LETTERS TO THE EDITORS

Comment on Cavallo and Baken (1985)

I would like to comment on the rationale set forth in the article titled "Prephonatory laryngeal and chest wall dynamics," coauthored by Cavallo and Baken, in the March, 1985, issue of the *Journal of Speech and Hearing Research*. Such comment is best appreciated against a backdrop of three quotations.

(1) There is considerable controversy regarding the way in which the prephonatory oppositional chest wall movement is accomplished. Mead, et al. (1974, p. 64) concluded that

> the shift from the relaxation configuration to relative expansion of the rib cage is apparently the result of the dominant action of the abdominal muscles rather than of inspiratory activity of muscles operating on the rib cage.

Several findings reported in the literature argue against this position, however. We have reported that, almost without exception, lung volume changed during the prephonatory chest wall adjustment (Baken and Cavallo, 1981). This implies that the vocal folds were abducted for some period of time during the [adjustment time] interval. If so, some degree of active intercostal participation was likely to have been required to change the rib cage size.

[Cavallo, 1982, p. 13–14]

(2) Hixon, et al. (1976) have suggested that the distortion of the chest wall during speech production in the upright posture is accomplished in a manner similar to the isovolume maneuver. They reported "the adjustment involved is somewhat analogous to an isovolume maneuver in which the major abdominal effort serves to displace the abdominal wall inward and the rib cage outward in the face of a lesser expiratory effort by the rib cage wall" (p. 349). This position implies that the vocal folds are closely approximated during the postural adjustment of the chest wall. Such a conclusion seems tenuous, however, since vocal fold involvement in the posturing of the chest wall would, of necessity, complicate the already complex laryngeal adjustments needed for efficient vocal fold oscillation. If the vocal folds were to close against the intrusion of the abdominal volume into the thorax before speech, phonatory function would most likely be compromised and problems of neuromuscular control confronting the speaker might be enormous.

[Cavallo & Baken, 1985, p. 79–80]

(3) To evaluate the importance of laryngeal action for the posturing of the chest wall, vocal fold position at utterance onset was varied in this experiment. . . . Production of /hɑ/ . . . provided a means of exploring whether or not glottal closure is important to the oppositional displacement that seems to be such a regular feature of prephonatory chest wall behavior. By extension, the /hɑ/ condition also explored the likelihood that rib cage movement during this adjustment is actively generated. If a significant rib cage expansion is observed in the absence of complete glottal closure, then it is likely that such enlargement involves active contraction of the inspiratory rib cage musculature. If, on the other hand, a patent glottis abolishes or severely attenuates rib cage expansion, it is likely that such enlargement was mainly the passive response of the rib cage to intrusion of the abdominal volume into the thorax with the creation of a significantly positive intrapulmonary pressure.

[Cavallo & Baken, 1985, p. 80]

Several misinterpretations and misconceptions are expressed in these three quotations. To begin, the controversy referred to in quotation (1) does not exist. What is said to be a controversy actually is the result of comparing data from the paradigm of Baken and Cavallo (1981) to data from a running speech production activity, which is what Mead, Hixon, and Goldman (1974, p. 64) were referring to in the statement cited in quotation (1).

In addition, the findings referred to in quotation (1) as arguments against the Mead et al. (1974, p. 64) conclusion do not, in fact, argue against that conclusion. Quotation (1) implies that Mead et al. believed that vocal fold adduction and a fixed lung volume were necessary conditions for accomplishing the shift from the relaxation configuration to relative expansion of the rib cage. However, Mead et al. neither state nor imply that vocal fold adduction is required to meet the conditions referred to in their conclusion. Nor do Mead et al. state or imply that lung volume has to remain unchanged to achieve the expansion of the rib cage that results from the dominant action of the abdominal muscles.

Further, there are no direct or implied statements regarding either laryngeal closure or fixed lung volume in the related articles by Hixon, Goldman, and Mead (1973) and Hixon, Mead, and Goldman (1976). Accordingly, the statement in quotation (2) concerning the Hixon et al. (1976, p. 349) "somewhat-analogous-to-an-isovolume-maneuver" suggestion is erroneous in its implication that Hixon et al. believed that the vocal folds had to be closely approximated during postural adjustments of the chest wall.

Cavallo and Baken seem to have generalized from the nonspeech isovolume maneuvers performed by the subjects of Mead et al. (1974) and Hixon et al. (1976) to the general chest wall deformation observed in the same subjects during speech production. The statement from Hixon et al. (1976, p. 349) merely suggests a colloquial analogue that depicts how one might conceptualize deformation of the chest wall as involving simultaneous expiratory activities of both the rib cage and abdomen, but with the latter dominating so that the rib cage is maintained at a high volume despite its expiratory effort. It is puzzling how such a misconception about the Mead et al. (1974, p. 64) and Hixon et al. (1976, p. 349) statements arose, because they were presented in association with extensive data that were obtained through the use of volume-pressure analysis methods. No speculations or inferences were involved. It seems from quotations (2) and (3), that Cavallo and Baken may believe that the performance of an "isovolume maneuver" requires that the larynx be closed. It is, in fact, a simple task to perform an isovolume maneuver (i.e., a configuration adjustment of the chest wall involving no change in lung volume) with the larynx wide open. Witness the pioneering work of Konno and Mead (1967) where the term "isovolume maneuver" was coined. In their work, reference is made *only* to chest wall configuration changes with the laryngeal airway open so that pulmonary pressure could be maintained within specified limits through the monitoring of mouth pressure. The important point, apparently not recognized in quotations (2) and (3), is that it is possible to perform an isovolume maneuver with the larynx fully closed, fully open, or at any intermediate degree of opening. The same is true under conditions where the upper airway is fully closed, fully open, or at any intermediate degree of opening.

Continuing, it is puzzling how, in the context of the Hixon et al. (1976, p. 349) statement, Cavallo and Baken came to suggest in quotation (2) that the functional interaction between the larynx and respiratory apparatus is such that "phonatory function

would most likely be compromised" and "problems of neuromuscular control confronting the speaker might be enormous" when vocal fold adduction and displacement of volume from the abdomen to the rib cage are simultaneous events. Contrary to these statements, there is no reason to believe the phonatory function would be altered by displacement of volume from the abdomen to the rib cage during chest wall posturing or that neuromuscular control problems would result when performing such an adjustment. Data provided in two of the articles cited by Cavallo and Baken (i.e, Hixon et al., 1973, p. 108, Figure 16; Mead et al., 1974, p. 66, Figure 5–11) demonstrate unequivocally that subjects can continuously shift volume back and forth between the abdomen and the rib cage during phonation without compromising phonatory function or posing neuromuscular control problems. Mead et al. are explicit in pointing out the functional separation between laryngeal and chest wall configuration events. In fact, in making reference to the phonatory maneuver that demonstrates this separation they state: "This looks tricky; but try it—you will find it easy" (p. 67).

In quotation (3), it is suggested that: (a) under conditions of laryngeal opening, movement of the rib cage in the inspiratory direction is likely to be caused by action of the rib cage wall's inspiratory muscles; and (b) under the condition of laryngeal closure, movement of the rib cage in the inspiratory direction is likely to be caused by an increase in pulmonary pressure. These notions appear to be related to the reasoning that led to the erroneous implication that Mead et al. (1974) and Hixon et al. (1976) believed that the larynx had to be closed to posture the chest wall via a dominant abdominal action that positioned the rib cage at a higher volume. It is difficult, however, to understand the bases for such notions. Baken and Cavallo (1981) acknowledge the research of Hixon et al. (1973) as having provided the impetus for their chest wall posturing line of research. The Hixon et al. (1973) research contains many utterance conditions involving /hʌ/-syllables that provide the test that Cavallo and Baken indicate is important to their exploration of whether or not glottal closure is relevant to chest wall posturing. How can the generalized deformation of the chest wall for /h/-segments performed in the midrange of the vital capacity be explained in Figures 10, 11, and 18 in the article of Hixon et al. (1973) by mechanism that does not involve a dominant abdominal action? Were the abdomen not to dominate under such an utterance condition, the /h/-segment data mentioned would have to lie to the right of the relative-diameter relaxation characteristics shown for the subjects.

Beyond this juncture, it is important to understand the crux of the misconceptions about respiratory mechanics that run throughout quotations (1), (2), and (3). It is not true, as implied, that rib cage enlargement in the presence of an open larynx needs to be caused by inspiratory rib cage muscles acting on the structure. Nor is it true, as implied, that actions of the abdominal muscles are transmitted to the rib cage to expand it only when the larynx is closed and pulmonary pressure is changed (i.e., in this case, elevated to raise the rib cage).

The central point underlying both of these misconceptions is related to not taking into account the fact that it is changes in *abdominal* pressure that drive the rib cage (e.g., De Troyer, Sampson, Sigrist, & Kelly, 1983; Goldman, 1974; Goldman & Mead, 1973; Hixon et al., 1976; Mead, 1974; 1979). In the present context, elevation in abdominal pressure, which tends to force the rib cage in the inspiratory direction, is germane. Although actions of both the diaphragm and abdominal muscles can elevate abdominal pressure, it is the action of the abdominal muscles that is relevant to the present discussion. Abdominal muscle contraction serves to increase abdominal pressure and to cause the rib cage to expand through at least two known mechanisms. The first of these mechanisms operates through the zone of apposition between the diaphragm and rib cage (i.e., where the diaphragm extends upward along the inner wall of the lower portion of the rib cage). In this zone, increases in abdominal pressure act directly on the inner surface of the rib cage and cause it to expand circumferentially (i.e., the lower rib cage is pushed outward and, as a consequence of its attachments, the entire rib cage moves axially headward). The second of these mechanisms operates longitudinally through the costal fibers of the diaphragm. These fibers insert into the lower ribs and are oriented parallel to the rib cage axis. Increases in abdominal pressure act on the diaphragm so that when the structure is placed under passive tension its costal fibers exert an obligatory inspiratory force on the rib cage. This force causes the rib cage to be pulled upward, whereupon it moves outward and expands circumferentially.

The significant implication of either or both of these mechanisms is that any contraction of the abdominal muscles, through the intermediary of abdominal pressure increase, will cause the rib cage to expand passively. This is true regardless of the status of the larynx, whether closed or open to any degree. It also is true regardless of the status of pulmonary pressure, whether positive, zero, or negative. This can be demonstrated by contracting the abdominal muscles while breatholding (at the larynx) or while resting tidal breathing and noting that under both of these conditions the rib cage rises passively in response to the inward movement of the abdominal wall. The mechanical linkage between the abdomen and rib cage also can be demonstrated by compressing the abdominal wall manually and noting the inward displacement of the abdomen and simultaneous outward displacement of the rib cage. (It should be noted that the abdomen will move further than the rib cage because the abdomen covers a smaller portion of the surface of the lungs.) Clearly, movement of the rib cage in the inspiratory direction does not have to be caused by the contraction of rib cage wall inspiratory muscles. The bases of function suggested by Mead et al. (1974) and Hixon et al. (1976) not only are theoretically consistent with the mechanisms described above, they also are empirically consistent with findings that show that abdominal pressure is elevated throughout the entire running speech breathing cycle. To further illustrate the power of the mechanical linkage between the abdomen and rib cage, it should be noted that contraction of the abdominal wall muscles will elevate the rib cage with the larynx open even if the rib cage wall is paralyzed. In fact, manual compression of the abdomen of a cadaver will elevate the rib cage passively.

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REFERENCES

BAKEN, R., & CAVALLO, S. (1981). Prephonatory chest wall posturing. *Folia Phoniatrica, 33,* 193–203.

CAVALLO, S. (1982). *The laryngeal component of prephonatory chest wall posturing.* Unpublished doctoral dissertation, Columbia University, New York.

CAVALLO, S., & BAKEN, R. (1985). Prephonatory laryngeal and chest wall dynamics. *Journal of Speech and Hearing Research, 28,* 79–87.

DE TROYER, A., SAMPSON, M., SIGRIST, S., & KELLY, S. (1983). How the abdominal muscles act on the rib cage. *Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology, 54,* 465–469.

GOLDMAN, M. (1974). Mechanical coupling of the diaphragm and rib cage. In L. Pengelly, A. Rebuck, & E. Campbell (Eds.), *Loaded breathing* (pp. 50–63). London: Churchill Livingstone.

GOLDMAN, M., & MEAD, J. (1973). Mechanical interaction between the diaphragm and rib cage. *Journal of Applied Physiology, 35,* 197–204.

HIXON, T., GOLDMAN, M., & MEAD, J. (1973). Kinematics of the chest wall during speech production: Volume displacements of the rib cage, abdomen, and lung. *Journal of Speech and Hearing Research, 16,* 78–115.

HIXON, T., MEAD, J., & GOLDMAN, M. (1976). Dynamics of the

chest wall during speech production: Function of the thorax, rib cage, diaphragm, and abdomen. *Journal of Speech and Hearing Research, 19,* 297–356.
KONNO, K., & MEAD, J. (1967). Measurement of the separate volume changes of rib cage and abdomen during breathing. *Journal of Applied Physiology, 22,* 407–422.
MEAD, J. (1974). Mechanics of the chest wall. In L. Pengelly, A. Rebuck, & E. Campbell (Eds.), *Loaded breathing* (pp. 35–49). London: Churchill Livingstone.
MEAD, J. (1979). Functional significance of the area of apposition of diaphragm to rib cage. *American Review of Respiratory Diseases, 119,* 31–32.
MEAD, J., HIXON, T., & GOLDMAN, M. (1974). The configuration of the chest wall during speech. In B. Wyke (Ed.), *Ventilatory and phonatory control systems* (pp. 58–67). London: Oxford University Press.

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A Response to Hixon

It may be useful to begin with a reminder of just what is being examined in the research's rationale Hixon critiques in his letter. During ordinary speech production, the chest wall has a posture that is different from its configuration for tidal breathing. Briefly, and colloquially stated, the rib cage is more expanded and the abdominal wall is more contracted during speech than during nonspeech ventilation. This fact is clearly demonstrated in the relative motion diagrams (for example, Figures 6 and 7 of Hixon, Goldman, & Mead, 1973) to which Hixon refers. Unfortunately, these diagrams provide no information about the time course of events, nor about movements of the chest wall in the intervals before speech onset or between "breath groups." All that is apparent is that chest wall movements during utterances occur to the left of the line representing the relaxation configuration. That is, we find that speech is produced with an enlarged rib cage and a relatively "pulled in" abdominal wall.

What our series of studies (Baken & Cavallo, 1981; Baken, Cavallo, & Weissman, 1979; Baken, McManus & Cavallo, 1983; Cavallo & Baken, 1985) addressed were events occurring in just those intervals about which the relative motion diagrams provide no information. That is, we wanted to examine the time-related movement pattern that brought the chest wall from its relaxed, neutral posture to the configuration that is needed for speech. How long did it take to achieve this posture? Did motions of the chest wall differ according to prespeech lung volume? Were they affected by the ventilatory phase in progress at the time that "speech" was called for ? And finally, what was the larynx doing during this prespeech preparatory interval? Did the vocal folds play an essential role in the chest-wall posturing, or was the larynx simply adjusted for sound production?

In brief, in the context of the task we set for our subjects, we found that during the 100 ms or so before phonation begins the rib cage expands while the abdomen contracts, achieving the requisite speech posture in a rather stereotyped manner. Ludlow, Connor and Melcon (1984), using different instrumentation, found similar prephonatory chest wall motions in their normal subjects.

The first quotation to which Hixon takes exception is not, in fact, from the article he is discussing, but rather is taken from the dissertation (Cavallo, 1982) upon which the article is based. The article itself—which is the published report of our research—makes no mention of "controversy," and focuses on the fact that two goals need to be attained during the period just before phonation: the chest wall must be reconfigured *and* subglottal pressure must be raised to an appropriate level. We pointed out that:

> Gould and Okamura (1974) concluded that "the abdominal musculature plays a key role in producing the subglottal pressure necessary for phonation" (p. 358). If a rapid rise in subglottal pressure does accompany contraction of the abdomen, the vocal folds are implicated as important participants in the chest wall adjustment because they would need to be closely approximated for some period of time before phonatory onset. Vocal fold adduction in opposition to abdominal compression is not an unreasonable strategy for quickly raising alveolar pressure and could explain the significant expansion of the rib cage that typically occurs before phonation" (Cavallo & Baken, 1985, p. 79).

There follows the quotation (Mean, Hixon, & Goldman, 1974, p. 64) that is also embedded in the citation Hixon provides in his critique.

Clearly, the point being made is that a reasonable way to raise the subglottal pressure and to reconfigure the chest wall at the same time is to shift volume from the abdomen to the rib cage while impounding air by laryngeal valving. Certainly, the possibility also exists that the establishment of a new chest wall configuration could be achieved independently of pressurization just prior to phonatory onset. But if we assume *a priori* that a prephonatory chest well shift such as that referred to in Mead et al. (1974) must also result in a significant rise in pressure, then we believe laryngeal closure is implied. If we have overinterpreted Hixon's "somewhat analogous to an isovolume maneuver," or allowed our reading of the statements of Mead et al. to be colored unfairly by our simultaneous need to consider the requisites for pressure generation, we apologize.

To be sure, the actions of the diaphragm and abdominal wall can have complex consequences for the configuration of chest wall components, including the lower rib cage as shown by De Troyer, Sampson, Sigrist, and Macklem (1981) in supine anesthetized dogs. The rib cage movement is dependent, at least in significant part, on the generation of abdominal pressure. Suppose, however, that the diaphragm is not vigorously contracting and its costal fibers are in a relatively short postcontractile state, such as at the end of an inspiration. Under these circumstances, abdominal contraction might serve primarily to elongate the costal portion of the diaphragm (obtrude the abdominal contents into the thoracic space) with only a relatively small increase in abdominal pressure. In this case (and in a case that was eminently possible, given our experimental protocol) rib cage expansion due to abdominal motion might be very much reduced. To address Hixon's final point, we can only state that we have seen no data on rib cage and abdominal movements in fresh, unfixed standing cadavers.

The issue of *passive* enlargement of the rib cage also needs to be considered in light of the requirement that alveolar pressure may increase. For if the rib cage pressures increase, we would expect the enlargement of the mid-to-upper rib cage (where our measurements were made) to be greater than the expansion observed when thoracic pressure remains constant. And an increase in rib cage pressure would require a closed glottis. Hence we said:

> if . . . a patent glottis abolishes *or severely attenuates* rib cage expansion, it is likely that such enlargement was mainly the passive response of the rib cage to intrusion of the abdominal volume into the thorax with the creation of significantly positive intrapulmonary pressure (Cavallo & Baken, 1985, p. 80, emphasis added).

In the end, our research showed that inspiratory muscles do tend to be active during a chest wall reconfiguration (rib cage enlargement, for instance, very frequently preceded abdominal movement), and we adduced evidence that the glottis remains patent until just before phonation is called for.

Would a closed glottis during the adjustment meaningfully have complicated the problem of vocal onset? We believe that Hixon dismisses the possibility too easily. For one thing, he generalizes from findings of Mead et al. (1974) that address the possibility of *maintaining* phonation *during* a rib cage-to-abdomen volume shift. What is at issue in our rationale, however, is whether adjusting the larynx to cope with a configuration shift

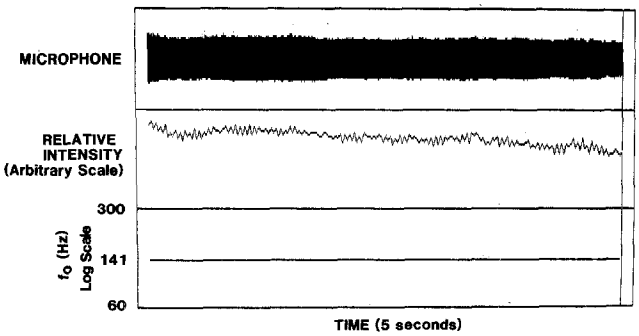

FIGURE 1. Records of relative intensity and fundamental frequency during sustained phonation of the vowel /ʌ/.

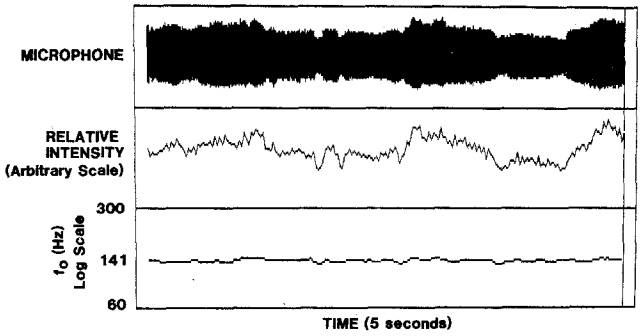

FIGURE 2. Records of relative intensity and fundamental frequency during sustained phonation of /ʌ/ while the subject shifted volume back and forth between the thorax and abdomen.

and an elevation in subglottal pressure is likely to complicate the problem of an immediate phonatory *initiation*. But even putting that issue aside, we find that phonatory function is not as immune to the effects of chest wall shifting as Hixon would have us believe. In fact, we accepted Mead's invitation to try shifting volume back and forth between the rib cage and abdomen during phonation. We asked a male subject who was naive about ventilatory function and who was unfamiliar with any of the research at issue in the present case to do two things. First, our subject was instructed to phonate /ʌ/ for as long as possible, keeping pitch and loudness as steady as possible. Figure 1 shows the result: overall phonatory f_0 and intensity are reasonably stable. Next, the same subject was asked to produce the sustained vowel again, but to pull in and then relax his abdominal wall relatively slowly while he phonated. Figure 2 shows what happened. The stability of both f_0 and intensity degenerated considerably. And that, of course, is just the point. Our subject was able to maintain phonation while shifting volume back and forth. And, indeed, common experience indicates that he could probably maintain phonation under more radical maneuvers as well. But our speaker was apparently unable to effect adequate laryngeal adjustments to compensate fully for these thoracoabdominal volume shifts.

What is needed for speech is not simply phonation, but rather, *stable, controlled* phonation. From the point of view of speech production, we believe we were justified in hypothesizing that were the larynx required to participate in the chest wall adjustment, "phonatory function would most likely be compromised and the problems of neuromuscular control confronting the speaker might be enormous" (Cavallo & Baken, 1985, p. 80). Perhaps, the very highly practiced speakers who were the subjects of the research cited by Hixon did much better—Mead et al. (1974) do not provide any information on this point—but our normally skilled talker did not fare so well.

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REFERENCES

BAKEN, R. J., & CAVALLO, S. A. (1981). Prephonatory chest wall posturing. *Folia Phoniatrica, 33*, 193–203.

BAKEN, R. J., CAVALLO, S. A., & WEISSMAN, K. L. (1979). Chest wall movements prior to phonation. *Journal of Speech and Hearing Research, 22*, 862–872.

BAKEN, R. J., MCMANUS, D. A., & CAVALLO, S. A. (1983). Prephonatory chest wall posturing in stutterers. *Journal of Speech and Hearing Research, 26*, 444–450.

CAVALLO, S. A. (1982). *The laryngeal component of prephonatory chest wall posturing.* Unpublished doctoral dissertation, Columbia University, New York.

CAVALLO, S. A., & BAKEN, R. J. (1985). Prephonatory laryngeal and chest wall dynamics. *Journal of Speech and Hearing Research, 28*, 79–87.

DE TROYER, A., SAMPSON, M., SIGRIST, S., & MACKLEM, P. T. (1981). The diaphragm: Two muscles. *Science, 213*, 237–238.

GOULD, W. J., & OKAMURA, H. (1974). Interrelationships between voice and laryngeal mucosal reflexes. In B. D. Wyke (Ed.), *Ventilatory and phonatory control systems* (pp. 347–360). New York: Oxford University Press.

HIXON, T. J., GOLDMAN, M. D., & MEAD, J. (1973). Kinematics of the chest wall during speech production: Volume displacement of the rib cage, abdomen and lung. *Journal of Speech and Hearing Research, 16*, 78–115.

LUDLOW, C. L., CONNOR, N. P., & MELCON, M. C. (1984, November). *The coordination of prephonatory respiratory and laryngeal movements in spastic dysphonia.* Paper presented at the 26th annual convention of the American Speech-Language-Hearing Association, San Francisco.

MEAD, J., HIXON, T. J., & GOLDMAN, M. D. (1974). The configuration of the chest wall during speech. In B. D. Wyke (Ed.), *Ventilatory and phonatory control systems* (pp. 58–67). New York: Oxford University Press.

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A Response to Hixon

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