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Original Research

Variations and Implications of the Gross Morphology in the Longus colli Muscle in Thoroughbred and Thoroughbred Derivative Horses Presenting With a Congenital Malformation of the Sixth and Seventh Cervical Vertebrae

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ABSTRACT

During the dissection of seven Thoroughbred (Tb) and two Thoroughbred derivative (TbD) horses (9) displaying the congenital malformation of C6 and/or C6 and C7; variations in the gross morphology of the Longus colli muscle were noted. In the absence of the caudal ventral tubercle (CVT) on C6 only, the insertion of the medial and ventral layers and thoracic portion of the L. colli muscle attached to the cranial ventral tubercle (CrVT) on C6. However, on transposition of the CVT from C6 to the ventral surface of C7, the medial and ventral layers, a single deep bundle, and thoracic portion of the L. colli muscle attached to the CrVT on C6 and the transposed CVT on C7. In the unilateral malformation, this placed a distinct asymmetry in the paired left-to-right longitudinal presentation and crosssectional samples of the L. colli muscle. In the bilateral malformation, the CrVTs were longitudinally malaligned and the L. colli replicated the unilateral presentation to a lesser extent. In this presentation, asymmetry was noted in entheses patterns and articular process joints, implying abnormal mechanical load, as was confirmed in the cross sections. As the L. colli muscle has specific cybernetic roles linked to posture and locomotion, these anatomic variations imply dysfunction. Premortem examinations confirmed eight of the nine horses exhibited proprioceptive and neurologic dysfunction (stillborn not included). This raises questions as to the equilibrium of affected horses and therefore the safety in handling and riding such horses, as was found in this study.

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1. Introduction

The relationship between the equine neck and its influence on locomotion has, in recent times, been better understood due to the advances in modern technology [1-4]. These advances have allowed studies to focus on specific areas such as intersegmental vertebral motion in response to biomechanical loading [5,6]. In addition, it was shown that the articulating function between the cervical

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vertebrae has a specific range of motion explained in flexion, extension, lateral bending, and axial rotation [1,3,5–8]. A study by Rombach et al [9] discussed these actions and noted the importance of multibundled muscles in segmental stability between cervical vertebrae and, in particular, the ventral multifascicular layered *Longus colli* muscle. This muscle is a paired juxtavertebral muscle that is located along the ventral surfaces of the cervical vertebrae from C1 to T5 or T6 [5,10–12]. Referred to as a deep ventral perivertebral muscle [8,9], its function is to stabilize, fixate, flex, and rotate the vertebrae [1,5,11,12].

It is believed that the thoracic portion of the *L. colli* muscle with its strong reinforced tendon helps to ventrally support the neck in the cervicothoracic junction [9]. This is







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of particular importance as C6, C7, and T1 are centrally placed in the ventral convex curve [1], and as this muscle inserts on the caudal ventral tubercle (CVT) of C6 [9], it implies that this specific tubercle is a necessary component involved in supporting the ventral convex curve in the equine neck. This places further emphasis on the supportive role that the *L. colli* muscle has, especially due to its anatomic proximity to the cervical and cranial thoracic articular joints [5,11]. In addition, there is no ventral longitudinal ligament until T8 [8]; hence, the need for precise cervical anchor points such as the CVT on C6 for the thoracic portion of the *L. colli* muscle to attach and provide intervertebral stability in the cervicothoracic junction [5,9,10].

Furthermore, Denoix and Pailloux [5] report that the *L. colli* is a cybernetic muscle with rich proprioceptive innervation that functions as a source of postural and locomotive information. This rich innervation transmits the muscle's state of tension to the brain and is achieved by one neuron servicing 20–30 myofibrils. This is in distinct contrast to gymnastic muscles, whereby muscles such as the biceps femoris are serviced by one neuron to every 1,000 or so myofibrils [5]. The main function of cybernetic muscles is to sense motion and produce adaptive postural responses, thus allowing for very precise and subtle control of movement [13]. Similarly, this was confirmed in humans, whereby the proprioceptors in the deep seated cervical muscles were also mechanoreceptors and that pathologic

proprioceptive cervical afferentation results in considerable changes in the organization of spatial orientation [14]. In addition, Ridgway et al [15] noted that equine muscles used incorrectly or were improperly innervated, resulted in muscular asymmetry and that abnormal afferent or efferent input associated with hypomobility or fixation of the vertebral segments caused a singular, or group of muscles, to remain in a hypertonic state.

Subsequently, the reported absence of the CVT on C6 in 19 of 50 Tb horses and three of three Thoroughbred derivative (TbD) horses [16] implies that the *L. colli* muscle must attach elsewhere to function. If so, the implications are most likely asymmetry of the *L. colli* muscle and the potential for postural and locomotive dysfunction due to the influence of muscle tonicity on neural pathways. Therefore, the purpose of this study was to establish the relocation patterns of the *L. colli* muscle in those horses exhibiting the reported congenital malformation of C6 and C7 [16] and where possible, observe horses premortem for postural and/or locomotive dysfunction.

2. Methods

According to Rombach et al [9], the ventral and medial layers of the *L. colli* muscle attach to the CVT of C6 from a cranial direction, whereas the thoracic portion and a single



Fig. 1. A partially dissected normal presentation of the *Longus colli* muscle attaching to the CVT and CrVT of C6 in a 14-year-old Morgan mare. 1. The tendon of the thoracic portion. 2. Left and right CVTs. 3. The ventral crest. 4. Right CrVT. 5. Right transverse process. 6. Ventral layer. 7. Medial layer. CVT, caudal ventral tubercle; CrVT, cranial ventral tubercle.



Fig. 2. The ventral view of C6. Left: normal. Center: unilateral absence of CVT. Right: bilateral absence of CVT. CVT, caudal ventral tubercle.

deep bundle attach to the CVT of C6 and traverse caudally (Fig. 1).

Nine horses were used in this study that either died from unknown causes or were euthanized for purposes unrelated to this study. Premortem examinations were collected from seven of the nine; six had veterinary examinations due to various other conditions; six horses were observed for postural and locomotive dysfunction by the authors in a free state, in hand, or both; one foal was stillborn; five horses were videoed before euthanasia. Postmortem examinations were then collected from the nine horses (seven Tbs and two TbDs). The horses were sourced from Australia (8) and Japan (1), were of mixed gender, aged between 0 (stillborn) and 23 years.

The integument, lateral, and ventral neck muscles were dissected out to reveal the trachea, esophagus, major blood vessels, and long neck nerves. The trachea, esophagus, and major blood vessels were further dissected via cross section caudal to the mandible and cranial to the thoracic outlet or completely removed with the heart and lungs. The left and right layers of the *L. colli* muscle were then carefully dissected to ascertain the points of attachment from C5 to T1, during which comparative observations to normal specimens were made. Where possible, left-to-right cross

sections of the *L. colli* muscle were made for comparative left-to-right symmetry analysis.

The nine dissected horses displayed the congenital malformation of C6 and/or C6 and C7. The C6 only presentation was either unilateral or bilateral (Fig. 2), and the C6 and C7 presentation was either unilateral or bilateral (Fig. 3).

3. Results

Premortem examinations (Table 1) were only partially conclusive across the eight horses observed due to the inconsistency of pathologic issues per individual. However, the following issues and comments were noted:

Twenty-three-year-old Tb stallion (euthanized in Japan) (unilateral left C6)—lameness noted by veterinarian 2 of 5 left hind. Veterinary comments via interpreter: walk—not good weak, funny action in the front; trot—lame left hind. Postural: preferential left forelimb slightly forward (Fig. 4). Previous history: raced. Videoed by veterinarian. Before euthanasia, the front shoes worn by the stallion were removed by the veterinarian and this procedure showed that he was unstable on his forelimbs.



Fig. 3. The ventral view of C7. Left: normal. Center: unilateral transposition of the CVT from C6 to C7. Right: bilateral transposition of the CVT from C6 to C7. CVT, caudal ventral tubercle.

		•			2						
No.	Sex	Age at Death	C6, L R	C7, L R	Forelimb Base Wide	Forelimb Forward	Reported Stumbling	Proprioceptive Dysfunction	Abnormal Ribs	Observed By	Lifting Limbs
1	М	23	L	_		L				V A	D
2	F	20	L R	_	Yes		Yes	Yes		V A	D
3	F	18	L R	_	Yes					_	U
4	F	17	L R	L Rr	Yes		Yes	Yes	L R	A	D
5	F	15	R	R		R	Yes	Yes		Vv A	D
6	Μ	10	L R	L R	Yes		Yes	Yes	R	Vv A	D
7	F	8	L	L		L R	Yes	Yes		V A	D
8	Μ	4	L	L		L	Yes	Yes	L	V	U
8	3M 5F	Av. 14.4	6L 5R	4L 3R	4	3L 2R	6	6	2L 2R	4V 2Vv 6A	6D 2U

 Table 1

 Observations of eight mature horses exhibiting a congenital malformation of C6 and/or C6 and C7.

Abbreviations: A, author; D, difficult; F, female; L, left; M, male; R, right; r, rudimentary; V, veterinarian; Vv, veterinarian viewed video; U, unknown. Note: the stillborn foal is not included in the premortem observations table.

Twenty-year-old Tb mare (euthanized) (bilateral C6)—no lameness. Veterinary comments: apparent forelimb proprioceptive dysfunction. Stumbles at the walk in front but not at the trot. Postural: preferential base wide in front. Previous history: raced, trailed or pleasure, one viable foal, one nonviable foal. Videoed by veterinarian.

Eighteen-year-old Tb mare (unknown death) (bilateral C6)—no lameness. Postural: preferential base wide in front. Previous history: raced, trailed, or pleasure.

Seventeen-year-old Tb mare (euthanized) (bilateral C6 and C7, rudimentary transposition on right C7)—no lameness. Postural: preferential base wide and right forelimb forward. Previous history: eventing, difficulty in forelimb leverage (knocked poles and had fallen, became too dangerous to jump), unstable to trim the hooves in the

forelimbs. Noted in dissection: severe congenital malformation of the sternal ribs, notably on right.

Fifteen-year-old Tb cross Warmblood (Wb) mare (euthanized) (unilateral right C6 and C7)—lameness noted by veterinarian 2 of 5 right hind, 1 of 5 left, and right fore. Postural: preferential right forelimb forward. Previous history: multiple veterinary attendances due to falling over unimpeded in the paddock causing severe injuries on a number of occasions, unstable to trim the forelimb hooves. Deemed unrideable by age 8 years. Videoed and later viewed by a veterinarian: odd action in the right forelimb swings wide and stiff with head held high (Fig. 5).

Ten-year-old Tb gelding (euthanized) (bilateral C6 and C7)—mild foot lameness noted by veterinarian. Videoed when sound—denotes stumbling down the gradient.



Fig. 4. A 23-year-old Tb stallion with a slight left forelimb forward favored posture (white arrow). Tb, Thoroughbred.



Fig. 5. The 15-year-old Tb cross Wb mare observed walking free in her paddock. Note: extended neck, head tilt to the right, stiff right forelimb action, and crossover hinds. Tb, Thoroughbred; Wb, Warmblood.

Veterinarian comments—possible neurologic dysfunction. Postural: preferential base wide in front that became wider with age, especially after rolling, his wide right forelimb posture became more frequent with age (Fig. 6). Previous history: pleasure only, by 10 he was starting to bite his right forearm and had numerous falls in the paddock when playing, mostly due to collapsing hind limbs, unstable to trim the hooves of the forelimbs and hind limbs. Noted in dissection: severe congenital malformation of the sternal ribs, in particular the right.

Eight-year-old Tb mare (euthanized) (unilateral left C6 and C7)—multiple veterinary attendances due to fore and hind limb lameness's. Veterinarian comments: not moving the upper forelimbs correctly and appears uncertain where to place her feet going down a slope. Postural: preferential left or right forelimb forward. Previous history: barrier started

for racing, trailed or pleasure, stumbles on a down slope, difficult to trim forelimbs and hind limbs. Videoed and later viewed by veterinarian. Noted in dissection: subchondral cyst right shoulder joint.

Four-year-old Tb gelding (euthanized) (unilateral left C6 and C7)—no lameness. Veterinary comments: apparent forelimb proprioceptive dysfunction. Persistently falls over when trucked ultimately causing him to be euthanized. Postural: preferential left forelimb forward. Previous history: raced. Noted in dissection: severe congenital malformation of the sternal ribs, in particular the left. Stillborn Tb cross Quarter horse foal (bilateral C6).

Of the nine horses dissected in this study, four exhibited the absent CVT on C6 only (one unilateral and three bilaterally absent) and five presented with an absent CVT on C6 with transposition of the CVT from C6 to the ventral surface of C7 (three unilateral and two bilaterally absent), and the average age at death was 12.6 including the stillborn and 14.4 years without (Table 1).

In those horses only expressing an absence of the CVT on C6 (unilaterally or bilaterally absent), the ventral and medial layers of the *L. colli* muscle attached to the cranial aspect of the cranial ventral tubercle (CrVT) and the thoracic portion and single deep bundle attached to the caudal aspect of the CrVT distal to the transverse process (Fig. 7). Furthermore, the *L.colli* muscle was hypertrophied as it passed caudal and ventral to C7 through the thoracic outlet on the absent side of the CVT on C6, in comparison with the nonabsent side of C6 (Fig. 8).

In those horses expressing a transposition of the CVT from C6 to the ventral surface of C7, the medial and ventral layers of the *L. colli* muscle attached to the CrVT on the absent side of C6, whether in a unilateral or bilateral state. In the unilaterally absent state, the normal CVT on C6 expressed normal attachment of the medial and ventral layers, as well as the thoracic portion and a single deep bundle of the *L. colli* muscle.

In contrast, the normal arrangement of attachment in the thoracic portion of the *L. colli* muscle was changed in those horses expressing a transposed CVT from C6 to the ventral surface of C7 (unilaterally and bilaterally absent). The thoracic portion attached with a small tendon to the



Fig. 6. A 10-year-old Tb gelding with base wide forelimb posture. Left: as an 8-year-old. Center: as a 9-year-old. Right: as a 10-year-old after rolling (the wider right forelimb posture became more habitual with age). Tb, Thoroughbred.



Fig. 7. The Longus colli attachment in a 20-year-old Tb displaying bilateral absence of the CVT on C6. CVT, caudal ventral tubercle; CrVT, cranial ventral tubercle; Tb, Thoroughbred.

caudal aspect of the CrVT on C6 and then reattached to the transposed CVT on C7 with a tendons fibrous tissue, after which a larger tendon traversed caudally through the thoracic outlet as shown in Fig. 9. The single deep bundle attached from the caudal aspect of the CVT on C6 and to the transposed CVT on C7.

cross-sectional observations of the ventral and medial layers, a single deep bundle and the thoracic portion at C6 and C7, noted a relationship between cervical malformation and attachment points of the relative layers. Clear specimens were difficult to ascertain due to variables, such as unilateral absence of the CVT on C6 and whether it transposed to the ventral surface of C7. Bilaterally absent horses had longitudinal asymmetry in the CrVT and the transposed CVT from C6 to the ventral surface of C7. This created asymmetrical attachments of the *L. colli* muscle as previously noted. However, where possible, left and right cross sections were obtained, compared and duly noted.

- 1. Unilaterally absent C6 horses displayed a larger thoracic portion and single deep bundle on the absent side at the thoracic inlet (Fig. 7).
- Unilaterally absent C6 horses displayed a smaller ventral and medial layer on the absent side cranial to the CrVT.
- 3. Bilaterally absent C6 horses remained relatively even left to right.
- 4. Unilaterally absent C6 and C7 horses could not be tested due to the rearrangement of the ventral and medial layers and the thoracic and a single deep bundle.
- 5. Bilaterally absent C6 and C7 horses displayed similar issues as per no. 4, due to the longitudinal variations of the CrVT and the transposed CVT from C6 to the ventral surface of C7 causing deviations in the *Lcolli* (Fig. 10).



Left: Thoracic portion of the L. colli muscle in a normal C6 and C7 horse.

Right: Thoracic portion of the L. colli muscle in a left unilateral absence of the CVT on C6.

Fig. 8. Left: the thoracic portion of the *Longus colli* as it enters the thoracic outlet in a 13-year-old normal Quarter Horse mare. Right: the thoracic portion of the *L. colli* as it enters the thoracic outlet dorsal to the trachea in a 23-year-old Tb stallion displaying left unilateral absence of the CVT on C6. Note the hypertrophy of the left thoracic portion. CVT, caudal ventral tubercle; Tb, Thoroughbred.



Transposed CVT tubercle from C6 onto C7

Fig. 9. Left: the right thoracic portion of the *Longus colli* muscle in a 10-year-old Tb attaching to the caudal aspect of the CrVT on C6 (blue arrow) and traversing to the transposed CVT on C7. Right: the thoracic portion of the *L. colli* muscle in the same 10-year-old Tb attaching to the transposed CVT from C6 on C7 (black arrow). CVT, caudal ventral tubercle; CrVT, cranial ventral tubercle; Tb, Thoroughbred.

An additional finding in this study was that all horses displayed asymmetry in the articular process joints of C6 and C7 (Fig. 11).

4. Discussion

This study showed that the gross morphology of the *L. colli* muscle was variable in the presence of the previously reported congenital malformation in C6, and/or C6 and C7 [16]. In particular, disparities existed in the points of attachment and in this presentation, the *L.colli* muscle ought to be deemed an associative congenital malformation. As a direct consequence, left-to-right cross-sectional asymmetry was significantly noted in the bundles, layers,

and portions in the unilaterally absent presentation of the congenital malformation. Furthermore, the thoracic portion appeared hypertrophied on the unilaterally absent side of C6 (Fig. 7) in direct correlation with ventral load [9], implying that an increase in workload exceeded the preexisting capacity of the muscle fiber [17]. This is highly probable when considering the lack of support provided by the Nuchal ligament lamellae on C6 and C7 [18].

In addition, as the *L. colli* muscle has changed gross morphology relative to its attachments, the likelihood of biomechanical abnormalities leading to overuse injuries is extremely high [19]. As a predominately type I muscle fiber [13], these types have been shown to significantly hypertrophy in response to progressive overload [20–22]. Such



Fig. 10. The Longus colli muscle in a 10-year-old Tb from C5 to C7. Note the curvature in the midline (black line) and associated layer deviations (red and white lines). Tb, Thoroughbred.



Pedicles

Fig. 11. Asymmetric caudal and cranial articular facets of C6 in a 15-year-old Tb cross Wb. Note pedicle asymmetry. Tb, Thoroughbred; Wb, Warmblood.

overuse injuries lead to enthesopathies, especially when anatomic variations and malalignments exist [23] as shown in Figs. 2 and 3, where the ventral crest has become offset with load. This implies that the *L. colli* is under asymmetrical biomechanical load in both the unilateral and bilaterally absent states, and as a cybernetic muscle, the proprioceptors deep within analyzing muscle tension [11] must be sending incorrect postural and locomotive signals to the brain.

This may explain the proprioceptive dysfunction found in this study, even with a limited study group of eight horses (stillborn not included), a relationship was established between the congenital malformations to posture and locomotion. The relevance to posture was demonstrated in preferential forelimb placement in the standing position, whereby a bilaterally absent malformation was noted with base wide posture and a unilaterally absent malformation was noted with forelimb forward posture on the absent side. However, one exception to this was the 8year-old Thoroughbred (Tb) mare (Table 1, no. 7), which displayed no specific left or right postural preference in the left unilateral C6 and C7 malformation. However, on dissection, she had degenerative changes to her right; fetlock, knee, elbow, and a subchondral cyst in the shoulder. Hence, her intermittent left and right forelimb forward posture may be deemed compensatory (Table 1, no. 7).

Base wide posture in horses suffering neurologic dysfunction has been primarily reported in the hind limbs as shown in Figs. 4 and 6 [24]. However, a neurologic forelimb base wide posture and ataxia has been noted in horses with neuroaxonal dystrophy and presents with similar symptoms to cervical vertebral malformation or malarticulation [25]. The unilateral forelimb postural presentation in this study may be attributed to incorrect neural messages transmitted by the corresponding asymmetric cybernetic *L. colli* muscle [5], or axial rotation of C6 and C7 due to asymmetric biomechanical load as shown in the entheses patterns on the ventral crest of C6 and C7 (Figs. 2 and 3) influencing mechanical loading into the forelimb [23], or spinal cord compression or nerve root compression due to defects in the vertebral canal [24]

corresponding to the malformations on the external surface of C6 and C7. These suppositions were beyond the scope of this study but warrant investigating when 22 of 53 Tbs and TbDs expressed the C6 malformation and 11 of 22 involved C6 and C7 [16].

Locomotive evaluation was achieved in six of eight horses with the assistance of a veterinarian (four in person and two via video), and six of eight horses were evaluated by the authors. According to de Lahunta et al [26], neurologic dysfunction influenced by C6–T1 can be represented by the following in the forelimbs: ataxia, paresis, limb flaccidity, decreased-to-absent limb tone and reflexes, muscle atrophy, and hypalgesia. Each horse in this study presented with one or a number of these conditions except for hypalgesia (not assessed within this study). In particular, the 10-year-old Tb became more unstable with age and would fall when playing, leave his right leg in a lateral position after rolling (Fig. 6), fail the hoof replacement test [26], and began biting his upper right forearm after exercise. The 15-year-old Tb cross Wb would walk naturally with her head high and tilted to the right along with a stiff right forelimb action (Fig. 5).

Each horse presented with a different set of neurologic issues from the "two engines" image [26] to traveling with a tilted head extension [24]. Stumbling was another noted locomotive issue among the study group and according to Rush [24], this may be attributed to caudal cervical stenosis between C6-C7, C7-T1. In cervical static stenosis (CSS), the condition is characterized by constant compression of the spinal cord between C5-C7 through a number of degenerative issues such as, vertebral instability due to abnormal mechanical forces [27]. Vertebral malformation has also been noted to create an asymmetry of the articular facets. Ironically, the "Wobbler" example used by Palmer [27] was a left unilateral C6 horse with severe asymmetry of the caudal and cranial articulating facets, and this example displays similar characteristics to all the horses in this study, including the stillborn. Moreover, the 15-year-old Tb cross Wb mare in Fig. 5 displayed the most severe asymmetry of the articular facets and pedicles (Fig. 11), and those two conditions have been well established as contributing factors to neurologic dysfunction in a number of species [26,27].

Neurologic dysfunction in equines is multifaceted, and Tbs are a predisposed breed due to factors such as nutrition and growth rates [24]. However, the occurrence of the C6 congenital malformation in 38% of Tbs [16] has not been previously factored into the discussion as a causative agent nor the C6 and C7 collective malformation. Furthermore, the mechanical forces of the L.colli muscle clearly place an asymmetric load to its points of attachment and falters in its role as an intersegmental stabilizer, subsequently leading to vertebral instability, degenerative joint changes, and asymmetrical articular processes [27]. In addition, as a cybernetic muscle, its associative congenital presentation in C6 and/or C6 and C7 would lead to the brain receiving incorrect neural messages due to abnormal paired left and right tension in the muscle, and as a direct consequence, the horse would adjust its posture accordingly [5,14,15]. In fact, this study has shown that the function of the L. colli muscle has been severely compromised in the presence of the congenital malformation in C6 and C7 as previously reported and that new strategies will need to be implemented to limit the potential damage to affected horses and danger to riders.

5. Conclusions

This study has clearly demonstrated that the *L.colli* muscle has altered its points of attachments and subsequently loss of function in the presence of the congenital malformation in C6 and C7. The potential ramifications are neurologic dysfunction, loss of performance, and potential harm to horse and rider. It would be a recommendation of the authors to investigate management programs to stabilize the musculoskeletal system in the cervicothoracic junction to prevent further potential wastage within the industry.

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