Mechanical advantage of the canine triangularis sterni

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De Troyer, André, and Alexandre Legrand. Mechanical advantage of the canine triangularis sterni. J. Appl. Physiol. 84(2): 562–568, 1998.—Recent studies on the canine parasternal intercostal, sternomastoid, and scalene muscles have shown that the maximal changes in airway opening pressure (ΔPao) obtained per unit muscle mass (ΔPao/m) during isolated contraction are closely related to the fractional changes in muscle length per unit volume increase of the relaxed chest wall. In the present study, we have examined the validity of this relationship for the triangularis sterni, an important expiratory muscle of the rib cage in dogs. Passive inflation above functional residual capacity (FRC) induced a virtually linear increase in muscle length, such that, with a 1.0-liter inflation, the muscle lengthened by $17.9 \pm 1.6$ (SE) % of its FRC length. When the muscle in one interspace was maximally stimulated at FRC, Pao increased by $0.84 \pm 0.11$ cmH2O. However, in agreement with the length-tension characteristics of the muscle, when lung volume was increased by 1.0 liter before stimulation, the rise in Pao amounted to $1.75 \pm 0.12$ cmH2O. At the higher volume, ΔPao/m therefore averaged $+0.53 \pm 0.05$ cmH2O/g, such that the coefficient of proportionality between the change in triangularis sterni length during passive inflation and ΔPao/m was the same as that previously obtained for the parasternal intercostal and neck inspiratory muscles. These observations, therefore, confirm that there is a unique relationship between the fractional changes in length of the respiratory muscles, both inspiratory and expiratory, during passive inflation and their ΔPao/m. Consequently, the maximal effect of a particular muscle on the lung can be predicted on the basis of its change in length during passive inflation and its mass. A geometric analysis of the rib cage also established that the lengthening of the canine triangularis sterni during passive inflation is much greater than the shortening of the parasternal intercostals because, in dogs, the costal cartilages slope downward from the sternum.

mechanics of breathing; respiratory muscles; maximal respiratory effect; expiratory muscles

Although the actions of most respiratory muscles on the chest wall have been qualitatively described, the question of how much lung expansion (or deflation) each of these muscles can produce has not been answered. This is a difficult question because a number of muscles cannot be maximally activated in isolation, but recent theoretical studies by Wilson and De Troyer (16, 17) have proposed an indirect approach. Thus, by using a standard theorem of mechanics, the Maxwell reciprocity theorem, these investigators have postulated that the potential change in airway opening pressure (ΔPao) produced by a muscle contracting alone against a closed airway is related to the mass (m) of the muscle, the maximal active muscle tension per unit cross-sectional area (τ), and the fractional change in muscle length per unit volume increase of the relaxed chest wall

\[
\Delta P_{ao} = m \tau \left( \frac{\Delta L}{V_{L}} \right)_{rel}
\]

such that

\[
\Delta P_{ao} = m \tau \left( \frac{\Delta L}{V_{L}} \right)_{rel}
\]

(1)

Studies in anesthetized dogs have shown that the internal intercostal muscles of the parasternal region of the rib cage (the so-called parasternal intercostals), the sternomastoids, and the scalenes behave in agreement with this equation (3, 10, 11). Indeed, for these three sets of inspiratory muscles, ΔPao values obtained per unit muscle mass (ΔPao/m) during maximal stimulation were closely related to the fractional changes in muscle length during passive inflation, such that a greater fractional shortening was associated with a greater fall in Pao per gram of muscle mass. If this relationship could be extended to other muscles, in particular some expiratory muscles, then one would have strong evidence for estimating the respiratory effect of any particular muscle simply on the basis of its fractional change in length during passive inflation.

The present study was therefore undertaken to assess the extent to which Eq. 1 also applies to the canine triangularis sterni, a well-established expiratory muscle of the rib cage (4). We initially measured the change in length of this muscle during gradual, passive inflation of the chest wall in a group of anesthetized, paralyzed animals. As anticipated, passive inflation caused muscle shortening, but the amount of shortening, although variable among animals, was much larger than the shortening of the parasternal intercostals. To understand this large muscle shortening and the variability among animals, we subsequently developed a geometric model of the ventral area of the rib cage and obtained a relationship between muscle shortening and the angles describing the orientation of the muscle and costal cartilage. We then measured these angles in another animal group. Last, as a direct test of Eq. 1, we measured the pressure-generating ability of the muscle.

METHODS

Experiment 1. The relationship between lung volume and the length of the triangularis sterni muscle was examined in four adult mongrel dogs (15–29 kg). The animals were deeply anesthetized with pentobarbital sodium (initial dose = 30 mg/kg iv), placed in the supine posture, and intubated with a cuffed endotracheal tube, after which the parasternal region of the rib cage was exposed on the right side of the sternum. The animal was then anesthetized with pentobarbital sodium (initial dose = 30 mg/kg iv), placed in the supine posture, and intubated with a cuffed endotracheal tube, after which the parasternal region of the rib cage was exposed on the right side of the sternum. Two interspaces between the third and the sixth were studied in each animal. In each interspace, the triangularis sterni was exposed by sectioning the caudal insertion of the parasternal intercostal from the lateral border of the sternum to the chondrocostal junction. The orientation of the triangularis sterni fibers could therefore be carefully defined, and a pair of small screws was inserted into the sternum in the midline and into the lateral portion of the costal cartilage at the points of insertion of the muscle bundle. The animal was then para-
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Lyzed with an intravenous injection of 2 mg pancuronium and ventilated mechanically.

Then, mechanical ventilation was stopped, the chest wall was allowed to relax to equilibrium, and the linear distance between the two screws of each pair (i.e., the length of the triangularis sterni muscle bundle at functional residual capacity [FRC]) was measured with a caliper (2, 3, 10). The tracheal cannula was then connected to a calibrated supersyringe, and lung volume was either increased at random by 0.2, 0.4, 0.6, 0.8, and 1.0 liter above FRC or decreased by 0.2 liter below FRC. At each lung volume, three measurements of muscle length were made.

Model. Figure 1 shows a simple model of the geometry of the triangularis sterni; the model is similar to the one we have previously developed for the parasternal intercostals (3). Triangularis sterni muscle length \( L \) is related to the distance \( d \) along the costal cartilage between the sternum and the muscle attachment, the distance \( s \) along the sternum between the attachments of the muscle and cartilage, and the angle \( \alpha \) between the sternum and the costal cartilage by the law of cosines

\[
L^2 = d^2 + s^2 - 2ds \cos \alpha
\] (2)

The values of \( d \) and \( s \) are constant with changes in lung volume. Therefore, if the values of \( L \) and \( \alpha \) before and after a 1.0-liter passive inflation are denoted by subscripts 1 and 2, respectively

\[
L^2_2 - L^2_1 = 2ds (\cos \alpha_1 - \cos \alpha_2)
\] (3)

and the fractional change in muscle length, \( \Delta L/L_1 = L_2/L_1 - 1 \), is given by the following equation

\[
\Delta L/L_1 = \left[1 + (2ds/L_1)(\cos \alpha_1 - \cos \alpha_2)\right]^{1/2} - 1
\] (4)

If \( \beta \) denotes the angle between the sternum and the muscle fibers, simple trigonometric relationships yield the results

\[
d/dL_1 = \sin \beta/\sin \alpha_1 \quad \text{and} \quad s/dL_1 = \sin (\beta - \alpha_1)/\sin \alpha_1
\]

With these substitutions, Eq. 4 becomes

\[
\Delta L/L_1 = [1 + (2 \sin \beta \sin (\beta - \alpha_1)/\sin^2 \alpha_1]
\]

\[
\times (\cos \alpha_1 - \cos \alpha_2)^{1/2} - 1
\] (5)

Experiment 2. Nine adult mongrel dogs (15–22 kg) were subsequently studied to identify the factors responsible for the amount and variability of muscle lengthening observed in experiment 1 and to assess the muscle's pressure-generating ability. As in experiment 1, the animals were deeply anesthetized with pentobarbital sodium (30 mg/kg iv), placed in the supine posture, and intubated with a cuffed endotracheal tube, and the rib cage and intercostal muscles in the parasternal area were exposed on both sides of the chest from the first through the eighth interspaces. The triangularis sterni in this study was investigated in one interspace between the third and the sixth in six animals and in both the third and the sixth interspaces in three animals. For the 9 animals, a total of 12 interspaces were thus studied. All measurements were obtained while the animals were made apneic by mechanical hyperventilation.

In each interspace, we first measured the fractional change in muscle length during passive inflation. The technique and experimental protocol were essentially similar to the ones used in experiment 1. It is noteworthy, however, that by sectioning the parasternal intercostal fibers exclusively along their caudal insertions, we produced only minimal, if any, alteration in local rib cage compliance. In addition, we could ensure that the distal part of the internal intercostal nerve (which supplies the triangularis sterni and runs near the cranial border of the interspace) was left intact; the triangularis sterni could therefore be stimulated maximally later. Furthermore, the data obtained in experiment 1 indicated that the relationship between lung volume and muscle length was virtually linear above FRC (see results). Consequently, to have the greatest signal-to-noise ratio in the measurement, all passive inflations in the study equaled 1.0 liter.

When measurements of muscle length were completed, we aligned the lower edge of a protractor with the sternum and measured the acute angle between the sternum and the direction of the triangularis sterni muscle fibers thus exposed (\( \beta \) in Eq. 5) and the acute angle between the sternum and the lateral part of the costal cartilage at FRC (angle \( \alpha_1 \)). Lung volume was then passively increased by 1.0 liter, and the angle between the sternum and the lateral part of the cartilage (angle \( \alpha_2 \)) was measured again. All these measurements were also made in triplicate.

We then assessed the muscle's pressure-generating ability. The muscle fibers of the left parasternal intercostal were thus sectioned along their caudal insertion from the sternum to the chondrocostal junction, and the ventral part of the external intercostal muscle was removed bilaterally. The ventral part of the right and left internal interosseus intercostal muscle was also sectioned along its caudal insertions, after which the internal intercostal nerve was exposed. Nerve exposure was made bilaterally 1–2 cm lateral to the chondrocostal junction by using the procedure previously described (2). A pair of stainless steel hook electrodes spaced 3–4 mm apart was then implanted into the triangularis sterni muscle, and the freed sector of the nerve was positioned across a bipolar stimulating electrode. With the animal apneic, pulses of 0.2-ms duration were delivered to the nerve at intervals of 1 s, and stimulus intensity was increased progressively until it was 50% greater than that required to produce a compound muscle action potential of maximal
amplitude. The internal intercostal nerve was subsequently sectioned ~1 cm dorsal to the site of stimulation.

A Validyne differential pressure transducer was then connected to a side port of the endotracheal tube to measure Pao. The tube was occluded with the animal at FRC, and the distal end of the nerve was stimulated bilaterally by applying square pulses of 0.2-ms duration and supramaximal voltage at a frequency of 50 impulses/s. With these stimulation parameters (8) and our surgical preparation, we could ensure reproducible, maximal contraction of all the triangularis sterni fibers supplied by the nerve while avoiding simultaneous contraction of the external and internal intercostal muscles in the same interspace. Sectioning the nerves also avoided stimulation of the spindle afferent fibers, which are known to have extrasegmental projections (5) and could have induced contraction of intercostal muscles in adjacent interspaces. Muscle stimulation at FRC was performed three times, after which lung volume was passively increased first by 0.5 liter and then by 1.0 liter. At each lung volume, three trials of bilateral, supramaximal stimulation were also performed.

In each interspace, we finally assessed muscle mass. Although the internal intercostal nerves supply the triangularis sterni in a segmental fashion, previous studies have shown that, in dogs, the motor territory of a particular nerve frequently extends beyond the muscle fibers situated in the corresponding interspace (13). To identify the muscle bundles responsible for the pressure changes thus recorded, the animal was killed by an overdose of pentobarbital sodium and the ventral aspect of the rib cage was quickly removed en bloc. Pulses of 0.2-ms duration and supramaximal voltage were then immediately delivered at intervals of 1 s first to the right and then to the left internal intercostal nerve. The muscle bundles thus stimulated could therefore be easily visualized; these bundles were harvested and weighed.

Data analysis. The length of each muscle bundle at each lung volume was averaged over the three measurements that were performed. The angles describing the orientation of the muscle fibers and costal cartilage relative to the sternum and L were obtained during nerve stimulation were also averaged over the three measurements. To allow comparison among the different animals, the changes in muscle length induced by passive inflation (deflation) were subsequently expressed as percent changes relative to the muscle length at FRC, and ΔPao values were divided by muscle mass to yield specific ΔPao (spec ΔPao). Data were finally averaged for the animal group, and the values are presented as means ± SE. Statistical comparison between ΔPao measured at the different lung volumes was made by analysis of variance with repeated measures and Student-Newman-Keuls tests. The criterion for statistical significance was taken as P < 0.05.

RESULTS

Relationship between lung volume and muscle length (experiment 1). The triangularis sterni lengthened with passive inflation above FRC and shortened with passive deflation below FRC in the four animals (8 interspaces) studied. As shown in Fig. 2, the relationship thus obtained between lung volume and muscle length was curvilinear, such that, for a given volume change, the amount of muscle shortening associated with deflation was about one-half the amount of lengthening caused by inflation. Above FRC, however, even though the amount of muscle lengthening induced by a given increase in volume also decreased at high lung volumes, the relationship was virtually linear; on average, the departure from linearity was only 10–15%.

Figure 2 illustrates two other features of the response of the triangularis sterni to passive inflation. The amount of muscle lengthening associated with a 1.0-liter inflation in the eight interspaces averaged 15.9% of the muscle length at FRC and was therefore substantially larger than the amount of para-sternal intercostal shortening previously observed (3). However, the values measured in the individual animals ranged from 5.9 to 28.1%, and this variability could not be explained on the basis of animal size. Indeed, identical values were found in the animals of the group with the smallest (15 kg) and the largest (29 kg) body mass.

Muscle and cartilage orientation (experiment 2). The changes in triangularis sterni muscle length during passive inflation in the 9 animals (12 interspaces) studied were similar in all respects to those observed in experiment 1. With a 1.0-liter inflation, the amount of muscle lengthening thus averaged 17.9 ± 1.6% of the muscle length at FRC but varied from 5.7 to 25.3%. These extreme values were obtained in two animals with the same body mass, thus confirming that animal size was not the primary determinant of this variability.

The obtuse angle between the sternum and the triangularis sterni fibers (angle β) was also variable in the different animals, ranging between 109 and 145° (126 ± 3 (SE)°). Similarly, the acute angle between the sternum and the lateral part of the costal cartilage at FRC (angle α₁) varied between 40 and 67° (54 ± 2°), and its increase with passive inflation ranged from 4 to 18° (11 ± 1°). As a result, the angle α₂ ranged from 51 to 85° (65 ± 2°), and when we substituted the values of angles β, α₁, and α₂ into Eq. 5 so as to calculate the...
changes in muscle length in each individual interspace, the computed values were very close to the measured values (Fig. 3). The computed value for the 12 interspaces studied averaged 18.4 ± 1.8%. Thus the differences in muscle lengthening between the different animals were well accounted for by the differences in the orientation of the muscle fibers and costal cartilage and by the differences in cartilage rotation during passive inflation.

Pressure-generating ability (experiment 2). The D\textsubscript{Pao} values recorded during maximal, tetanic stimulation of the triangularis sterni at different lung volumes are shown for one representative animal in Fig. 4. In this example, the rise in Pao generated at FRC was 0.54 cmH\textsubscript{2}O. However, when lung (chest wall) volume was increased by 0.5 liter above FRC, the rise in Pao caused by the same stimulation amounted to 1.49 cmH\textsubscript{2}O. Increasing lung volume by an additional 0.5 liter accentuated this effect such that the rise in Pao was 2.02 cmH\textsubscript{2}O.

This influence of lung volume was seen in all interspaces, as shown in Fig. 5. For the 12 interspaces, the rise in Pao induced by stimulation thus increased progressively from 0.84 ± 0.11 cmH\textsubscript{2}O at FRC to 1.75 ± 0.12 cmH\textsubscript{2}O at 1.0 liter above FRC (P < 0.001). No consistent difference was seen between values for the third and the sixth interspaces in the three animals in which both interspaces were studied.

The mass of triangularis muscle thus stimulated ranged from 2.16 to 4.49 g [3.34 ± 0.21 (SE) g]. As a result, spec D\textsubscript{Pao} at 1.0 liter above FRC ranged from 0.22 to 0.77 cmH\textsubscript{2}O/g (mean ± SE = 0.53 ± 0.05 cmH\textsubscript{2}O/g). The interanimal variability in spec D\textsubscript{Pao} was therefore of the same order of magnitude as the variability in muscle lengthening during passive inflation. More importantly, there was a close relationship between these two variables such that a greater fractional muscle lengthening corresponded to a greater spec D\textsubscript{Pao} (Fig. 6). The correlation coefficient of this relationship was 0.88, and its slope was 2.6.

**DISCUSSION**

The triangularis sterni in dogs and cats is invariably active during the expiratory phase of the breathing cycle, and this contraction pulls the ribs caudally to deflate the rib cage compartment of the chest wall (4, 9, 13). Therefore, one would anticipate that this muscle would lengthen during inflation of the relaxed chest wall, and indeed passive inflation caused lengthening of the triangularis sterni in all animals. Our study of the geometry of the rib cage in individual animals (Fig. 3) also indicated that this change in muscle length is
determined by three factors, namely, the orientations of the muscle fibers and costal cartilage relative to the sternum and the rotation of the cartilage during inflation.

We have previously shown that these three factors also determine the changes in length of the parasternal intercostals (3). However, whereas the canine parasternal intercostals shorten by 5–10% with a 1.0-liter passive inflation (3), the triangularis sterni for the same inflation lengthened, on average, by 17.9%. The mechanism of this difference in magnitude is illustrated in Fig. 7. In this figure, the fractional changes in muscle length during passive inflation, calculated from Eq. 5 for the average values of angle $\alpha_1$ (54°) and angle $\alpha_2$ (65°) measured in the present study, are plotted as a function of angle $\beta$ ($\beta_5$). According to Eq. 5, the values of $\Delta L/L_1$ are negative for angle $\beta < \alpha_1$ and positive for angle $\beta > \alpha_1$. Therefore, all muscle fibers with angle $\beta < 54^\circ$ shorten during passive inflation, and all muscle fibers with angle $\beta > 54^\circ$ lengthen. However, the curve is clearly asymmetric, such that the potential maximum shortening is only 6%, whereas the potential maximum lengthening amounts to 18%. These values are similar to the parasternal shortening measured in our previous studies (3) and to the triangularis sterni shortening measured in this study, respectively, which indicates that the orientations of both the parasternals and the triangularis sterni in dogs are close to the orientations for maximum length changes. On the other hand, if the costal cartilage extended out from the sternum at a right angle (angle $\alpha_2 = 90^\circ$), then the curve ($\circ$) would be antisymmetric about $\beta = 90^\circ$, and the magnitudes of maximum shortening and lengthening would be equal. In other words, the lengthening of the canine triangularis sterni during passive inflation is two to three times greater than the shortening of the parasternal intercostals because, in dogs, the costal cartilages slope downward from the sternum.

Previous studies have shown that in supine dogs the length of the triangularis sterni at FRC corresponds to ~75% of the muscle's in vitro optimal length ($L_o$) (8). In view of the large increase in muscle length during passive inflation, one would therefore expect that an increase in lung volume would displace the muscle toward $L_o$ and, hence, would induce a gradual increase in the muscle's pressure-generating ability. As shown in Figs. 4 and 5, this is exactly what we observed. With a 1.0-liter passive inflation, the muscle lengthened, on average, by 18% of its FRC length. Consequently, at this lung volume, the muscle was at 0.75 $\times$ 1.18 or 90% passive inflation, one would therefore expect that an increase in lung volume would displace the muscle toward $L_o$ and, hence, would induce a gradual increase in the muscle's pressure-generating ability. As shown in Figs. 4 and 5, this is exactly what we observed. With a 1.0-liter passive inflation, the muscle lengthened, on average, by 18% of its FRC length. Consequently, at this lung volume, the muscle was at 0.75 $\times$ 1.18 or 90%
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of $L_o$. However, we measured muscle length as the linear distance between two screws implanted in the costal cartilage and the sternum. These measurements, therefore, encompassed a portion of cartilage and the tendinous attachments of the muscle to the sternum. These attachments are 5–10 mm in length. As a result, our values of $L_1$ were greater than the actual muscle length, and our measured values of $\Delta L/L_1$ underestimated the fractional muscle shortening by 10–15%. Thus, when lung volume was increased by 1.0 liter above FRC, the triangularis sterni was very close to its $L_o$. Presumably, therefore, its pressure-generating ability was then maximal or near maximal.

Our previous theoretical studies have led to the prediction, summarized in Eq. 1, that the maximal $\Delta Pao$ produced by a given respiratory muscle per unit muscle mass (i.e., spec $\Delta Pao$) is equal to the product of the fractional change in length of the muscle during a 1.0-liter passive inflation and $\sigma$ (16, 17). In agreement with this prediction, we have previously shown that the spec $\Delta Pao$ values for the parasternal intercostals, the sternomastoids, and the scalenes in dogs are proportional to the fractional changes in muscle length during passive inflation (3, 10). Furthermore, the coefficient of proportionality ($\sigma$) between spec $\Delta Pao$ and the fractional change in length for these three sets of inspiratory muscles was $\sim 3.0$, and this provided further evidence in support of Eq. 1; indeed, when measured in vitro, maximal active tension in both limb and respiratory muscles is 2.2–3.5 kg/cm² (1, 6, 15). Similarly, spec $\Delta Pao$ for the triangularis sterni in the different animals of this study was closely related to the fractional muscle shortening during passive inflation (Fig. 6), and when the data obtained for this muscle in the animal group were averaged and plotted together with the data previously obtained for the parasternal intercostals and the neck inspiratory muscles (3, 10), it appeared that a line with a slope of 3.0 fits all data remarkably well (Fig. 8).

The observation that the coefficient of proportionality between spec $\Delta Pao$ and the change in muscle length is 3.0 (i.e., similar to the values of maximal muscle tension measured during isometric contractions in vitro) for both the parasternal intercostals and the triangularis sterni is somewhat surprising. To induce a fall in Pao, the contracting parasternal intercostals require that the ribs be displaced cranially. There is, therefore, a shortening of the muscles. In supine dogs, however, the length of these muscles at FRC is $\sim 115\%$ of $L_o$ (7, 8). As a result, during a maximal stimulation at FRC, the canine parasternal intercostals should still operate on the most advantageous portion of their length-tension relationship such that the force developed should be close to the optimal force generated during isometric contractions in vitro. Although the changes in triangularis muscle length during stimulation were not measured in the present studies, the observed rise in Pao also implies a significant (caudal) displacement of the ribs and a significant muscle shortening. However, as previously emphasized, a 1.0-liter passive inflation places this muscle in the immediate vicinity of $L_o$ rather than beyond it. A maximal stimulation of the triangularis sterni at this lung volume should therefore have displaced the muscle to a less advantageous portion of its length-tension relationship, leading to a reduction in $\sigma$. Our failure to detect such a reduction suggests that when the rib cage muscles contract, the loss of force due to muscle shortening plays a relatively small role in the translation of muscle tension into rib displacement and Pao.

Although this issue requires further study, the present findings have three important conclusions and implications. First, for a machine, such as a lever, “mechanical advantage” is the ratio of the force delivered at the load to the force applied at the handle, and by analogy, we have previously defined the mechanical advantage of a respiratory muscle as $\Delta Pao/m\sigma$ (17). On the basis of the present findings, we therefore conclude that the inspiratory mechanical advantage of the triangularis sterni in dogs is substantially greater than the inspiratory mechanical advantage of the parasternal intercostals. Second, because this difference results from the downward orientation of the canine costal cartilages, we speculate that in humans, in whom the costal cartilages are nearly perpendicular to the sternum, the mechanical advantage of the triangularis sterni is almost similar to the mechanical advantage of the parasternal intercostals (Fig. 7). Finally, the findings summarized in Fig. 8 establish that there is a unique relationship between the spec $\Delta Pao$ values of the respiratory muscles, both inspiratory and expiratory, and their fractional changes in length during passive inflation. As a corollary, the spec $\Delta Pao$ of a particular muscle can be estimated simply by measuring its fractional change in length during passive inflation. If the mass of the muscle is also measured, then the $\Delta Pao$ that the muscle would produce during a maximal contraction at $L_o$ can be computed. With this procedure, one could therefore assess the actions of muscles with respiratory effects that cannot be studied directly, such as the respiratory muscles in humans.

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