

Subvocal Activity and Auditory Hallucinations: Clues for Behavioral Treatments?

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Abstract

Several investigators have suggested that schizophrenic patients may show an increase in subvocal speech (as measured by electromyographic [EMG] activity) during auditory hallucinations (AH), and that the subvocal activity might be antecedent to the hallucinatory experience. The possible relationship between AH and subvocal activity guided the present approach to studying behavioral interventions for AH.

Duration of AH and EMG activity were recorded from 20 frequently hallucinating schizophrenic patients under baseline and experimental conditions. Three conditions were designed to interfere with activity of the speech musculature and two were imposed as controls.

The data concerning the temporal relationship between EMG activity and self-report of hallucinations were inconclusive. However, one of the experimental conditions (humming a single note softly) reduced the self-report of hallucinations by 59 percent. This condition also increased EMG amplitude over baseline levels. Possible explanations for the effect of humming on hallucinations are discussed.

Auditory hallucinations (AH) continue to present a serious problem for many drug-refractory psychotic patients, as well as for patients who are otherwise optimally medicated. In addition, many patients are reluctant to take neuroleptic medications because of the associated side effects. Hence, a simple and effective behavioral technique for controlling AH would be of great value. However, no such technique has been validated.

Studies of behavioral interventions with AH have usually relied on anecdotal reports from patients rather than on theoretical formulations to suggest possible techniques. Improved effectiveness should result from selecting behavioral techniques that are predicted (by a specific theory of hallucinations) to interfere with the generation of the hallucination. But the mechanism for AH remains unclear. A possible theoretical basis for mechanisms and behavioral treatments is provided by the literature on subvocal activity and AH.

Subvocal activity (or "covert oral behavior") refers to low-amplitude activity in the speech musculature associated with cognitive activity or effort (Sokolov 1972; McGuigan 1978). Subvocal activity is typically measured by electromyographic (EMG) recordings from the lips, chin, or larynx. There is some uncertainty as to whether subvocalizations are specific to linguistic processes or if they reflect general cognitive activity.

Theoretical interest in subvocal activity stems from two widely divergent traditions: the early American behaviorists and the Soviet psychologists. The former (e.g., Watson 1913) took an extreme peripheralist position. They viewed the motoric events of the speech musculature as primary and the events in the central nervous system as relatively unimportant. To the behaviorists, measurement of speech musculature activity presented a way to tap directly the processes of thought and language.

The Soviet psychologists (e.g., Vygotski 1962; Sokolov 1972) took a centralist view. The activity of the speech regions reflected "inner speech," which was considered the

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result of a normal developmental process by which external speech becomes internalized. Hence, EMG recording of the speech regions was a means to study the motoric component of central processes.

Several studies have suggested that AH may be accompanied by an increase in subvocal activity. The earliest studies to make this association were conducted by Gould (1948, 1949). Gould (1948) recorded EMG activity from the lips and chin of hallucinating as well as nonhallucinating psychiatric patients. More of the hallucinating patients showed increased EMG activity than did nonhallucinating patients, suggesting that the hallucination had a psychomotor component. Alternatively, hallucinating patients may show more motor activity *in general*, and the increase in EMG activity may not be specific to the speech region. To control for this alternative, EMG activity would need to be recorded from a nonspeech region.

Gould (1949) presented a single case study and used an amplified stethoscope to record the content of a patient's subvocalizations. The transcripts of a "soundscribe," who listened to the stethoscope, were compared with the subject's own report of the content of the hallucinations. The two transcripts were very similar, indicating a close correspondence between the subvocalization and the hallucinatory experience. Note that this study might have measured vocalizations instead of, or in addition to, subvocalizations. Interestingly, Gould (1949) also recorded a "marked increase of muscle potential, which was practically constant during the entire test" (p. 424) from the lower lip, suggesting that the EMG increase was not limited to the period of the hallucination.

McGuigan (1966) recorded the EMG activity from the chin, tongue, and nondominant arm of a single subject. The subject experienced 25 hallucinations. For 10 of these, the subject was instructed to report the content of the hallucination immediately upon hearing it; for the other 15 hallucinations, the subject pressed a response button when experiencing the AH, but was instructed to remain silent. McGuigan inferred that changes in subvocal activity occurred *before* the hallucinatory experience because both chin EMG activity and whispering increased during the 2-second period before the report of AH. However, this finding of increased EMG activity 2 seconds before onset of hallucination was observed only for the hallucinations on which the subject reported. For the other 15 hallucinations, the chin EMG activity appeared to peak concurrent with the AH onset, and the tongue and arm EMG recordings showed essentially no change.

Interestingly, McGuigan started with 10 subjects, but because the equipment was insensitive and because some subjects did not hallucinate during the session, data for only a single subject were reported. For this single subject, McGuigan presented the data from only one of three testing sessions. This severe attrition undermines the generalizability of the findings.

One additional early study failed to find an association between AH and EMG activity. Roberts et al. (1951) recorded a total of 43 reports of hallucinations from six subjects. Nineteen instances of EMG bursts were recorded, but only four were coincident with the reports of hallucinations. Of the six subjects, one showed a correspondence between AH and EMG activity for the first test but not upon retest. Although

this report argues against a close temporal correspondence between EMG activity and hallucinations, three of the six hallucinating subjects showed *no* EMG activity, which raises questions about the sensitivity of the EMG measures. An additional question concerns the temporal relationship of EMG changes and AH. Although the authors expected the EMG changes to occur simultaneously with the AH, EMG changes might be expected to appear before or after the report of the AH.

Two later studies have yielded conflicting results. Inouye and Shimizu (1970) recorded EMG activity from nine schizophrenic patients and found that hallucinations were associated with increased activity from the speech regions 47.6 percent of the time. (The number of subjects who showed this pattern was not specified.) The scoring procedure was quite liberal: an increase in EMG activity was scored if a burst twice that of the background occurred any time during the report of hallucination. The EMG burst tended to begin within ± 1.5 seconds of the start of the AH.

Junginger and Rauscher (1987) recorded EMG activity from the chin and left forearm in 19 hallucinating (mainly schizophrenic) and 22 nonhallucinating (mainly affective) patients. They compared EMG activity (measured by peak amplitude) during the 3-second period before AH with EMG activity from the 3-second period after AH. No significant differences were noted. However, the authors did not examine EMG measures for time periods that overlapped with reports of AH.

In a separate analysis, Junginger and Rauchser (1987) reported that hallucinators showed greater activity from *both* the speech musculature and forearm than the nonhallucina-

tors, and they concluded that hallucinators may have more muscle activity in general (possibly due to differences in medication or behavioral activity level) but that it is not specific to the vocal musculature. Interestingly, their analyses showed a trend ($p = 0.10$) for an interaction of mean EMG activity (group by electrode placement). When they conducted two one-way analyses of variance (ANOVAs), they found that hallucinators showed significantly more EMG activity than nonhallucinators for the oral region ($p < 0.05$), but not for the forearm ($p < 0.75$). Hence, their results appear consistent with a subvocal explanation. It is not entirely surprising that the interaction is nonsignificant (even if we accept a subvocal explanation) because the hallucinators are not hallucinating for the entire 30-minute testing session. In fact, three hallucinators did not hallucinate at all. In view of these considerations, Junginger and Rauscher's (1987) findings are difficult to interpret and do not necessarily contradict the subvocalization theory of AH.

All the studies mentioned above are complicated by problems inherent when investigators try to quantify and characterize an elusive, subjective experience. For example, we do not know the length of the delay between the hallucinatory experience and the subject's acknowledgment that an AH has occurred (i.e., latency to button press). It has been suggested (R. Cohen, personal communication) that patients might only be able to report hallucinations after they are over, not while they are in progress. Nor do we know whether to expect the changes to occur for the duration of the hallucination, as Inouye and Shimizu (1970) assumed, or as a brief burst preceding or following the AH. Unfortunately, there

are no obvious strategies to test the validity of the button press as a measure of AH.

In summary, the studies that have considered subvocal activity and AH have often used very small sample sizes, poor or absent diagnostic procedures, relatively insensitive measurements, and inappropriate analyses. Nonetheless, the preceding studies have yielded provocative findings and some support for a potentially useful theory.

If AH are associated with subvocal activity, this relationship could be helpful in developing an effective intervention technique. However, the specific nature of this relationship is not entirely settled. Subvocal activity could generate the AH or be a reaction to them. Patients might respond to or mimic their hallucinations and thereby generate increased EMG activity in the speech musculature. Alternatively, the subvocal activity itself might be primary. Patients could generate the hallucinations through their own subvocal activity, which they attribute to some foreign source. "Preliminary research indicates facile participation of the hallucinating patient's own speech mechanism during imagined speaking and hearing" (Gould 1950, p. 118).

In a preliminary effort to test these two competing causal theories, Bick and Kinsbourne (1987) reasoned that if subvocal activity is *primary* to the AH, then any procedure that interferes with the subvocal activity should indirectly interfere with the AH as well. However, if the subvocal activity is *secondary* to the AH, then interference with the subvocal activity should have no effect on the self-report of AH.

Bick and Kinsbourne (1987), instructed patients to perform three simple procedures: opening the mouth wide (the experimental condi-

tion), closing the eyes tightly, and making a fist (two control conditions). After each condition, subjects were asked whether the condition increased, decreased, or left unchanged their hallucinations. It was assumed that opening the mouth would interfere with subvocal activity and therefore reduce self-report of hallucinations. The results indicated that opening the mouth did reduce self-report of hallucinations but the control procedures did not. The authors concluded that AH may result from the disinhibition at a central level that yields an unintended and unrecognized activity of the speech musculature. This formulation converges with Hoffman's (1986) suggestion that the "experience of unintendedness" is central to the hallucinatory experience.

Bick and Kinsbourne (1987) assumed that the reduction of hallucinations would only occur if subvocalizations were causal to hallucinations. Interpretation of their study also rests on the assumption that opening the mouth interfered with subvocal processes. But the safety of this assumption is questionable (McGuigan 1978), and there were no independent measures of EMG/subvocal activity. In addition, there were no quantitative measures of the influence of the procedures on hallucinations.

The current study represents a preliminary effort to organize and understand an inconsistent literature. Two related hypotheses follow directly from the preceding discussion. First, AH may be associated with subvocal activity, and this relationship should be reflected in a specific temporal relationship between EMG activity and the self-report of AH. Second, behavioral techniques that interfere with subvocal activity should reduce AH while control ma-

nipulations should not affect them. Both these hypotheses can be tested empirically.

Method

Subjects. Twenty patients (16 men, 4 women) who received a diagnosis of schizophrenia according to *DSM-III* (American Psychiatric Association 1980) criteria were drawn from the inpatient units of Camarillo State Hospital, California. Diagnoses were based on an expanded version of the Present State Exam (Wing et al. 1974). Subjects were excluded if they had mental retardation, an identifiable neurological condition, a history of drug or alcohol dependence, or if they were over 55 years of age. Subjects were chosen if they complained of frequent auditory hallucinations (many times daily). The mean age for the patients was 33.5 (SD = 8.1), the mean medication in chlorpromazine equivalents was 1,207 mg (SD = 777), and the mean number of years since first hospitalization was 15.5 (SD = 6.4). All interviewers were trained to at least 85 percent agreement for symptom presence by the Diagnosis and Psychopathology Unit of the UCLA Clinical Research Center for the Study of Schizophrenia (R.P. Liberman, Principal Investigator).

Procedure. All testing was conducted in a soundproof room, and data were recorded on a polygraph in an adjacent room. Both self-report of AH and EMG activity were recorded during five conditions: holding the mouth open, biting the tip of the tongue, quietly humming a single note, making a fist, and raising the eyebrows. Subjects practiced each activity before testing. At the beginning of each condition, a card was

placed in front of the subject indicating which activity to perform. The conditions lasted 90 seconds each, were conducted twice within a testing session, and were counterbalanced across subjects. The first three conditions were predicted to interfere with subvocal activity either by reducing it (holding the mouth open and biting the tongue) or by overriding it (humming) (see Erikson and Gustafson 1968). The last two conditions were chosen as control conditions. After each condition, subjects were shown a card that read "swallow and nod when ready." This procedure was designed to minimize swallowing (and hence, movement artifact) during the conditions. In addition, subjects were observed with a video monitor so that movement artifacts could be noted on the polygraph tracing. Baseline recordings (when subjects were simply instructed to "relax") were obtained four times during the testing session.

To record self-report of hallucinations, subjects were instructed to press a response button for the entire duration of the hallucination. This procedure was practiced several times to ensure that the subject understood the task. The duration of the button press was recorded by the polygraph. Time (in seconds) of the button press was the dependent measure for hallucinations.

EMG recordings were obtained from the oral and laryngeal musculature. Electrodes were attached to the upper and lower lips (orbicularis oris) and directly over the larynx, as described by McGuigan (1979). EMG activity was recorded from Beckman silver-silver chloride electrodes attached with Sensormedic adhesive collars. Tracings of the EMG activity were recorded with a Sensormedic EMG Coupler (#9852A) on a Beck-

man Dynograph Recorder Model R612 operating at a chart speed of 10 mm/sec.

We had no means to integrate and average the EMG activity, so we obtained an EMG score with a hand-scoring method suggested by McGuigan (1979). The polygraph tracing for each condition was first divided into a consecutive series of 3-second epochs. The largest peak-to-peak amplitude within each epoch was then selected, and the amplitude of the peak was measured in millimeters. Finally, the mean EMG score (for all epochs within the experimental and baseline conditions) served as the dependent measure for each condition. Two EMG scores were determined: one for the oral region and one for the laryngeal region.

Results

If subjects did not experience a hallucination during the first two baseline recordings (3 minutes), we discontinued testing and brought the subjects back on another day. Most subjects required two or three testing sessions before sufficient AH were recorded. The analyses addressed three questions: the temporal relationship between AH and EMG activity, the effect of the conditions on AH, and the effect of the conditions on EMG activity.

1. **Are the hallucinations associated with increased EMG activity?** The general question concerning the association between AH and EMG activity was addressed by comparing EMG scores during periods of hallucinations with EMG scores during periods of no hallucinations. Only data recorded during the baseline conditions were used because we were interested in the relationship between AH and EMG activity with-

out the effects of the experimental conditions on EMG activity. Three subjects were excluded because they were hallucinating throughout nearly the entire baseline period, and two were excluded because they had no measurable EMG activity during the baseline conditions.

The 3-second epochs were divided into two categories: those that overlapped with a reported hallucination and those that did not. For each category, the mean EMG score was calculated for the oral and laryngeal recordings. An EMG ratio score (hallucination EMG minus nonhallucination EMG activity/hallucination EMG activity plus nonhallucination EMG activity) was calculated for each subject (see figure 1). The scale for figure 1 ranges from +1 (EMG activity during hallucinations only) to -1 (EMG activity during nonhallucinations only) with a midpoint of 0 (no difference). As can be seen from the figure, the scores are distributed around 0. Four of the subjects had positive scores greater than 0.25 for the oral recordings. Two of these subjects, plus one additional subject, had laryngeal scores of 0.25 or greater. The remaining subjects were clustered around 0 or had strong negative scores.

This type of analysis relies on the assumption that increases in EMG activity last for the duration of the report of hallucination. It is plausible that EMG activity increases as a burst near the onset of AH but does not continue for the duration of the report. To examine this possibility, we selected the hallucinations from the baseline condition and divided the tracing into a series of 1-second epochs. The EMG scores for each of the five epochs before the onset of AH and for the one following onset were averaged for each subject. Table 1 shows the means and standard

Figure 1. Electromyographic ratio scores (hallucinating minus nonhallucinating/total)

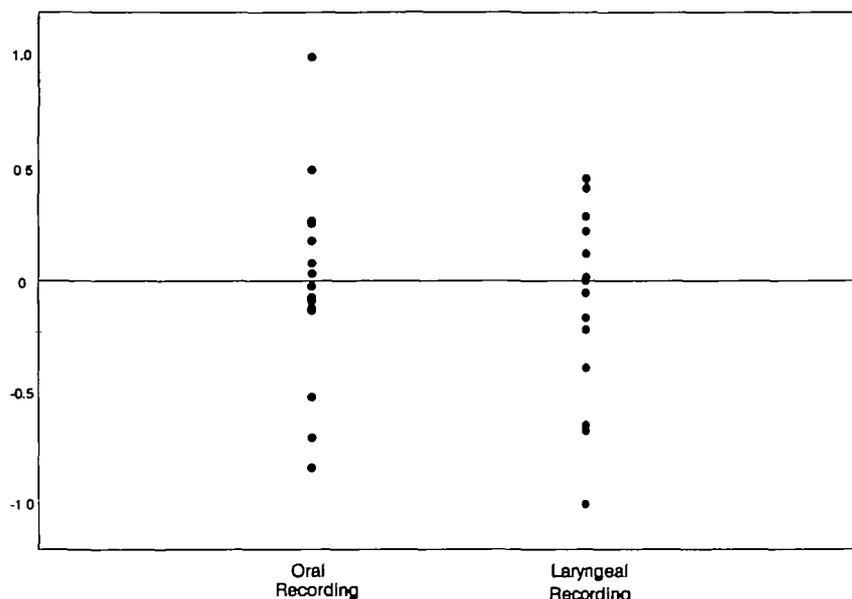


Table 1. Mean electromyographic scores in reference to onset of auditory hallucinations

Epochs	Oral		Laryngeal	
	Mean	(SD)	Mean	(SD)
-5	0.96	(0.77)	0.80	(1.11)
-4	0.74	(0.64)	0.45	(0.51)
-3	0.69	(0.57)	0.55	(0.81)
-2	0.85	(1.02)	0.33	(0.48)
-1	0.80	(0.80)	0.52	(0.79)
+1	0.66	(0.51)	0.77	(1.66)

deviations for these epochs. The data from the oral region of one subject and from the laryngeal region of another were deleted because the values exceeded the mean by more than 3 standard deviations. The *F* values for the repeated measures ANOVA were nonsignificant for linear and quadratic components for both regions, indicating no evidence for a slope before onset.

2. Do the conditions affect the self-report of hallucinations? A re-

peated measures ANOVA for the mean hallucination scores (time in seconds) of the baseline and five experimental conditions revealed a strong effect for the type of condition ($F = 4.54, p = 0.0009$). Each experimental condition was then contrasted with baseline. Figure 2 shows the mean scores for each condition. The humming condition was the only one to reduce significantly the self-report of hallucinations compared with baseline ($p = 0.0012$). This dif-

ference remains significant after correcting for the number of comparisons ($p < 0.01$ with the Bonferroni method). The results are not due to outliers: 17 of the 20 subjects showed a reduction in the humming condition. Figure 3 shows the data as ratio scores (experimental condition/baseline). The humming condition reduced the self-report of AH by an average of 59 percent.

3. Do the conditions affect EMG recordings? Figure 4 shows the means of the EMG scores across conditions for the oral and laryngeal regions. The oral recordings from one subject were excluded due to excessive movement artifact. The EMG measures showed considerable variability, particularly for the humming condition. In the oral region ($n = 19$), the overall F value for the repeated measures ANOVA was 5.18 ($p = 0.0003$). The EMG score for each experimental condition was contrasted with the baseline condition. Humming increased EMG activity above baseline ($p = 0.031$), which was consistent with our prediction that it generates interfering activity. However, this contrast does not remain significant after correcting for number of comparisons ($p > 0.10$ with the Bonferroni method). Raising the eyebrows significantly reduced EMG amplitude ($p = 0.009$) although this was intended to be a control condition. It is possible that raising the eyebrows was associated with a more rigid jaw and mouth. The EMG recordings for the laryngeal region ($n = 20$) showed no significant differences. There was a trend for humming to increase EMG activity in this region ($p = 0.058$). Both the means and the standard deviations increased for the humming condition, indicating that some subjects showed large increases and others showed very little.

Figure 2. Mean time hallucinating (in seconds) for each condition

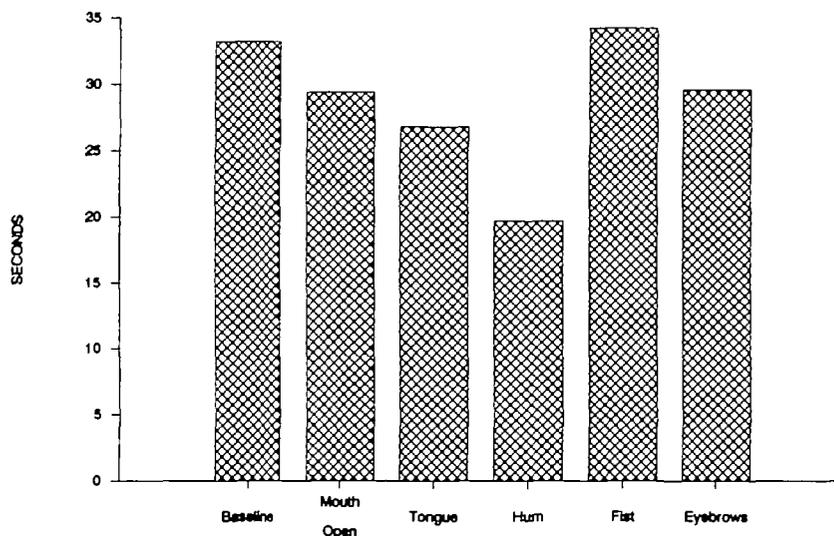


Figure 3. Mean ratio scores (condition/baseline) for hallucinations

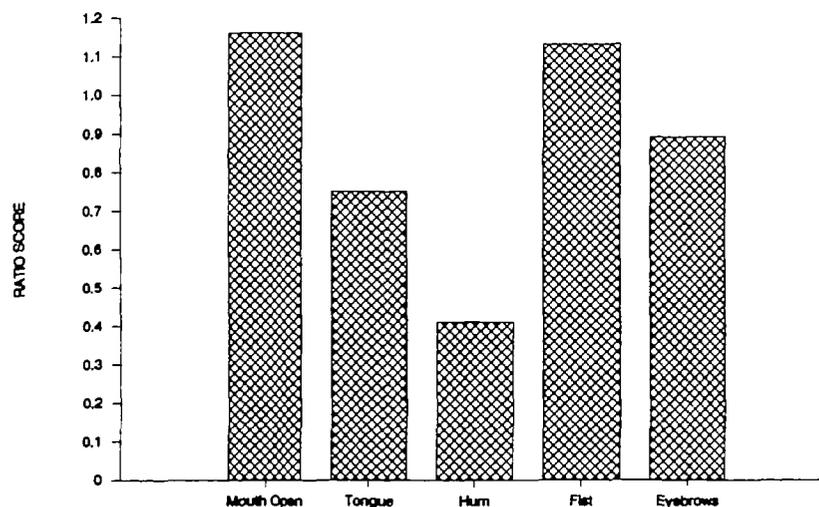
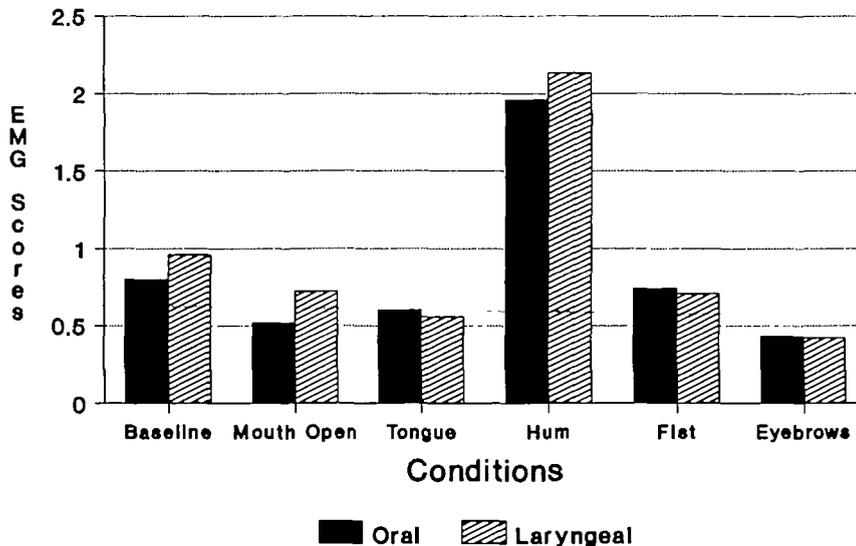


Figure 4. Mean electromyographic scores for oral and laryngeal regions



Discussion

The current study addressed two conceptually related hypotheses: first, that AH are associated with changes in EMG activity, and second, that behavioral techniques that interfere with subvocal activity will suppress AH. Interestingly, the first hypothesis was not supported, but the second one received some support. Hence, the effectiveness of an intervention may not depend on a close temporal relationship between AH and EMG activity.

Although our data did not reveal a clear relationship between AH and increased EMG activity, our method for scoring the EMG data was relatively insensitive. Because our equipment did not integrate the EMG activity, we derived a general index of EMG activity by handscoring the largest peak of each epoch. This method fails to use the data fully because it ignores all activity except for the largest deflection. Future

studies will benefit from more sophisticated ways to average EMG activity.

The major positive finding of the present study is the beneficial effect of quietly humming a single note on self-report of hallucinations. Consistent with our earlier brief report (Green and Kinsbourne 1989), humming reduced AH by 59 percent compared with baseline. This finding gives empirical support to the model under scrutiny. What are some possible alternative explanations for this effect?

It is possible that humming provides an auditory distraction that effectively "drowns out" the AH (Margo et al. 1981). However, subjects were instructed to hum as quietly as possible to minimize auditory interference. Anecdotally, many of our subjects also complained that they were troubled by hallucinations in noisy situations, such as when they were sitting in the day room or listening to television. In addition,

Alpert (1985) found that patients reported *more* hallucinations at moderate levels of white noise than at low levels. A way to test the theory that humming affects hallucinations through auditory distraction would be to record the subject's humming and then play it back to the subject.

A related possibility would be that humming provides a distraction by causing an internal vibration rather than an external noise. This theory could be tested by applying a vibrator to the larynx so that the subject is passively "humming."

Alternatively, humming might require more effort than the other conditions and therefore might have distracted the patients from their AH. Anecdotally, the subjects did not complain about the humming condition. They commented more on the difficulty of raising their eyebrows for the required time period. Furthermore, our patients do not generally find that effortful activities (e.g., classes and groups) reduce the frequency of hallucinations. Nonetheless, we cannot rule out the possibility that the effects are mediated by an interference created by the amount of effort required for humming. This theory could be tested by manipulating the difficulty of tasks while subjects report AH.

It has been suggested that hallucinations in schizophrenia may result from verbal activity in the nondominant hemisphere (Green et al. 1979; Birchwood 1986). Could this theory explain the current findings? Although formulating a melodic line is a nondominant hemisphere task, humming a single note softly is probably mediated by the left hemisphere (Gordon and Bogen 1974). Also, positron emission tomography reveals an increase in blood flow in the *left* temporal lobe during hallucinations (Wood et al. 1987), which

argues against a nondominant locus. We certainly suppose that AH have a central origin, and we regard the subvocalizations as secondary to the central abnormality (see Kinsbourne, in press). However, we favor the left rather than right hemisphere as the locus for the abnormality.

According to the theory that subvocal activity generates the hallucinations, humming might have interfered with hallucinations by overriding the subvocalizations. However, the present data only partially support this theory. In support of the subvocalization theory, the humming condition increased EMG activity. However, raising the eyebrows reduced EMG activity in the oral region but did not reduce report of AH.

As mentioned above, there are no independent measures of AH, and it is possible that the button press does not reliably measure the hallucinatory experience for some patients. Such a source of error might mask a temporal relationship between EMG activity and AH, but it should not change the effects of the experimental conditions on AH because we would expect the error to be relatively constant within each subject across the conditions.

Most of the patients used in subvocalization studies were receiving neuroleptic medications, which commonly have extrapyramidal side effects, including muscular tremor. Perhaps these side effects create a certain amount of motoric "noise" in the system. Although EMG has been used with psychiatric patients for over 40 years, no study has discussed validity issues for this population or tried to demonstrate the validity of EMG as a measure of subvocal activity with medicated schizophrenic patients.

McGuigan (1978) specified the following criteria to assess the validity of EMG activity, as a measure of subvocal activity. First, linguistic activity should increase EMG activity over baseline levels. Second, the increase should be specific to linguistic but not to nonlinguistic tasks. Third, EMG activity should increase with a linguistic task for the speech musculature but not for other musculature. Future studies are encouraged to assess the validity of the EMG procedure by testing patterns of EMG activity across different cognitive tasks and different recording locations. Note that even if EMG is not a valid measure of subvocal activity in medicated patients, it still could show a temporal relationship with AH in some patients. However, interpretation of the relationship becomes more difficult in this case.

In summary, we have conducted a preliminary investigation into an area that has not only substantial theoretical and treatment potential, but also formidable complexities. A review of the literature reveals inconsistent results and numerous methodological and conceptual concerns. Our preliminary data indicate that one of the experimental conditions (humming a single note quietly) reduced the duration of AH by 59 percent. Several possible explanations for this effect have been discussed. The EMG data were inconclusive regarding the possibility that the beneficial effects are mediated by subvocal activity. More sensitive EMG procedures are necessary to test this theory adequately. In addition, basic questions remain unanswered regarding EMG as a valid measure of subvocal activity.

Falloon and Talbot (1981) asked patients who suffered from AH to list all the techniques they used to try to control the hallucinations. No

single method seemed to be particularly effective, and humming was never mentioned. It appears that humming is not used spontaneously by patients even though it is simple, socially appropriate, and apparently fairly effective.

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