Overreactivity of the Psyche or the Soma? Interindividual Associations Between Psychosomatic Symptoms, Anxiety, Heart Rate, and End-Tidal Partial Carbon Dioxide Pressure

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Current research has all but refuted previous suggestions about the role of hyperventilation as a proximal, common cause of psychosomatic symptoms. As an alternative, it has been proposed that the experience of psychosomatic symptoms is primarily associated with psychological mechanisms, i.e., with enhanced tendencies of distressed individuals to focus their attention on bodily sensations and to evaluate these in a catastrophic manner. Although this hypothesis has received considerable empirical support, physiological influences on symptom reporting have not, as yet, been fully explored. In this study, contributions of psychological and physiological factors were studied among a group of 83 normal healthy male subjects by an assessment of the interindividual relationships between symptom experience in daily life, situational and dispositional anxiety, baseline end-tidal partial carbon dioxide pressure (PCO_2), and heart rate. Trait anxiety and end-tidal PCO_2 each contributed separately to the prediction of the psychosomatic symptom score. Trait anxiety explained nearly one third of the symptom variance, and an additional 4% was explained by PCO_2. Psychological symptoms were more strongly associated with anxiety and somatic symptoms, more strongly with PCO_2. Heart rate only tended to be correlated with symptom reporting. Analysis of covariance among subgroups of extreme-symptom reporters supported the correlational findings by demonstrating that the association between hyperventilation and symptom reporting remained intact when psychological influences were factored out. The findings suggest that reports of psychosomatic symptoms represent two distinct components: one that is primarily psychological (and is unrelated to physiological factors) and a second that reflects objective variance in physiological functioning. The influence of the first component is probably greater than that of the second.

Key words: psychosomatic symptoms, anxiety, heart rate, hyperventilation.

INTRODUCTION

Many apparently healthy individuals regularly complain about psychological and somatic symptoms (1–5). It has frequently been suggested that some of their symptoms are a consequence of hypocapnia (low arterial levels of carbon dioxide), which, in turn, is brought about by episodic or chronic overbreathing (hyperventilation) (6–8). This conjecture is based on the observation that symptoms produced by voluntary hyperventilation often seem highly similar to spontaneous symptoms (9–11). However, claims concerning the role of hyperventilation in spontaneous symptom production have, as yet, lacked a sound empirical underpinning. Moreover, diagnostic tests to identify patients with hyperventilation syndrome by an evaluation of the recognition of symptoms during voluntary hyperventilation have repeatedly been shown to be methodologically and empirically flawed (6, 12–15). On the basis of these findings, several authors raised serious doubts concerning the role of hyperventilation in somatic symptom formation and the validity of hyperventilation syndrome as a distinct clinical disorder (6, 12–14). Alternatively, it has been proposed that the experience of psychosomatic symptoms may be associated with enhanced tendencies of anxious and distressed individuals to focus their attention on bodily sensations and to appraise these in a negative or catastrophic manner (4–6, 16, 17). In fact, there currently appears to be substantial consensus that the experience of psychosomatic symptoms is predominantly tied to psychological rather than to physiological mechanisms.

Nevertheless, a hitherto inadequately explained phenomenon appears to be at odds with the evidence that completely dismisses hyperventilation as a factor in the causation of psychosomatic symptoms. Compared with symptom-free controls, individuals who regularly have psychosomatic symptoms have repeatedly been shown to have somewhat depressed baseline levels of end-tidal partial carbon dioxide pressure (PCO_2) (18–23). Because the tem-

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poral and transsituational stability of individual differences in end-tidal PCO_2 appears to be relatively great (24, 25), this observation suggests that symptom reporters may, across many situations in daily life, be characterized by hyperventilatory tendencies. This, of course, does not necessarily imply that hyperventilation contributes in a major way to the production of the symptoms. Because anxiety and stress have been implicated in mild-to-moderate hyperventilation (26–29) and in psychosomatic symptom experience (4–6, 15–17), an alternative explanation might be that the concurrence of ventilatory and somatic-perceptual overreactivity of symptom reporters is solely due to the underlying relationship that each shares with situational or dispositional distress (6). An important empirical implication of this proposition is that a substantial proportion of the variance in psychosomatic symptom reporting should be predictable on the basis of differences in situational and dispositional distress and that additional, independent contributions of PCO_2 and other physiological factors to the prediction of symptom reporting should be negligible. This hypothesis was tested in the present study by the evaluation of the degree to which individual differences in psychosomatic symptom reporting in daily life could be predicted by end-tidal PCO_2 and heart rate (HR) and by dispositional and situation-specific anxiety. The HR was included in the study to evaluate the specificity of the association between ventilatory and symptom responses. In contrast to hyperventilation, this measure has not previously been implicated in psychosomatic symptom formation.

The design of the study was simple. A group of 83 healthy normal male subjects filled in a state/trait-anxiety questionnaire and a psychosomatic symptom checklist. Subsequently, end-tidal PCO_2 and HR were measured during a 15-minute baseline period. Multiple regression analyses with a predetermined order of entry of variables were used to assess the degree to which several categories of psychosomatic symptoms were predicted by the psychological and the physiological measures. Furthermore, multivariate analysis of covariance was performed on extreme groups of symptom reporters to evaluate differences in physiological activity, corrected for state and trait anxiety.

METHOD

Subjects

Ninety-one male students were recruited by advertisements in university periodicals.[1] They were paid for their participation. Before the commencement of the measurement session, the subjects were interviewed to ensure that no medical condition existed. Because of unexpected cardiac problems that emerged during electrocardiogram (ECG) recording (e.g., ectopic beats) and equipment failure, the data of eight subjects had to be excluded from the analyses. Mean age (± standard deviation) of the remaining 83 subjects was 22.8 years (± 2.5). The ages ranged from 19 to 31 years.

Apparatus

During the experiment, the subjects were seated on an easy chair in the laboratory, with their backs to the measurement equipment. End-expiratory PCO_2 was measured by infrared analysis of the expired air by means of a Beckman LB-2 capnograph (Yorba Linda, CA) (21, 30). In healthy individuals with normal lung function, the measurement of end-tidal PCO_2 is considered to provide a valid approximation of alveolar and arterial levels of CO_2 (21, 31–35). The physiological basis for the accuracy of this measure was presented by Comroe et al. (33) and Lambertsen (35) who reported differences of 1 to 2 mm Hg between end-tidal and arterial measures. Even during extreme hyperventilation, Weimann (8) reported differences of not more than 2 mm Hg among alert, upright individuals. Furthermore, bronchospasm and increased airway resistance does not seem to alter the close relationship between end-tidal and arterial concentrations (36). In our study, the expired air was collected through a 3-mm diameter catheter inserted in an open face mask and positioned toward the intercept of the nasal and oral air flow 5 to 10 mm from the mouth. The drawn fraction of expiratory air proceeded through a heated sample tube to dry the air before analysis. A sample flow rate of 500 ml/min was used. Before each experimental session, the capnograph was calibrated with medical calibration gas that contained 5% CO_2. The output of the LB-2 was corrected for the ambient barometric pressure and was expressed as a partial pressure (in millimeters of mercury). Only breaths that achieved a clear alveolar plateau were included in the analyses. The expired air first mixes with the air in the anatomical dead space, and then the PCO_2 rapidly rises and finally equilibrates to alveolar the concentration at the end of expiration (34, 35, 37, 38). Deformations in the capnograph waveform indicate that the alveolar plateau may not have been reached. Our measurement procedure was similar to one previously used with alert, upright subjects (34, 37) and is in accordance with modern principles of capnography (38). In a pilot study, we found that PCO_2s were identical whether the catheter was positioned about 5 mm within one nostril (18, 21) or inserted in the face mask, as described earlier.

The ECG was measured by a Hellige Servomed ECG monitor (Freiberg, Germany) that used three Hellige electrodes. The phys-

[1] The advertisement invited healthy volunteer subjects to participate in a psychophysiological study of psychosomatic symptoms such as might be experienced in daily life.

iological signals were recorded on a Watanabe six-channel chart recorder (Tokyo, Japan). The data were hand scored across three 1-minute segments (minutes 9, 12, and 15 of the 15-minute baseline period) and were subsequently averaged. Scoring of the physiological data was performed without knowledge of the questionnaire data. The mean HR was obtained by counting the number of R-waves per minute. The mean end-tidal PCO_2 was scored by averaging the breath-by-breath end-tidal amplitudes of the CO_2 waveforms that reached a distinct plateau (21).

Two questionnaires were used in the study. To measure situational and dispositional anxiety, the Dutch version of Spielbergers' State-Trait Anxiety Inventory (39) was utilized. Psychosomatic symptom experience in daily life was evaluated by a 35-item checklist (Table 1) that assessed the frequency with which hyperventilation-related symptoms were experienced in daily life on a four-point scale that ranged from "not at all" to "sometimes" to "regularly" to "often" (11). These response categories were associated with weights that ranged from zero to three. The overall symptom score was obtained by summing the scores for all items. In addition, scores were calculated in a similar way for six separate symptom subscales (I, gastrointestinal symptoms; II, tingling sensations; III, respiratory symptoms; IV, cardiac symptoms and sensations of warmth; V, dizziness and fainting; and VI, psychological symptoms; Table 1). These subscales were based on a principal component analysis of the checklist that was performed on the data of a pool of 546 subjects (see Appendix).

On arrival in the laboratory, the subjects were given a brief outline of the experiment and were interviewed about their health status and other potentially relevant issues. Then, they were asked to fill in questionnaires. After these were completed,

TABLE 1. Percentage of Positive Responses to the Psychosomatic Symptom Checklist for Response Categories "sometimes," "regularly," and "often." Items in the six subscales are listed separately

Item	Sometimes	Regularly	Often*
Gastrointestinal symptoms			
Nausea	39.8	1.2	
Stomach cramps	34.9	1.2	
Shivering	32.5	2.4	
Stomach feels blown up	53.0	6.0	
Tingling sensations			
Tingling in feet	15.7	1.2	
Tingling in legs	8.4	1.2	
Tingling in arms	9.6		
Tingling in fingers	16.9		
Tingling in face	7 2		
Respiratory symptoms			
Unable to breath deeply enough	28.9	1 2	3.6
Suffocating feeling	31.1	2.4	
Need for air	14.5	2.4	
Pressure on chest	15.7		
Cardiac symptoms and sensations of warmth			
Rapid HR	57.8	6.0	
Feeling of heat	48.2	16.9	1.2
Pounding heart	55.4	4.8	
Irregular HR	20.5	1.2	
Feeling of head warmth	25.3	3.6	
Dizziness and fainting			
Dizziness	34.9		
Blacking out	25.3	1.2	
Fainting	4.8		
Psychological symptoms			
Feeling of unrest, panic	45.8	6.0	1.2
Feeling anxious	34.9	4.8	
Tenseness	62.7	16.9	1.2
Unclassified symptoms			
Confused or dreamlike feeling	26.5	1.2	
Fits of crying	14.6		
Toe or leg cramps	42.2	1.2	
Hands tremble	43.4	3.6	2.4
Chest pain around heart regin	31.3	1.2	
Stiffness in fingers or arms	31.3	1.2	
Colds hands or feet	33.7	12.1	
Pressure or knot in throat	41.0	3.6	
Faster/deeper breathing than normal	36.2	2.4	1.2
Tiredness	62.7	18.1	1.2
Headaches	48.2	1 2	

* Blank spaces indicate zero responses.

the physiological sensors were attached. Subsequently, the subjects were asked to relax, and the baseline condition was administered. The total duration of the baseline period was 15 minutes. During the first 8 minutes of the baseline period, the subjects were allowed to adapt to the experimental situation. After this adaptation period, the face mask was attached, and physiological measurements were started. The baseline condition was followed by a number of other experimental conditions (i.e., paced breathing and voluntary hyperventilation) that are irrelevant for the present study. The results concerning these experimental conditions have been published elsewhere (40).

RESULTS

The percentages of positive responses to the individual items on the psychosomatic symptom checklist are given in Table 1, separately for the response categories "sometimes," "regularly," and "often." The overall response rates were high, i.e., 98.8% of the subjects reported experiencing at least one of the psychosomatic symptoms on the checklist sometimes, and 49.4% of the subjects reported experiencing at least one of the symptoms regularly. On the other hand, only 7.2% of the subjects reported often experiencing at least one of the symptoms.

The means, standard deviations, and ranges of the psychological and physiological measures were, respectively, as follows: state anxiety (SA), 31.9 (± 6.3, 20–48); trait anxiety (TA), 34.7 (± 7.8, 21–62); PCO_2, 36.6 mm Hg (± 2.8, 28.7–44.2); HR, 64.1 beats/min (± 9.5, 42.5–97.0). Compared with the distribution of scores of a norm group of male Dutch university students, the mean SA and TA scores were within the range of the fourth and fifth deciles, respectively (39), and could therefore be considered normal. The mean HR also was normal. The mean end-tidal PCO_2 values were consistent with other investigations of normal, mostly young adult subjects in varying postures (21, 31, 32, 34, 41); the mean partial pressures after adaptation varied in these studies from 36 to 38 mm Hg. These end-tidal levels are somewhat lower than the normal arterial PCO_2 values reported in the literature, ranging from 36 to 45 mm Hg (33, 42, 43–45).

Correlations between symptoms, psychological measures, and baseline physiological measures are shown in Table 2. Consistent with our expectation, psychosomatic symptom scores were strongly associated with the anxiety measures. Correlations with TA were generally higher than those with SA. On the other hand, scores in several symptom categories (total symptom score, gastrointestinal symptoms, cardiac/warmth symptoms, and dizziness/fainting) were negatively correlated with PCO_2. Although there were a few weak tendencies for HR to be positively associated with the symptom scores, there were no significant correlations between symptoms and HR. Not presented in Table 2 are the correlations between the psychological and the physiological measures. The only marginally significant r value in this series was between SA and PCO_2 ($r = -.18$, $p < .10$).

TABLE 2. Pearson Correlations Between SA Scores, TA Scores, End-Tidal PCO_2, and HR

Symptoms	SA	TA	PCO_2	HR
Total score	.32**	.56***	−.27*	.20[a]
Gastrointestinal	.17	.25*	−.24*	.04
Tingling	.19[a]	.43***	−.05	.02
Respiratory	.23*	.27*	−15	.21[a]
Cardiac/warmth	.26*	.31**	−.28*	.19[a]
Dizziness/fainting	.11	.22*	−.24*	.11
Psychological	.35***	.68***	−.15	.14

[a] $p < .10$.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

The next question was whether the psychological and physiological measures each contributed independently to the symptom scores. This was assessed by multiple regression (with forward inclusion and alpha-to-enter and remove = .05); SA, TA, PCO_2, and HR were used as predictors (Table 3). To address our major research question, psychological variables were entered first into the model to ascertain whether physiological measures would subsequently provide an independent, supplemental prediction of symptom scores. The results indicated that 35% of the variance in the total symptom score was predicted by TA and PCO_2 together; PCO_2 contributed an additional 4% over the variance already accounted for by TA. In regard to gastrointestinal symptoms, the percentages of variance accounted for by TA and PCO_2 were comparable (6% and 5%, respectively), whereas both predictors together explained 15% of the variance in cardiac/warmth symptoms (TA accounted for 10% and PCO_2, for an additional 5% of the criterion variance). Of the variance in dizziness/fainting symptoms, 6% was explained by PCO_2. Interestingly, in this instance, none of the other predictors added significantly to the prediction. Conversely, tingling, respiratory, and psychological symptoms were only significantly predicted by TA (the percentages of variance accounted for were 18%, 7%, and 46%, respectively). SA and HR in no instance added significantly to the predictions despite the fact that there were several significant zero-order correlations between SA and the symptom scores (Table 2). This, of course, reflects

TABLE 3. Stepwise Multiple Regressions of Psychological Measures (SA and TA) and Physiological Measures (End-Tidal PCO_2 and HR) on Total Psychosomatic Symptom Score and on Symptom Subscale Scores[a]

Criterion	Predictor	B[b]	R	R;2 change
Total symptom score				
Step 1	A	.53	.56	.31***
Step 2	PCO_2	−.21	.59	.04*
Gastrointestinal symptoms				
Step 1	A	.22	.25	.06*
Step 2	PCO_2	−.21	.33	.05*
Tingling symptoms				
Step 1	A	.43		.18***
Respiratory symptoms				
Step 1	A	.27		.07*
Cardiac symptoms and warmth sensations				
Step 1	A	.28	.31	.10**
Step 2	PCO_2	−.24	.39	.05*
Dizziness/fainting				
Step 1	PCO_2	−.24		.06*
Psychological symptoms				
Step 1	A	.68		.46***

[a] Psychological variables were entered first and somatic symptoms, second. Alpha criteria of inclusion and removal = .05. Superscripted significance levels indicate independent contributions to explained variance (summed R^2 change = total explained variance).
[b] Standardized regression coefficient.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

the significant correlation ($r = .52$) between SA and TA; once the influence of TA was factored out, SA was no longer significantly associated with the symptom scores.

To supplement the regression findings, we formed two extreme groups of symptom reporters (lowest and highest 33% of the distribution of total symptom scores; $N = 27$ in each group). The means and standard deviations for the low-symptom (LS) and high-symptom (HS) groups were, respectively, as follows: SA, 30.0 (± 5.7) vs. 33.3 (± 7.1); TA, 30.5 (± 5.7) vs. 38.7 (± 8.2); PCO_2, 37.4 (± 2.3) vs. 35.4 (± 3.3) mm Hg; HR, 61.6 (± 8.2) vs. 67.5 (± 10.7) beats/min. As might be expected, multivariate analysis of variance (MANOVA) indicated that the groups differed with regard to the anxiety measures, Wilks' lambda ($F(2,51) = 8.35$, $p < .001$). TA was higher in the HS group ($F(1,52) = 16.90$, $p < .001$), but both groups were only marginally different in SA ($F(1,52) = 3.59$, $p < .10$). Next, we evaluated between-group differences in PCO_2 and HR. MANOVA revealed a significant multivariate groups effect, Wilks' lambda ($F(2,51) = 5.34$, $p < .01$). PCO_2 was significantly lower in the HS group ($F(1,52) = 5.62$, $p < .05$), and HR was marginally higher in this group ($F(1,52) = 3.45$, $p < .07$). To test whether these group differences would be eliminated after adjustment for the anxiety measures, we performed a multivariate analysis of covariance (MANCOVA). A preliminary test for homogeneity of slopes revealed no significant group by covariate interactions (all F ratios < 1). The MANCOVA yielded a significant multivariate groups effect, Wilks' lambda ($F(2,49) = 5.07$, $p < .01$). The univariate analyses revealed that PCO_2, when corrected for the anxiety measures, was significantly lower in the HS group ($F(1,50) = 4.83$, $p < .05$), and that HR was marginally higher in the HS group ($F(1,50) = 3.78$, $p < .06$).

DISCUSSION

Our findings indicated that between-individual variations in baseline end-tidal CO_2 independently predicted psychosomatic symptom reporting beyond the contribution of individual differences in psychological factors. This independent association between symptom reports and tendency to hyperventilate was apparent for the overall symptom score and for several individual symptom categories. An analysis of covariance performed on extreme symptom groups was consistent with the multiple regression results, i.e., when variance associated with anxiety was factored out, the HS group maintained a lower baseline PCO_2 than did the LS group. Our results, therefore, suggest that not only variations in psychological distress but also individual differences in physiological activity are independently associated with the reporting of psychosomatic symptoms.

These findings, however, leave little doubt about the pervasive influence of psychological dimensions to explain variations among individuals in symptom complaints. Nearly one third of the variance in the total symptom score was predicted by TA, which was consistent with the findings of a number of other reports (1–5) and supported claims that psychosomatic symptom complaints are closely tied to psychological distress (1–6). Nevertheless, the correlations between TA and the scores in different symptom categories suggest a potentially meaningful distinction, i.e., TA was more closely associated with psychological, rather than somatic, symptoms. The percentage of variance in somatic symptom categories explained by trait anxiety was small compared with TA's rather large contribution to the prediction of more clearly psychological symptoms. In contrast, the associations between PCO_2 and symptom reporting were stronger for somatic symptom categories than for the psychological symptom

category. It may be noteworthy that PCO_2 was the only significant predictor of dizziness/fainting symptoms. On the other hand, there was no significant relationship between psychological symptoms and PCO_2. Although issues of statistical reliability of individual measures, including the instability of standardized beta coefficients, must temper any conclusions concerning the relative contributions to prediction of psychosomatic complaints, our findings still might suggest that individual variations in ventilatory activity are more closely linked to physical symptoms, often regarded as psychosomatic, whereas individual differences in psychological traits may be more tightly associated with the psychological aspects of psychosomatic symptom reporting.

Taken together, these findings suggest that somatic symptom reporting has two distinct components, one that is primarily psychological and unrelated to physiological factors and one that at least partially reflects the subjects' perceptions of genuine, objective levels of physiological activity. Actual symptom reports apparently embody a blend of both components, although the influence of the psychological component may often be greater than that of the physiological component. It seems, therefore, fair to conclude that an association between ventilatory activity and somatic symptoms may exist that goes unrecognized in the current literature (4–6, 16, 17). Our findings should, perhaps, mollify assertions that "... the distinction between psychological and physical complaining is clearly arbitrary and inadequate" and that "... self-reported distress represents a single pervasive trait that is expressed through a broad range of negative affect states and somatic complaints" ((5), page 248).

Finally, we should note some potential limitations of the present study. First, it is conceivable that the psychological dimensions assessed (state and trait anxiety) were too limited to characterize interindividual relations between psychological influences, physiological activity, and symptom reporting adequately. For example, somatic anxiety (i.e., specific fear for somatic symptoms) might be more strongly associated with somatic symptoms than our anxiety measures. In the same vein, it could be argued that variations in reduced positive affectivity, sometimes considered the most distinctive marker of depression (46), may be more tightly coupled to hyperventilation than is negative affect. Indeed, low levels of PCO_2 have repeatedly been observed among patients with clinical depression (31, 32, 47). Given these considerations, it is clearly important that the present findings be replicated, both among normal subjects and among clinical populations. Finally, because of the interindividual character of this investigation, we have no direct evidence that bears on the roles physiological and psychological processes may play in the *development* of psychosomatic complaints. This is an extremely important issue for which future longitudinal research is required to resolve. Regarding this aim, our between-subject data, like most of the available scientific literature in this area, only provide hints for possible hypotheses.

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APPENDIX

The 35-item psychosomatic symptom checklist that was used in this study was first published by Grossman and de Swart (11), who assessed the validity of the checklist. However, these authors did not evaluate the underlying factor structure. Therefore, a principal component analysis (PCA) of the checklist was performed on the pooled data of 546 subjects, who had filled in the checklist in the course of several experiments. The subject pool was composed of 273 students, 243 employees of various companies, and 30 military conscripts. The sample that was used in the present study was not included in the pool. In total, there were 399 male and 121 female subjects. In 26 subjects, gender could not be established because it had not been indicated on the questionnaire. The ages ranged from 16 to 63 years, and mean age was 30.2 years (± 10.7). All subjects considered themselves healthy.

PCA with an orthogonal (Varimax) rotation was performed on the 35 items of the checklist. Inspection of the scree plot indicated that a six-factor solution was the most suitable. This solution explained 50.7% of the total variance. The first factor (gastrointestinal symptoms) explained 9.4% of the total variance; the second factor (tingling sensations), 8.5%; the third (respiratory symptoms), 8.7%; the fourth (cardiac symptoms and sensations of warmth), 8.6%; the fifth (dizziness and fainting), 5.6%; and the sixth (psychological symptoms), 10.0% (Table 1). These six factors were considered to constitute subscales that were well interpretable.

The reliability of the total checklist and the subscales was assessed by a calculation of Cronbach alpha coefficients. The standardized alpha coefficient for the total scale is high, .92. Similarly, all subscales had relatively high coefficients, which

ranged from .65 (dizziness/fainting) to .81 (psychological symptoms; all other subscales between .70 and .78). Hence, it was concluded that the total scale and the six subscales were reliable.

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ANNOUNCEMENT

Academy of Behavior Medicine Research
Neal E. Miller Investigator Award

Neal E. Miller was the first psychologist to receive the National Medal of Science, the second president of the Society for Neuroscience, 21st president of the American Psychological Association, and a founder and the first president of the Academy. He pioneered the application of learning theory to behavioral therapies and the use of chemical and electrical stimulation to analyze the brain's mechanisms of behavior, homeostasis, and reinforcement. His career, now spanning more than half a century, exemplifies ingenuity, perservance, integrity, and humanitarian goals.

The annual Neal E. Miller New Investigator Award is to be presented for work imaginatively conceived and carefully conducted before the recipient's appointment as an assistant professor or equivalent rank. Miller said, "Be bold in what you try; cautious in what you claim." In keeping with the Academy's interdisciplinary traditions, the award will be given to M.D. or Ph.D. recipients. It will consist of a plaque and a cash award, and an invitation to attend the annual meeting of the Academy as its guest. Ordinarily the recipient will also be invited to present his or her work in a speical Academy lecture. The following subject matter will be considered:

1. The interaction between behavior and biological mechanisms in homeostasis, the maintenance of health, the pathophysiology of disease, or the susceptibility to illness.
2. Development or evaluation of behaviorally based therapeutic interventions for primary and secondary prevention, or treatment of disease or injury.
3. Basic theoretical or empirical studies in any scientific discipline with implications for behavioral medicine research—this includes instrumentation, methods of analysis, studies of fundamental brain mechanisms including anatomy, physiology, biochemistry, or pharmacology of regulatory systems.

The submitted work must be in the form of either a published article, chapter or book or a manuscript that has been accepted for publication. If it is "in press," a copy of the final letter of acceptance from the editor or publisher should be included. A complete application will also include a current *curriculum vitae*, and in the case of works of multiple authorship, a letter from the supervisor or department chairman, indicating that the applicant had primary responsibility for the project.

All materials are to be sent by February 1, 1995 to the Secretary of the Academy, Dr. Thomas Garrity, Department of Behavioral Science, College of Medicine, University of Kentucky, Lexington, KY 40536-0086; Telephone (606) 323-5308, FAX (606) 323-5350.