HE stain, ×200)

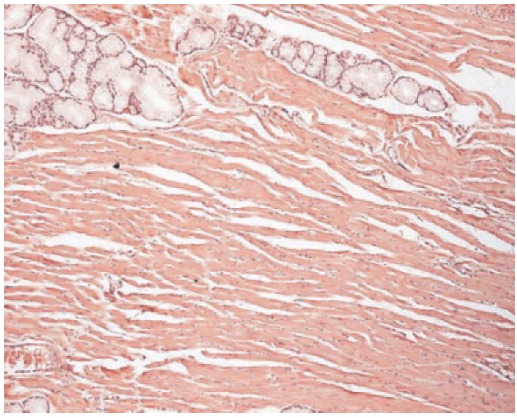

Fig. 13.16 Group 1: one month after surgery. The muscle fibers are almost orderly and a little loosen (HE stain, ×100)

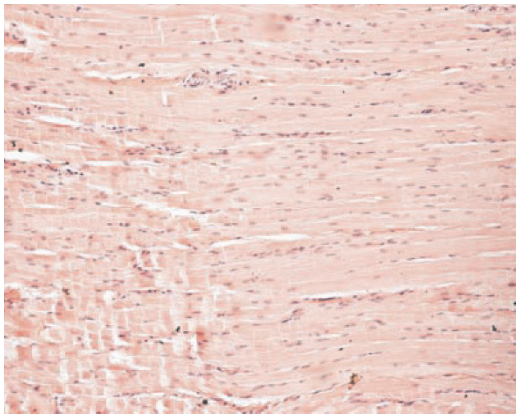

Fig. 13.17 Group 1: three months after surgery. The LVP fibers return to orderly and tightly arranged. The cross striations are clear. No proliferation of fiber tissues in mesenchyme (HE stain, ×200)

The levator muscle in group 2 exhibited mild damage during the first month after surgery (Fig. 13.18). Histological sections showed disordered muscle fibers whereas the blood vessels supplying the muscle were still normal. The muscle fibers were rearranged in an orderly fashion three months post-operatively. No hyperplasia of fibrous connective tissue or infiltration of inflammatory cells was observed (Fig. 13.19). These findings indicate that moderate dissection does not cause irreversible damage to the LVP muscle: there is a transient disturbance but this subsides spontaneously after operation.

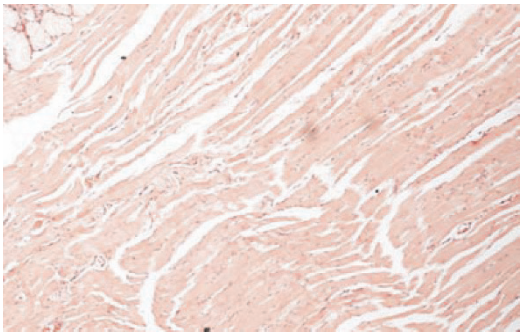

Fig. 13.18 Group 2: one month after surgery. The muscle fibers loosen a little (HE stain, ×100)

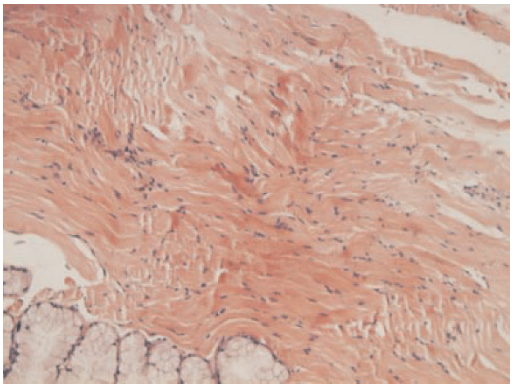

Fig. 13.19 Group 2: three months after surgery. The muscle fibers are orderly. No inflammatory cells infiltrate. No collagen fibers formed (HE stain, ×100)

On the other hand, the arrangement of the LVP muscle fibers in group 3 was disrupted and cross-striations were indistinct at one month after operation. Some muscle fibers were even disconnected and infiltrated by inflammatory cells. Some newly formed muscle fibers with centralized round nuclei were found. No collagenous fibers were formed at this time (Fig. 13.20). Two to three months later, the arrangement of the muscle fibers was restored to their normal condition and the newly formed fibers appeared to have matured. The number of inflammatory cells was significantly reduced and no fibrous connective tissue was present (Fig. 13.21). These observations suggest that extensive dissection can cause noticeable damage to the muscle at an early stage following operation; however, the muscular structure repairs and is restored to its normal condition within three months.

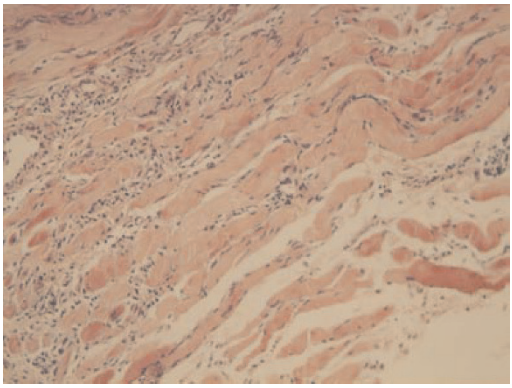

Fig. 13.20 Group 3: one month after surgery. The muscle fibers are almost tightly arranged. Many inflammatory cells infiltrate. Some new formed muscular fibers with centralized round nuclear (HE stain, ×100)

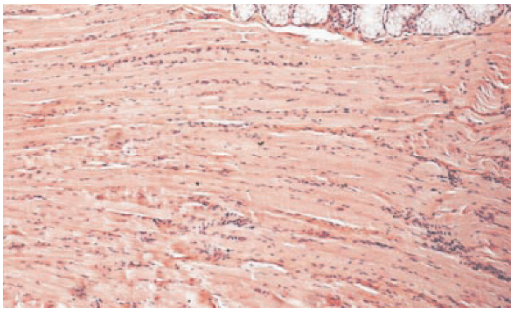

Fig. 13.21 Group 3: three months after surgery. The muscle fibers are orderly and tightly arranged. No proliferation of fiber tissues (HE stain, ×200)

13.2.2 Ultrastructural Changes of the LVP Following Muscle Dissection

Mitochondria exist in all aerobic eukaryotic cells. Their major function is to supply energy in the form of adenosine triphosphate to support functions such as contraction and transport. As the centre of energy production, cellular respiration and metabolism, mitochondria are sensitive to all kinds of detrimental factors and can be regarded as an indicator of cell injury. The progression of muscle injury and the extent of tissue repair after dissection of the palatal muscles can be detected by observing changes in the ultrastructures of mitochondria.

By electron microscopy, it was found that the mitochondria underwent no change, either at the early or late stage after operation when the dissection of the LVP was of mild extent (Figs. 13.22 – 13.23). There was also neither infiltration by inflammatory cells nor hyperplasia of fibrous connective tissue. The myofibril Z

lines were distinct and the sarcomeres normal. Therefore, the ultrastructure of muscle fibers was not altered by mild dissection. When the dissection was extended to the middle part of the soft palate in group 2, increasing mitochondrial cristae and dilated sarcoplasmic reticulum were observed at the early stage after operation (Fig. 13.24). Nevertheless, the myofibril Z line was still distinct and the sarcomeres appeared normal. At the later stage of repair after operation, the number of mitochondrial cristae and the extent of the sarcoplasmic reticulum gradually returned to normal, which implies muscular self-repair over time (Fig. 13.25).

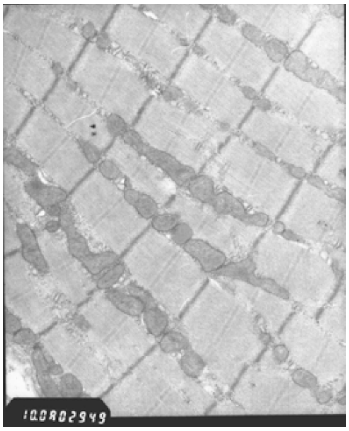

Fig. 13.22 Group 1: one month after surgery. The myofibrils are aligned. The Z lines are clear and the sarcomere was normal (×10000)

Fig. 13.23 Group 1: three months after surgery. The myofibrils are aligned. The Z lines are clear and the sarcomere was normal (×10000)

Fig. 13.24 Group 2: one month after surgery. The Z lines are clear and aligned. More mitochondrial cristae are observed (×10000)

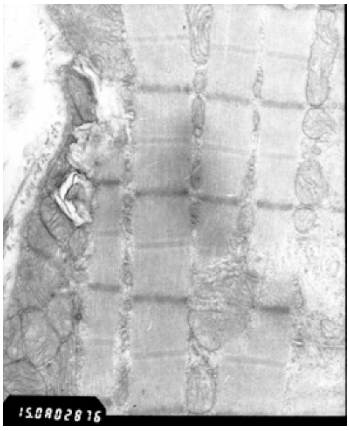

Fig. 13.25 Group 2: three months after surgery. A few enlarged mitochondria. The Z lines are clear and aligned (×15000)

With more extensive dissection, more severe damage was apparent. Mitochondrial cristae and glycogen particles were typical findings in group 3 at the early stage after operation (Fig. 13.26). In addition, the myofibril Z line was indistinct and the arrangement of the muscle fibers was irregular. However, there was no reduction of mitochondrial cristae or any vacuolated change in the mitochondria. By three months after operation, all the structures had returned to normal (Fig. 13.27). In conclusion, extensive dissection of the LVP does not lead to permanent pathological changes of the muscle if the dissection is implemented accurately to carefully protect the deep fascia and the muscle's blood supply.

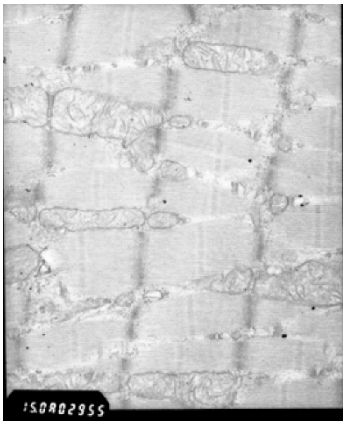

Fig. 13.26 Group 3: one month after surgery. The number of mitochondrial cristae is reduced. The Z lines are not quite clear (×15000)

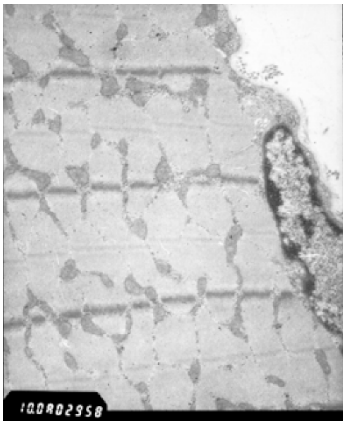

Fig. 13.27 Group 3: three months after surgery. The number of mitochondria is increased. The Z lines turn clear (×10000)

13.3 Geometrical Basis of Using Palatal Mucoperiosteal Flaps to Close the Cleft without Leaving a Denuded Surface

Many surgeons assume that one of the pre-requisites for successful closure of the palatal cleft is that the width of the cleft must be less than the gross width of the bilateral palatal flaps (shown in Fig. 13.28 as $C \leqslant A'+B'$). Based on this opinion, relaxing incisions are made to reduce the occurrence of fistula in classic two-flap palatoplasty. This, however, can be criticized because the palatal flap is not flat and it can in fact supply more tissue than earlier thought (Figs. 13.29 – 13.30). The diagram in Fig. 13.31 helps explain our theory. $\angle\alpha$ and $\angle\beta$ are defined as the angles between the horizontal plane and the individual palatal flap. A and B are the real width of the palatal flap while A' and B′ are the visual width. The relationships between these measurements are: $A' = A \times \cos(\alpha)$, $B' = B \times \cos(\beta)$. If $(A - A') + (B - B') \geqslant C$, which means that we can use the palatal flaps to close the cleft without resorting to relaxing incisions: the greater the angles α and β, the

wider cleft we can close without relaxing incisions. Further, the palatal mucoperiosteal flap has a degree of flexibility and extensibility. If we extensively free and stretch the palatal mucoperiosteal flaps, based on our theory in palatoplasty, we can achieve a much wider coverage. The space between the palatal flap and the nasal layer will disappear soon after operation provided no significant post-surgical hemorrhage occurs.

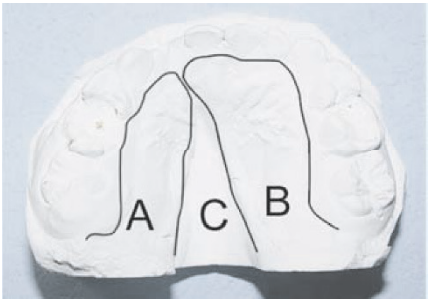

Fig. 13.28 The two dimensional projected view of cleft palate

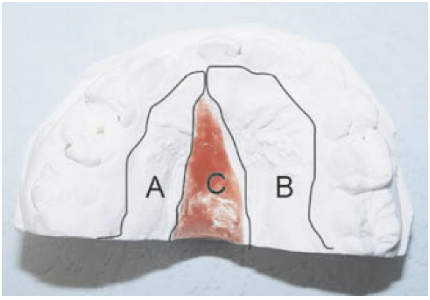

Fig. 13.29 The correlation between the cleft (C) and the real available palatal flaps (A, B)

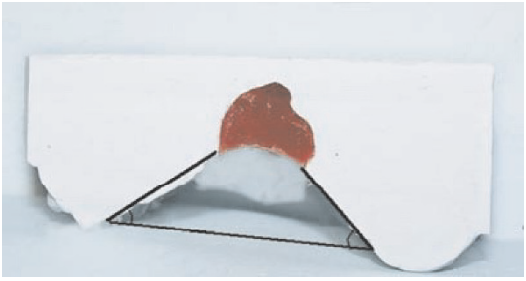

Fig. 13.30 The correlation between the palatal plane and the real available palatal flaps

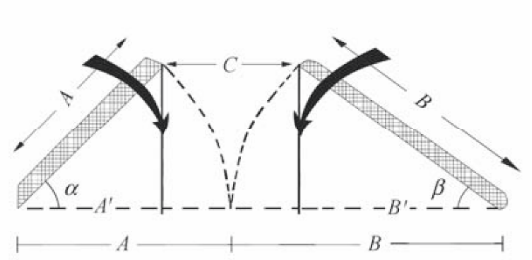

Fig. 13.31 Geometrical basis of using palatal mucoperiosteal flaps to close the cleft without leaving any denuded surface geometric in Sommerlad's reconstruction of LVP

Thus, the palate cleft can be closed without leaving any denuded surface and less scar tissue is formed (Figs. 13.32 – 13.34).

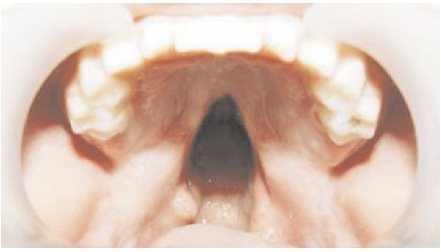

Fig. 13.32 Before palate repair

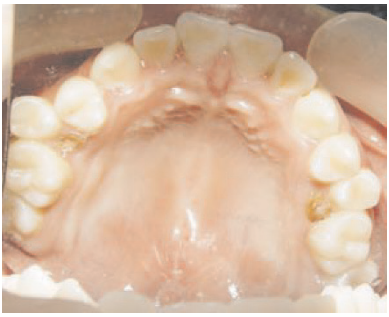

Fig. 13.33 One month after palatoplasty, no release incision

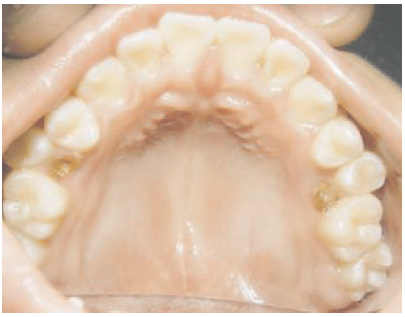

Fig. 13.34 Two months after palatoplasty, the scar was quite mild

References

[1] Tian W, Yin H, Li Y, Zhao SF, Zheng Q, Shi B (2010) Magnetic resonance imaging assessment of velopharyngeal structures in Chinese children after primary palatal repair. *Journal of Craniofacial Surgery*, 21(2):568-577

[2] Tian W, Li Y, Yin H, Zhao SF, Li S, Wang Y, Shi B (2010) Magnetic resonance imaging assess ment of velopharyngeal motion in chinese children after primary palatal repair. *Journal of Craniofacial Surgery*, 21(2):578-587

[3] Tian W, Yin H, Redett RJ, Shi B, Zhang R, Zheng Q (2012) Magnetic resonance imaging assessment of the velopharyngeal mechanism at rest and during speech in Chinese adults and children. *Journal of Speech-Language-Hearing Research*, 53:1595-1615

[4] Lu Y, Shi B, Zheng Q (2010) Effect of dissection and reconstruction of palatal muscles on histological structure and ultrastructure of soft palate. *Journal of Practial Stomatology*, 26(2):185-189 (In Chinese)