A frequent, if not predominant, complaint of persons reporting symptoms of multiple chemical sensitivities (MCS) is that of heightened sensitivity to smells. In this study odor detection thresholds for phenyl ethyl alcohol (a major component of rose oil) and methyl ethyl ketone (a common solvent) were measured in 18 persons exhibiting symptoms of MCS and in 18 matched normal controls. In addition, nasal resistance, blood pressure, heart rate, and respiration rate were determined before and after the olfactory tests. Scores on the Beck Depression Inventory were obtained prior to testing. Although olfactory thresholds were equivalent in the two study groups, the MCS group evidenced significantly higher nasal resistances, respiration rates and Beck Depression Inventory scores. Decreases in systolic blood pressure and pulse were noted in both groups across the test sessions. These results do not support the hypothesis that MCS is associated with greater olfactory threshold sensitivity (at least to the two target chemicals), but do suggest that MCS is associated with depression, increased respiration rate, and decreased nasal airway patency.

The primary goal of the present research was to determine whether persons who evidence symptoms of multiple chemical sensitivities (MCS) display alterations in olfactory sensitivity, blood pressure, heart rate, respiration rate, nasal airflow resistance, and/or psychologic depression. In addition, this work sought to determine whether brief exposure to low levels of either phenyl ethyl alcohol (PEA; the main component of rose oil) or methyl ethyl ketone (MEK; a common solvent) influences such measures.

SUBJECTS AND METHODS

The 18 subjects in the MCS group were recruited from two sources: advertisements placed in newsletters distributed by lay groups concerned about chemical hypersensitivity and referrals from physicians who examine and treat patients complaining of such symptoms (12 women and six men; average age, 46.1 years; SD, 11.2 years). Some physicians believe that MCS is a chronic illness with many manifestations and probably multiple causes.\(^\text{14-16}\) Since no common underlying physiologic basis for MCS has been established and since there is lack of consensus regarding the specific factors necessary for a primary diagnosis of MCS, we operationally defined MCS on the basis of the following: historical evidence of adverse effects to environmental chemicals; the achievement of a high score on a quantified version of Randolph's Environmental Questionnaire\(^\text{16}\) (REQ; an index of somatic reactions to a variety of chemicals and household products); and careful analysis of the patients' medical histories to determine whether a medical basis for their problems, and the most common complaints (eg, edema, fatigue, headache, inflammation, nausea, pain, mucosal irritation, disorientation, and dizziness) are non-specific and rarely suggest selective organ system dysfunction.\(^\text{17,18}\)

Although a heightened sense of smell is among the most common symptoms reported by individuals with apparent somatic hypersensitivity to petroleum based environmental chemicals,\(^\text{14}\) to our knowledge the sense of smell has not been assessed quantitatively in such persons. Chemically sensitive individuals have been the subject of a number of medical reports and books, and are the basis for a nontraditional popular medical movement focused on "environmental illnesses."\(^\text{14-16}\) Unfortunately, careful study of the clinical histories, physical findings, and laboratory tests of patients with a diagnosis of environmental illness does not provide a uniform medical basis for their problems, and the most common complaints (eg, edema, fatigue, headache, inflammation, nausea, pain, mucosal irritation, disorientation, and dizziness) are non-specific and rarely suggest selective organ system dysfunction.\(^\text{17,18}\)
bases other than exposure to chemicals might explain the symptoms. The section of the REQ used to help establish the MCS classification consisted of 91 items to which subjects responded with “like,” “neutral,” “dislike,” or “made sick from.” These categories were assigned the values of 0, 1, 2, and 4, respectively, and the average item score was calculated. The mean (±SD) MCS item score of these patients was 2.38 ± 0.54.

The 18 control subjects were matched on the basis of gender, age (within 2.5 years), ethnic background, and smoking habits to the MCS group and they received equivalent testing (average age, 46.7 years; SD, 12.3 years). These individuals were obtained from a number of sources, including advertisements posted on neighborhood bulletin boards and at public events. To ensure that these individuals did not include persons with MCS, a prerequisite for their participation in the study was an REQ item average score of less than 1.86 (a value that falls 2.5 SDs below the mean of an independent group of 72 persons from the Delaware Valley who were asked to complete the questionnaire in a pilot study). The mean (±SD) REQ item score of the 18 control subjects was 1.38 ± 0.24.

Several aspects of the symptomatology of the subjects with MCS who participated in this study are noteworthy. Three of these individuals reported having their symptoms since early childhood; the remainder reported later symptom onsets with an average symptom duration of 15.6 years (SD, 6.3 years). In addition to reporting hypersensitivities to several types of environmental chemicals, 15 subjects (83.3%) noted somatic reactions to specific foods, with the most offenders being citrus fruits, vegetables, red meat, eggs, and simple sugars. Eleven subjects (61.1%) had histories suggesting that the reactions were initially induced by exposure to a specific chemical, whereas the other subjects were less definite on this point. Sixteen subjects (88.9%) had symptoms indicative of multiple organ system involvement, whereas two (11.1%) had symptoms seemingly confined to a given system (ie, respiratory). The systems reported to be most frequently involved were the central nervous system (88.9%), the respiratory system (66.7%), the gastrointestinal system (66.7%), the cardiovascular system (27.8%), the endocrine system (thyroid, 22.2%), and the integument (16.7%).

General Experimental Design

Before selection as a participant, each subject completed, at home, a detailed medical history questionnaire and the Beck Depression Inventory (BDI). Following our receipt of the completed materials, each potential participant was interviewed by telephone. Persons who evidenced a history of psychiatric or other medical problems that might explain their symptoms were excluded from participation. The BDI scores were not used as a basis for exclusion or inclusion in the study, but they were used later to establish whether depression was associated with MCS.

Olfactory testing was performed in two test sessions separated by a 15- to 30-minute rest period. In the first session, heart rate, blood pressure, respiration rate, and the nasal airflow resistances of the left and right nasal chambers were determined, followed by a bilateral odor detection threshold measurement using PEA. A second set of heart rate, blood pressure, pulse, respiration rate, and nasal airflow resistance measurements was then obtained. In the second session, the same physiologic measurements were taken, followed by an odor detection threshold test of each side of the nose using MEK. After completion of this threshold test, the heart rate, blood pressure, and respiration rate measurements were again obtained. The MEK testing always followed PEA testing, since MEK has a greater capability of stimulating intranasal trigeminal afferents than does PEA, and we anticipated that some chemically sensitive subjects would report adverse reactions and request to terminate testing. However, none of the subjects reported any untoward reactions to the very low concentrations of either odorant used in this study, and all were able to complete both threshold sessions. In a few cases, data are missing for some of the cells of the design because of equipment failure or technician error; in such cases missing cell data were not estimated and analyses were performed on the available data set.

All testing was performed in an environmentally controlled stainless steel chamber specifically designed for olfactory testing. To minimize exposure to outgassing from painted equipment surfaces located in the chamber, such surfaces were covered with aluminum foil, and every effort was made to eliminate or minimize contact between the subjects and any plastic surfaces that might contribute to their problem.

Olfactory Detection Thresholds

The bilateral PEA, single-staircase, forced-choice, odor detection threshold test is described in detail elsewhere. Briefly outlined, a trial consisted of the presentation of two glass sniff-bottles in rapid succession to the subject. One bottle contained 20 mL of a given concentration of the odorant dissolved in propylene glycol, whereas the other contained 20 mL of the diluent alone. The bottles were opened and immediately placed over a subject's nose in a standardized manner. The subject's task was to report which of the two randomly presented stimuli evoked the stronger sensation. If no sensations were perceived or if no difference was apparent between the bottles, the subject was required to guess one or the other bottle (ie, the test was forced choice). No feedback was given as to the correctness of the responses. The staircase was begun at the −6.00 log concentration step of a half-log volume/dilution series extending from −7.50 to −1.00 log concentrations, and was moved upward in full log steps until correct detection occurred on four trials at a given concentration. If a miss occurred on any trial before this time, the concentration falling one log step higher was presented. When four consecutive correct trials occurred at a given concentration, the staircase was reversed and subsequently moved in 0.5-log steps, with either one or two trials at each step (ie, if the first trial was missed, the second one was not given, and the staircase was moved to the next higher concentration). The geometric mean of the last four of seven staircase reversal points was used as the threshold estimate.

The MEK thresholds were determined in an analogous manner, although a separate threshold value was determined for each side of the nose and a ten-stage air-dilution olfactometer was used to generate the stimuli. Ten concentrations (0.6, 1.0, 1.8, 3.2, 5.6, 10.0, 17.8, 31.6, 56.2, and 100 ppm) of MEK were delivered, along with blank air, to a circular, rotating, stimulus-sampling table with 1.5-L stimulus delivery flasks arranged around its circumference (Figure). The subject sampled the contents of a flask from a small glass nozzle. The order in which each naris was tested was determined randomly from trial to trial. The subject wore opaque goggles during testing to eliminate knowledge as to which stimulus was rotated into position for sampling on a given trial. Odorant concentrations were empirically established at the final delivery point using a photoionization detector (model 910, Analytical Instruments, Avondale, Pa). The olfactometer was calibrated before each session using a 250-ppm butadiene span gas and calibration factors supplied by the manufacturer.

Nasal Resistance

Nasal resistance was measured using anterior rhinomanometry. In this test, the subject breathed through a Scott full-face hood bulletin boards and at public events.
mask (lined with aluminum foil) with a pneumotachometer mounted on its airflow port. To measure nasal pressure during the breathing cycle, a 10-em-long tube connected to a differential pressure transducer was sealed inside one naris using surgical tape (Microfoam).25 The pressure and flow information (transduced by a Mercury NR1 nasal resistance system) were digitized (Interactive Structure AI13 interface) and collected, stored, and analyzed on a computer (Franklin 1200) using a procedure analogous to that described by Pallanch and others.25 Data were collected from the left and right nares independently. During sampling, three to four complete cycles of breathing were recorded. An overall nasal resistance measure was computed from the data collected from each of the two nares. Data points for total resistance were derived by adding flows at fixed pressures. In this manner, the corresponding average flows from the left and right data sets were summed at predetermined pressures to derive a total dataset for both inhalation and exhalation portions of the breathing cycle. Following calculation of total nasal resistance, an exponential function was fit to the average pressure/flow values.

To compare nasal resistances across subjects, we adopted the polar coordinate method of Broms et al27 in which resistance is calculated at radii designated on the pressure/flow curve. A successive approximation algorithm was used to determine the intersection of the pressure and flow values of the curves with the radii (R.E.F. and R.L.D., unpublished data, 1988).24 Dependent measures used in our analyses included inspiratory and expiratory resistances at (1) the radius 01 of Broms et al27 and (2) the maximum radius reached by the pressure/flow curve during the sampling epochs. Since nasal resistance is log normally distributed,26 we transformed the measures to log, values before analysis.

Blood Pressure, Heart Rate, and Respiratory Rate Measurements

Blood pressure and heart rate were measured using an automated sphygmomanometer (Taylor A-200 Blood Pressure Monitor). Respiratory rate was determined before the blood pressure measurement by quantifying inhalation-exhalation cycles for 60 s.

RESULTS

Olfactory Thresholds

The mean (±SD) olfactory detection threshold values are presented in Table 1. To assess whether the PEA and/or the MEK thresholds differed significantly between the study groups, the threshold values were subjected to separate univariate group × gender two-way analyses of variance (ANOVAs).28 In the case of MEK, the data from each person’s most sensitive nostril served as the dependent measure. No significant effects of subject group, gender, or their interaction were observed in these analyses (P ≤ .40). The MEK threshold data were also subjected to two multivariate analyses of variance that incorporated, respectively, (1) the left and right nostril threshold data and (2) the most and least sensitive sides of the nose. These analyses also demonstrated no significant group, gender, or interaction effects.

To determine whether a relationship existed in the MCS group between the degree of self-reported chemical hypersensitivity and the olfactory thresholds, Pearson product-moment correlations were computed between the average REQ item score and each of the threshold measures. No significant correlations were found (PEA, r = −.01; MEK, r = −.13; P > .20).

Nasal Resistance

The nasal resistance measures were subjected to separate group × gender × trials (ie, prethreshold/postthreshold testing) ANOVAs (replications on last factor) for the PEA and MEK test sessions. Because the results of the analyses at the 01 radius of Broms et al27 and the maximum radius were analogous, only the data for the 01 radius are presented herein. Since, in a few instances, nasal resis-
tance was too high or maximum flow was too low to calculate a resistance value for subjects with MCS, analyses were performed on the data following exclusion of such cases.

Overall, the MCS group evidenced significantly higher total nasal resistances on both inhalation and exhalation than did the controls both before and after the PEA and MEK test sessions (Table 2; PEA: inhalation $F[1,22] = 6.54, P < .05$, exhalation $F[1,25] = 10.86, P < .01$; MEK: inhalation $F[1,24] = 3.95, P < .05$, exhalation $F[1,26] = 5.78, P < .05$). In the case of MEK, the MCS and control women demonstrated significantly greater average nasal resistances during exhalation than did the men (respective means $\pm$ SDs = 3.21 $\pm$ 4.10 and 1.27 $\pm$ 1.21 cm H$_2$O/L/s; gender $F[1,26] = 5.96, P < .05$).

Exposure to the threshold-level concentrations of the test odorants also appeared to influence, under some conditions, nasal resistance. Thus, the subjects with MCS and the controls showed a significant increase in nasal resistance following exposure to MEK (Table 2; trials $F[1,26] = 4.08, P < .04$). In the case of PEA, the women with MCS demonstrated a decrease in exhalation resistance following exposure to the test odorant, as indicated by a close examination of a significant gender X group X trial interaction (pretest and posttest respective means $\pm$ SDs = 7.45 $\pm$ 6.20 and 1.69 $\pm$ 1.27; $F[1,25] = 5.52, P < .05$). Thus, a breakdown by gender of this interaction revealed no trial effect for men ($F[1,10] = 0.08, P > .70$), but it did reveal a trials X group interaction for women ($F[1,15] = 11.17, P < .01$). Further analysis of this two-way interaction for women revealed a significant trials effect for the subjects with MCS but not for the controls ($F[1,15] = 6.19, P < .05$).

**Respiratory Rate**

Overall, the MCS group had a significantly higher average respiratory rate than did the controls in both the PEA and MEK test sessions (Table 3; three-way ANOVA group main effects: PEA $F[1,27] = 6.73, P < .05$; MEK $F[1,27] = 16.18, P < .001$). Furthermore, men had a lower respiratory rate than women in both test sessions (gender main effects; PEA $F[1,27] = 5.67, P < .05$; MEK $F[1,27] = 8.22, P < .01$). There were no significant differences in respiration rates between the prethreshold and postthreshold determinations for either odorant ($P < .20$).

**Blood Pressure**

Although no group or gender differences were observed for diastolic or systolic blood pressure in either test session, systolic blood pressure significantly decreased in both groups across the prethreshold/postthreshold test interval for both test sessions (Table 4; PEA $F[1,28] = 6.24, P < .05$; MEK $F[1,28] = 4.05, P < .05$).

**Heart Rate**

The average heart rate of the subjects with MCS did not differ significantly from that of the controls during either test session (three-way ANOVAs; group main effect $P > .05$). Similarly, no differences were found between the heart rates of the men and women under either test session (gender main effects; $P > .50$). However, both the subjects with MCS and the controls evidenced significant decreases in heart rate following exposure to PEA but not MEK (Table 5; trials main effects; PEA $F[1,30] = 7.06, P < .05$; MEK $F[1,30] = 0.55, P > .80$). In the case of MEK, a significant group X trial X gender interaction was observed ($F[1,30] = 5.96, P < .05$). Two univariate two-way ANOVAs within each gender revealed a trials effect for men ($F[1,10] = 1.07, P > .30$) but not for the controls ($F[1,15] = 6.19, P < .05$).

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### Table 2. — Mean (± SD) Nasal Resistance Values of Male and Female Subjects With MCS and Controls for Inhalation and Exhalation at Radius 01 of Broms et al37 Before and After PEA (Session 1) and MEK (Session 2) Threshold Testing

<table>
<thead>
<tr>
<th></th>
<th>MCS Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Inhation</td>
<td>Exhalation</td>
</tr>
<tr>
<td>Pretest</td>
<td>2.58 ± 3.52</td>
<td>6.45 ± 5.89</td>
</tr>
<tr>
<td>Posttest</td>
<td>1.59 ± 1.36</td>
<td>1.49 ± 1.21</td>
</tr>
<tr>
<td></td>
<td>2.78 ± 1.99</td>
<td>7.45 ± 6.20</td>
</tr>
<tr>
<td>Posttest</td>
<td>3.21 ± 3.71</td>
<td>1.69 ± 1.27</td>
</tr>
</tbody>
</table>

* MCS indicates multiple chemical sensitivities; PEA, phenyl ethyl alcohol; and MEK, methyl ethyl ketone. Values are in centimeters of water per liter per second.

### Table 3. — Mean (± SD) Respiration Rate of Male and Female Subjects Before and After PEA and MEK Threshold Testing

<table>
<thead>
<tr>
<th></th>
<th>PEA Test Session</th>
<th>MEK Test Session</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Inhation</td>
<td>Exhalation</td>
</tr>
<tr>
<td>Pretest</td>
<td>16.00 ± 6.53</td>
<td>16.25 ± 6.55</td>
</tr>
<tr>
<td>Posttest</td>
<td>16.40 ± 4.56</td>
<td>16.00 ± 4.90</td>
</tr>
<tr>
<td>Normal control group</td>
<td>17.89 ± 4.08</td>
<td>19.56 ± 5.98</td>
</tr>
<tr>
<td></td>
<td>19.38 ± 5.21</td>
<td>19.25 ± 4.27</td>
</tr>
</tbody>
</table>

*PEA indicates phenyl ethyl alcohol; MEK, methyl ethyl ketone; and MCS, multiple chemical sensitivities. Values are respirations per minute.
**BDI Scores**

To determine whether or not the MCS group was more depressed than the matched controls, the BDI scores were subjected to a group x gender ANOVA. Although no significant gender or gender x group interactions were noted ($P_s > .15$), the MCS group did evidence significantly higher depression scores than did the controls (respective means $\pm$ SDs = 6.88 $\pm$ 5.65 and 2.44 $\pm$ 2.46; $F[1,28]$ = 10.98, $p < .005$).

**COMMENT**

The data of the present study suggest that alterations in olfactory threshold sensitivity are not a major component of the symptom complex of patients reporting MCS. Although all of the patients with MCS we tested reported that they believed themselves to have greater-than-normal olfactory sensitivity, none evidenced threshold values markedly outside the general normal range. This disparity between self-perception and objective measurement could be due to several factors. First, although threshold sensitivity may not be altered in such persons, it is possible that suprathreshold sensitivity or the degree to which sensation increases across higher odorant concentrations is altered. Such phenomena were not measured in this work. Second, it is conceivable that patients with MCS are acutely aware of irritant effects of airborne chemicals mediated via trigeminal free nerve endings within the nasal chambers. Because olfactory thresholds are generally lower than trigeminal thresholds,21,29 and because the stimuli of the present study were presented in an ascending manner, the present study unlikely measured such sensitivity. Third, persons who experience adverse reactions to chemicals may become particularly aware of the odors of the offending environmental agents (as well as possibly related odors), leading them to focus on their already keen sense of smell, even though objectively their olfactory sensitivity is normal. Finally, it is possible that heightened olfactory sensitivity occurs in subjects with MCS as a result of stimulation from either combinations of environmental stimuli or agents other than those tested in the present work.

Given the lack of consensus regarding the factors required for the diagnosis of MCS, it is reassuring that the patients with MCS in this study fell within demographic profiles previously reported for persons with this diagnosis.30 Thus, the majority (66.7%) were well educated (15.1 mean years of schooling), and they reported experiencing symptoms before the age of 30 years, with the median number of years of symptoms being 13 years. Primary symptoms reflected the apparent involvement of three major organ systems in the majority of the subjects with MCS: the central nervous system (88.9%), the respiratory system (66.7%), and the gastrointestinal system (66.7%).

A major finding of the present work is that the MCS group evidenced significantly higher total nasal resistances on both inhalation and exhalation than did the matched normal controls, regardless of exposure to the PEA or MEK. Furthermore, exposure to the threshold-level concentrations of MEK resulted in significantly increased nasal resistance in both the MCS and control groups, likely reflecting well-established nasopharyngeal reflexes.31 Interestingly, only the female subjects with MCS evidenced a change in nasal resistance following PEA testing, and this change was in the direction of less nasal resistance. Whether this indicates a greater autonomic reactivity of patients with MCS to odors requires further study.

The fact that we did not observe markedly abnormal nasal resistance and cardiovascular physiologic reactions in the patients with MCS following exposure to the test odors may reflect their low concentration. Thus, occupational medicine studies suggest that most chemically sensitive workers have a threshold for such reactions at a concentration level of around 1% of the threshold limit value (TLV) of the American Conference of Governmental Industrial Hygienists, an exposure standard designed to provide a safe workplace.17 In the case of both MEK and PEA, the test concentrations of the odors were below this value.22

Although there are numerous reports that MCS is associated with nasal congestion,17 to our knowledge, the present study is the first empirical demonstration that the degree of congestion differs significantly from that seen in controls of the same general age and gender. This observation, along with the finding of heightened respiration and heart rates, suggests that persons with MCS have

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**Table 4.—Mean (± SD) Systolic Blood Pressure Values of Test Subjects Before and After PEA and MEK Threshold Testing**

<table>
<thead>
<tr>
<th></th>
<th>MCS Group</th>
<th>Normal Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEA test session</td>
<td>133.56±23.12</td>
<td>122.00±15.20</td>
</tr>
<tr>
<td>Posttest</td>
<td>128.81±21.42</td>
<td>116.50±17.61</td>
</tr>
<tr>
<td>MEK test session</td>
<td>131.19±22.95</td>
<td>119.31±17.11</td>
</tr>
<tr>
<td>Posttest</td>
<td>128.94±19.99</td>
<td>114.69±15.43</td>
</tr>
</tbody>
</table>

*PEA indicates phenyl ethyl alcohol; MEK, methyl ethyl ketone; and MCS, multiple chemical sensitivities. Values are in millimeters of mercury. (See text for details.)

**Table 5.—Mean (± SD) Heart Rate in Beats per Minute of Male and Female Subjects Before and After PEA and MEK Threshold Testing**

<table>
<thead>
<tr>
<th></th>
<th>MCS Group</th>
<th>Normal Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEA test session</td>
<td>81.36±21.28</td>
<td>76.94±13.29</td>
</tr>
<tr>
<td>Posttest</td>
<td>79.38±18.08</td>
<td>74.28±11.01</td>
</tr>
<tr>
<td>MEK test session</td>
<td>72.38±14.68</td>
<td>72.39±10.34</td>
</tr>
<tr>
<td>Posttest</td>
<td>73.69±15.93</td>
<td>71.00±11.80</td>
</tr>
</tbody>
</table>

*PEA indicates phenyl ethyl alcohol; MEK, methyl ethyl ketone; and MCS, multiple chemical sensitivities. (See text for details.)
laborated breathing that may be associated with a number of their somatic complaints. It is important to note in this context that the nasal airway represents the single largest component of man's total airway resistance and significantly influences tidal volume, respiratory frequency, and expiratory time.11,14

The present data confirm the observations of others (eg., Schottenfeld35) that depression is associated with MCS. On average, however, the degree of depression observed in our subjects was only moderate. Unfortunately, it is difficult to disentangle cause from effect, as many of the symptoms of MCS may be caused, or at least exasperated, by depressive and other types of psychologic illness. In cases where the depression is secondary to the MCS, it could result from the disease process itself or from discouragement concerning the lack of understanding and possible treatments for the disorder. It is of interest that patients without MCS who have alterations in smell function (particularly strange odor or taste sensations) also evidence heightened levels of depression (D.A.D., R.L.D., R. Gregg Settle, PhD, et al: unpublished data, October 1988).

The alterations in physiologic and psychologic function of the subjects with MCS are strikingly similar to the environmental illness reaction patterns listed by Bell.41 As noted by Bell, local allergic symptoms could account for the increased nasal resistance, inasmuch as engorgement of the nasal turbinates in an allergic reaction would decrease nasal airflow and thus, increase nasal resistance. Additionally, increased excitation (seen herein as increased respiratory rate) could be a physical manifestation of the reactive process, with depression as an associated symptom.

The present results confirm a number of the largely anecdotal reports of somatic and behavioral symptoms of patients with MCS.41,14 It is apparent from these observations that individuals suffering from MCS are experiencing alterations in a variety of autonomic functions, including respiration and nasal airway patency. However, why or how such symptoms are triggered remains a mystery. As summarized by Mooser,9 there is current interest in the possibility that viruses may be involved, at least in some cases, in such triggering. Impetus for this line of inquiry has been the similarity between the symptom complex of many cases of MCS and that manifested in such disorders as the Epstein-Barr virus syndrome. However, since few scientific data are available on this point, such comparisons must be viewed as highly speculative at the present time.

This investigation was supported by a grant to the University of Pennsylvania from Marilyn Brachman Hoffman for the study of chemical hypersensitivity and by grant NS 16965 from the National Institute of Neurological and Communicative Disorders and Stroke, Bethesda, Md. Ms Hoffman provided a significant contribution to all phases of the project, including the genesis of fruitful hypotheses, literature evaluation, and subject recruitment. Without her financial support, the environmental chamber and olfactometer used in this work would not have been developed.

Mark Ferguson-Segall, PhD, Brian Schwartz, MD, R. Gregg Settle, PhD, and Carolyn Krasnow gave comments on a previous version of the manuscript, and Richard Meinig gave technical assistance.

References