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Timing Deficits in Apraxia of Speech

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Summary. This paper deals with a particular aspect of speech motor control in patients suffering from apraxia of speech. Three experiments are reported concerning the phase relations between individual speech gestures. These include the timing of laryngeal, velar and labial movements relative to lingual gestures.

A total of 8 patients and 12 normal controls were examined using speech material which was designed according to appropriate phonetic paradigms. Evaluation was performed on the basis of speech signal parameters referring to the kinematics of inter-articulatory phasing. Deviations of the patient group were found in all three experiments. This suggests that disturbed phase relations of individual speech movements are a general feature of apraxic speech. It is further hypothesized that the described motor symptoms are the origin of a variety of phonemic errors. Support for this view is provided by appropriate examples which refer to the examined paradigms. By this argument, much of the disturbed phonemic structure of apraxic speech may be accounted for by timing deficits.

Key words: Speech motor control – Inter-articulatory phasing – Coarticulation – Phonemic errors – Signal analysis

Introduction

Evolution of man has added to the primate capacity of social signalling by means of a highly developed vocal apparatus (Jürgens and Ploog 1976; Ploog 1981) an even more versatile tool for social communication. The motor system of articulate speech allows the production of acoustic signals of unique complexity and gives rise to language communication as a species-specific faculty of man.

The assessment of vocal behaviour in non-human primates bases heavily on physical descriptions of the vocal repertoire, its cerebral organization being readily examined in animal experiments (Jürgens and von Cramon 1982). In assessing human verbal behaviour the evaluation of the acoustic signal, although requiring more sophisticated methods of analysis, is equally important when a quantification of observations is desirable. In contrast to the experimental work with animals, however, most current knowledge about the structural representation of speech and language has been gained from studying the anatomy of brain lesions following disorders of the central nervous system or traumatic injury.

Of major importance in the investigation of disordered speech and language production is a syndrome often termed "apraxia of speech". Its main symptoms are erroneous sound productions involving mainly distortions, substitutions and additions. The affected patients often demonstrate an effortful and groping syllable-by-syllable manner of speaking (Rosenbek et al. 1984). This pattern occurs most frequently after infarctions of the middle cerebral artery of the dominant hemisphere and is often associated with Broca's aphasia. However, cases of verbal apraxia without aphasia as well as coincidently with fluent aphasias have also been reported (Square et al. 1981; Deutsch 1984). Nevertheless, some disagreement exists both on the justification for considering the described symptoms as a syndromal entity and on the appropriateness of the term "apraxia" (e.g. Martin 1974).

In the tradition of Darley and his coworkers apraxia of speech is classed with the motor speech disorders. Darley et al. (1975) reported that apraxic speech results from an impairment of "certain brain circuits devoted specifically to the programming of articulatory movements". Nonetheless, both the methodology of assessment and the terminology of symptom descriptions have remained largely in a linguistic tradition. In studies which were based on broad transcriptions of apraxic speech the main features of aberrant sound structure were commonly referred to as "substitutions" and "additions" of phonemes, these terms being suggestive of errors at a stage of phonological encoding or organization (Johns and Darley 1970).

More recent studies have focussed upon speech motor control in apraxia of speech, using acoustic and physiological methods of investigation (for a review see Rosenbek et al. 1984). Most of the examined patients exhibited various signs of disturbed temporal control (Freeman et al. 1978; Itoh et al. 1980). These results provided the impetus for investigating the temporal co-ordination of speech gestures in apraxia of speech under the particular paradigm of inter-articulatory phasing. We examined eight apraxic speakers to determine whether the phase relations between individual movements involved in a complex speech gesture are preserved. Three experiments were conducted to assess the timing of lingual movements relative to laryngeal, velar and labial gestures. The method of assessment involved evaluation of speech signal parameters. The results are discussed with respect to possible explanations concerning the phonemic structure of apraxic speech.

Subjects

The study was conducted with 8 patients with apraxia of speech. They were diagnosed as such on the basis of the clinical signs mentioned above. Descriptive data of the patients are given in Table 1. The assessed apraxic symptoms were found in different aphasic syndromes, namely in Broca's

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Table 1. Clinical data of eight patients with apraxia of speech

Subject	Age	Sex	Aphasic syndrome	Time since lesion (months)	Additional dysarthria	Oral-facial apraxia	Spastic hemiparesis	Additional motor deficits
1	52	M	Broca	38	Mild spastic	−	+	Limb kinetic ataxia on the right
2	31	F	Not classifiable	2	−	+	+	−
3	47	M	Wernicke	5	−	+	+	−
4	41	M	Wernicke	6	−	−	+	−
5	53	M	Not classifiable	19	Spastic	−	+	−
6	47	M	Broca	5	−	−	+	−
7	39	F	Broca	6	−	+	+	Ideomotor apraxia on the right and left
8	55	M	Conduction	6	−	−	−	−

Table 2. Principal affected brain structures in eight patients with apraxia of speech

Lesion site	Patients							
	1	2	3	4	5	6	7	8
Internal capsule (ant. limb)	−	−	−	+	+	+	−	−
Caudatum (head)	−	−	−	+	+	−	−	−
Putamen	−	−	−	+	+	+	−	−
Thalamus	−	−	−	−	−	−	−	−
Superior thalamic peduncle	+	−	+	−	−	+	−	−
External capsules	+	+	+	+	+	+	−	−
Arcuate fascicle (superior longitudinal fascicle)	+	+	+	+	+	+	+	+

aphasia, Wernicke's aphasia, conduction aphasia and non-classifiable aphasias (Table 1).

In all cases the lesion was of cerebro-vascular origin. CT scan evaluation revealed that no cortical lesion sites were common to all patients. Yet, in all of them fronto-parietal white matter structures including the corona radiata, the fasciculus longitudinalis superior and the subgyral white matter in these areas were involved (Table 2). From these lesion sites it can be argued that an interruption situated predominantly between the anterior and posterior language areas may be responsible for apraxia of speech. Such a disconnection syndrome might well occur as part of conduction aphasia as well as other fluent aphasias (for a review see Square, in press). Contrary to suggestions by Kertesz (1984) or Marquardt and Sussman (1984) lesions in the neostriatum and/or the anterior limb of the internal capsule did not play a decisive role in our sample. As controls 12 subjects with no known speech or language deficits were examined, 9 of them being male. Their ages ranged from 21 to 63 years with a median value of 35. Note that no particular efforts were made to match the two groups for age or to balance duration and aphasic syndromes in the patient group.

Experiment 1: Lingual-laryngeal Phasing

In the first experiment the interaction of tongue and larynx was studied. Precise temporal control of laryngeal and lingual gestures is required in the production of voiceless lingual plosives (/t/ or /k/) in plosive vowel sequences (Löfqvist 1980). The voice onset time (VOT) can be considered an acoustic index of the phase relation between the lowering gesture of the tongue during oral closure release for the consonant and the vocal fold adduction required for the production of the subsequent vowel. Disturbances in the phasing of lingual and laryngeal movements can be expected to result in altered VOT values as was observed for both Broca's aphasics and patients with apraxia of speech in a number of previous studies (Freeman et al. 1978). Since the VOT is one of the main acoustic cues underlying the voiced-voiceless distinction it is easily understood that disturbed lingual-laryngeal phasing in a speaker may be the cause of a perceptual confusion of say /d/ with /t/ in the listener. Substitution errors of this "voicing" type have frequently been described in the literature (e.g. De Renzi et al. 1966; Trost and Canter 1974).

The experiment reported here was designed to assess the assumed temporal deviations in tongue blade lowering gestures relative to vocal fold adduction, i.e. in alveolar stop-vowel sequences (e.g. /ti/, /tu/ etc.). The relevance of this motor deficit for the voiced-voiceless distinction in apraxic speech was examined.

Methods

All 8 patients took part in this experiment. They were given a phonetic test which was performed in a sound treated room. The speech material consisted of the trisyllabic nonsense words /gətVtə/ where V was one of the vowels /i,y,u,a/. These utterances were embedded in the sentence "Ich habe /.../ gehört" ("I have heard /.../") and were repeated by the subjects upon presentation by an examiner in a quasi-randomized order. Several repetitions were elicited in each subject, yielding a total of 88 samples in the patient group. A large amount of control data ($n = 576$) was obtained by having four of the normal speakers repeat each test sentence 30 times in separate sessions. Care was taken that the four vowels /i/, /y/, /u/, and /a/ be equally distributed in the material.

In order to assess the voiced-voiceless distinction the pairs /midər/ ("corset") – /mitər/ ("tenant") and /budə/ ("stall") – /putə/ ("turkey-hen") were produced twice by each subject. In these pairs the alveolar stop cognates occurred in the context of a high front (/i/) and a high back vowel (/u/).

The utterances were recorded on tape using high quality equipment and then digitized at a sampling rate of 20 kHz

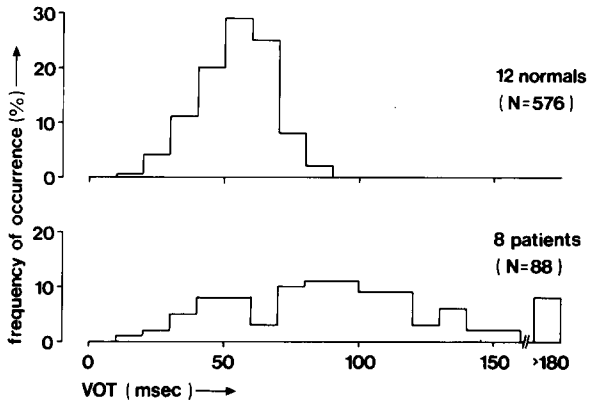

Fig. 1. Distributions of VOT values of pre-stressed inter-vocalic /t/ in normals *(top)* and patients with apraxia of speech *(bottom)*

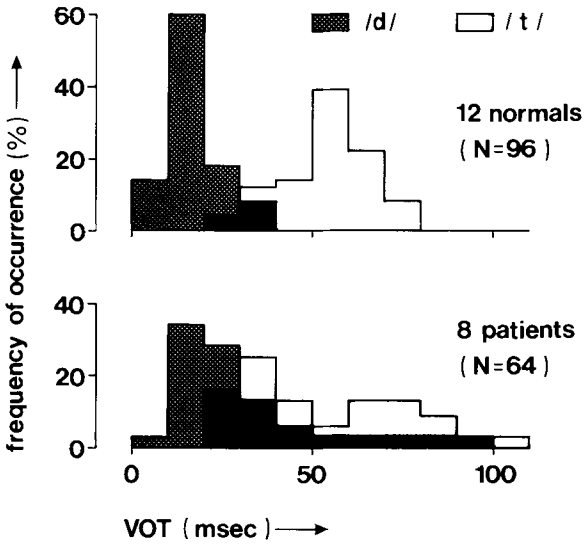

Fig. 2. Voiced-voiceless distinction by VOT differences in post-stressed inter-vocalic alveolar plosives of normals *(top)* and patients with apraxia of speech *(bottom)*

after appropriate low-pass filtering. The voice onset times of the alveolar plosives were determined from the speech signal.

Results and Discussion

The histograms presented in Fig. 1 describe the distributions of VOT values obtained for the two groups on the pre-stressed /t/ in /gətVtə/. The diagram reveals much scattering in the patients' data as compared to the normals, thus reflecting a considerable fluctuation in the timing of vocal fold adduction relative to the lingual closure release. There was a tendency for the onset of voicing to occur later than in normals. Obviously, in agreat number of cases the vocal fold adduction gesture was performed with an overlong delay relative to the tongue blade lowering gesture.

These deviations were measured in each patient individually: in all cases at least 25% of the productions showed VOT values outside the normal range.

With respect to the voiced-voiceless pairs the histograms of Fig. 2 reveal, similarly to Fig. 1, an increased variability of VOTs for the patient group. The values are generally lower in /putə/ and /mitər/ than in the items of Fig. 1 and the increase in the patients' data is less striking, most probably due to the post-stressed position of the considered stop cognates. The crucial point is, however, that the overlap between /d/ and /t/ was considerably larger in the patients than in the normals, in accordance with the results of Blumstein et al. (1980) on four Broca's aphasics. In a number of the present cases highly increased VOT values of /d/ occurred. In a broad transcription approach (as in fact used by various authors, e.g. Johns and Darley 1970) these items would have been classified as substitutions of voiceless consonants for voiced consonants. In view of the data reported here, however, they can be considered to mark the extreme points in a continuum of voicing lags. Such reasoning is difficult to reconcile with the operational phonetic-phonemic distinction of Blumstein et al. (1980) and would seem to agree better with a concept presented more recently by Hewlett (1985). A similar problem in assessing phonetic errors in aphasic speech was subject to a discussion between Ziegler (1984) and Blumstein and Shinn (1984).

Experiment 2: Lingual-velar Phasing

This experiment was aimed at investigating the phase relations between the lowering gesture of the tongue and the associated closure of the velar port as is required in the transition from a lingual nasal consonant (/n/ or /ŋ/) to a non-nasal sound.

During the production of /n/ or /ŋ/ the lowered velum permits the airstream to pass through the nasal tract while the oral cavity is completely occluded by the tongue. Proceding to a subsequent (non-nasal) vowel requires an elevation movement of the velum in combination with a lingual opening of the oral passage. Precise temporal co-ordination of the two gestures is necessary since premature velar elevation results in an inadequate build-up of intra-oral pressure.

The sound pressure level (SPL) was chosen as the appropriate speech signal parameter to describe the smoothness in the diversion of the airstream from nasal to oral. If the involved gestures are adequately synchronized the SPL value should not change markedly during the transition from nasal to oral. If, on the other hand, velar elevation occurs too early, at a moment where the oral passage is still occluded by the tongue, a damping of the SPL should reflect the resulting reduction in the emission of air.

Difficulties of velar timing in apraxia of speech were described by Itoh et al. (1980) in a single case study based on X-ray data. The present experiment was designed to replicate and extend this finding by concentrating explicitly on the inter-articulatory phasing of the considered subjects.

Methods

Seven patients and nine controls participated in this experiment. Phonetic testing was performed under the same conditions as in experiment 1.

The test material consisted of the trisyllabic nonsense words /gətinə/ and /gətanə̨/, embedded in the same carrier phrase as used in the first experiment. Real words containing alveolar and velar nasals after both high and low vowels were added, namely /binə/ ("bee"), /zanə/ ("cream"), /diŋə/ ("things") and /tsaŋə/ ("tongs"). Each of the six test words was repeated twice. One of the patients and two of the nor-

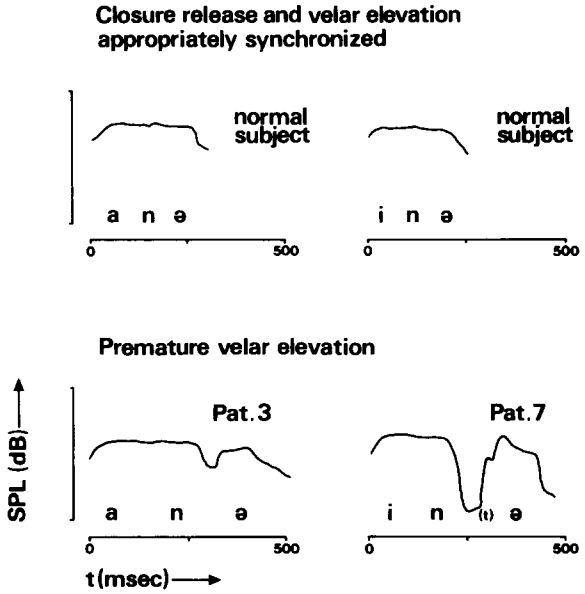

Fig.3. SPL contours of vowel-nasal-vowel sequences in two normal and two apraxic speakers

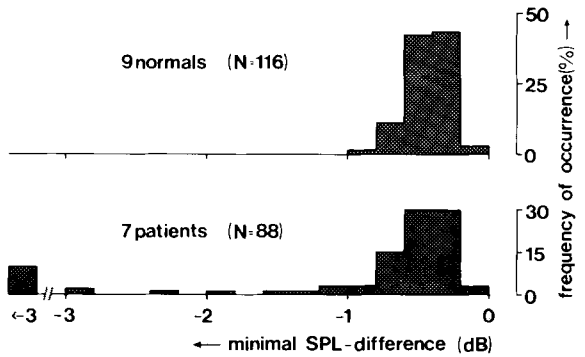

Fig.4. Distribution of minimum SPL differences in nasal-to-vowel transitions for the control group *(top)* and for the patient group *(bottom)*

mals had additional repetitions of the real words, resulting in a total of 88 utterances in the patients and 116 utterances of the normals. The SPL was determined every 3.2 ms with a 12.8 ms window. The raw data were smoothed by a 3-point filter. The minimal value of SPL differences between two consecutive points (a quantity proportional to the SPL gradient) was calculated within the relevant segments, i.e. over the nasal-oral transitions of the test utterances.

Results and Discussion

Figure 3 presents examples of SPL contours for two samples of normal speech and two samples of apraxic speech. The normal cases (upper panels) revealed little SPL differences between oral and nasal segments and smooth nasal to oral transitions. The final fall in the contours signifies the SPL decrease associated with the word ending.

The two contours given in the lower panels, in contrast, are characterized by a marked SPL damping during the transition towards the final vowel. Clearly in these cases a premature elevation of the velum had led to a decrease in sound pressure. With patient 7 the relatively delayed lingual opening gesture was even associated with a plosion-like release of intra-oral pressure which became apparent in a small peak during the post-nasal rise of the contour. In both samples of apraxic speech extremely low SPL gradients resulted within the segments of concern.

The histograms in Fig. 4 show the distributions of the minimal SPL differences between two consecutive points for the normal controls and for the patients. The data base is 12 utterances per speaker. In both groups the major portion of nasal to oral transitions had minimal gradients close to zero, corresponding to SPL contours as shown in the upper two panels of Fig. 3. These values were taken to be indicative of an appropriate synchronization of lingual and velar movements, leading to relatively stable conditions of sound pressure. For the normal speakers this was the rule: in none of the cases could SPL reductions of more than 1.0 dB be measured. Among the patients' utterances, however, SPL changes of up to −6 dB occurred. Of 88 items 13 attained values below −1.5 dB, signifying a more or less sharp damping of sound pressure. A premature occlusion of the velar port was inferred in these cases.

Apart from patients 2 and 4 all patients revealed abnormally high SPL damping (< −1.5 dB) in at least 2 out of 12 tokens. Additional testing with increased requirements on the precise temporal co-ordination of lingual and velar movements, however, showed that patients 2 and 4 actually had the same problem: in sequences where nasal consonants were followed by (oral) plosives instead of vowels the acoustic signs of premature velar elevation could be measured as well as perceived in these subjects. Consequently, the assessed velar mistiming appeared to be a general problem for all of the observed apraxic speakers.

Again, this kind of motor dysfunction bears obvious implications for the phonemic structure of apraxic speech. In extreme cases the violation of time conditions might go so far as to convert nasals completely into orals or vice versa. This type of "substitution" was reported by Trost and Canter (1974) and was observed in four patients of the present study. If the desynchronization span is less extensive the oral counterpart of the intended nasal consonant might be perceived as an appendage, i.e. /n/ as /nd/ or /nt/ and /ŋ/ as /ŋg/ or /ŋk/, respectively. This was the case in examples where after a damping of sound pressure a more or less marked plosion burst could be measured in the speech signal (as shown in the lower right panel of Fig. 3). Velar mistiming can therefore cause the occurrence of both substitutions and additions.

Experiment 3: Lingual-labial Phasing

In the last experiment the interaction of lingual and labial movements was examined under its temporal aspects. The coarticulation paradigm was used to assess the degree to which lip rounding gestures are anticipated in patients with apraxia of speech. In a case study reported previously (Ziegler and von Cramon 1985, in press) the authors were able to show that an apraxic speaker (patient 1 of the present study) failed to initiate the lip rounding movements required for the production of /y/ or /u/ as early as normals did. This approach was extended to the subjects reported here. As in the foregoing experiments the considered gesture – in this case rounding and protrusion of the lips – was examined in its temporal relation

to the lingual lowering gesture associated with the release of an alveolar occlusion. The acoustic influences of the presumed anticipation of a rounded labial configuration were traced in the burst noise of the closure release.

Methods

Phonetic testing was performed as in the previous experiments. With the exception of /gətatə/ and /gətanə/ the trisyllables of experiments 1 and 2 served as the test material. Again, each patient had at least two evaluable attempts at each utterance. The total numbers of speech samples in the two groups were matched approximately.

The degree of anticipatory lip rounding was assessed at the moment of burst release of the plosive /t/ preceding the respective target vowels /i/, /y/, and /u/. A 12.8 ms frame of the burst noise was modelled by the 12 coefficients of a linear predictive filter. The coefficient set of each speech sample was entered into a data pool where both the patients' and the normals' data were grouped according to the features "rounded" (/y,u/) and "unrounded" (/i/) of the target vowel to be anticipated. Discriminant analyses were then performed for patients and controls separately in order to find out whether relevant information on anticipatory lip rounding was contained in the respective speech segments.

To improve the homogeneity of both the control and the patient groups for the purpose of spectral analysis the female subjects were excluded. This left nine subjects in the control group and six in the patient group.

Results and Discussion

For the normal group discriminant analysis yielded a rate of 98% correct classifications of rounded and unrounded items. The rounding quality of the forthcoming /y/ or /u/ could therefore be detected acoustically as early as the lingual closure for the preceding /t/ was released. This corroborates the labial anticipation hypothesis. For the patients the rate of correct classifications was only 72%. A closer look at these results is provided in Fig. 5, where the distributions of discriminant scores resulting from the two analyses are presented. The histogram given for the controls demonstrates a clear separation of /ty,tu/ bursts from /ti/ bursts. There was only a small region of overlap, meaning that the acoustic signs of anticipatory lip rounding were indeed contained in the consonant segment. The patients' data, in contrast, revealed a considerably higher similarity of rounded and unrounded tokens which implies that anticipatory lip rounding often failed to occur in time.

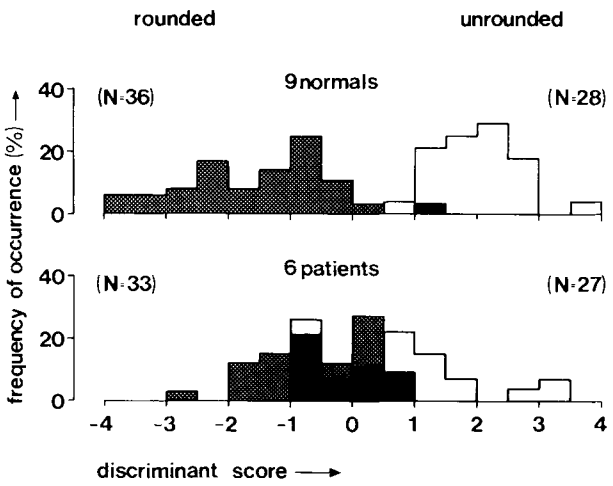

Fig. 5. Distribution of discriminant scores for the distinction of rounded and unrounded vowels on the basis of LP coefficients of the preceding plosive bursts. *Top*, normals. *Bottom* patients with apraxia of speech

This result corroborates the findings of Ziegler and von Cramon (in press) and suggests that disturbed coarticulation is a general problem in apraxia of speech. In fact, each of the examined patients produced at least two test utterances which assumed discriminant scores of the overlap region, i.e. between −1 and +1.

To an even greater extent than the results of experiments 1 and 2 this finding seems to have important implications for our understanding of the deficits underlying apraxic speech since it demonstrates a lack of organizational cohesion in the sequencing of speech movements. In the cases considered here the delayed onset of anticipatory labial movements did not lead to distortions on the intended target vowel. Yet, under more intricate time conditions, e.g. in the production of consonant clusters, a similar disturbance might cause a gross deviation in both the movement trajectory and the acoustic end product and thus give rise to a variety of substitution errors which can hardly be predicted from present knowledge on the mechanics of speech movements.

Conclusions

Taken together the three findings substantiate the assumption that patients with apraxia of speech have a basic problem in phasing individual speech gestures appropriately. It is readily understandable that this motor deficit has a considerable impact on the phonological structure of apraxic speech. The reported experiments provided evidence that disturbed inter-articulatory phasing could be responsible for a number of so-called phonemic errors. It is therefore not only the phonetically distorted sounds that indicate the motor nature of the disturbance, but also a presumably large number of errors that would previously have been described phonologically. Nevertheless the authors do not deny that "true", paraphasic substitutions may coexist with these articulatory deviations, depending highly on the respective symptom configuration, i.e. the associated aphasic syndrome. One should, however, consider as a taxonomic proposal that the term "apraxia of speech" be reserved for the symptom collection of motor type errors.

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