Comorbidity Between Balance and Anxiety Disorders: Verification in a Normal Population

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ABSTRACT. Comorbidity between balance and anxiety disorders has been documented in clinical psychiatric and neurological samples. The authors aimed to determine whether the comorbidity of balance and anxiety disorders has an analogous representation in the normal population. Participants were 20 undergraduate students ages 22–29 years. The authors assigned them to high or low trait anxiety groups and performed a balance task in 3 experimental stages: baseline, training, and test. The baseline and test stages consisted of 4 wobbly and 4 stable trials each. The authors measured state anxiety in the form of auditory startle responses (ASRs) during the stable trials. In the baseline stage, the ASR amplitudes were higher in the high trait anxiety participants. In the test stage, the low trait anxiety participants performed the balance task better than the high trait anxiety participants did. These data suggest that the clinical entity designated as a comorbidity of balance and anxiety disorders has an analogous representation in the normal population.

Keywords: anxiety, balance, comorbidity, high trait anxiety, low trait anxiety

RESEARCH ON COMORBIDITY between balance and anxiety disorders was reviewed in the 2001 special issue of the Journal of Anxiety Disorders (Sklare, Konrad, Maser, & Jacob, 2001). Studies have shown that balance disorders are associated with panic disorder, agoraphobia, and acrophobia (Balaban & Thayer, 2001; Pratt & McKenzie, 1958; Sklare, Stein, Pikus, & Uhde, 1990). In addition, vestibular symptoms are common in patients with panic disorder (Jacob, Furman,

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Durrant, & Turner, 1996), and an epidemiological study revealed the prevalence of vertigo in approximately 35% of anxious participants (Andrade, Eaton, & Chilcoat, 1994). Similarly, children with clinical diagnosis of generalized or separation anxiety frequently demonstrate poor balance control (Erez, Gordon, Sever, Sadeh, & Mintz, 2004). Cumulatively, these studies indicate frequent comorbidity among samples of people who had clinical levels of balance or anxiety disorders. The attempts to explain this type of comorbidity relate to interactions between the vestibular and autonomic systems (Yates & Bronstein, 2005).

Studies of normative populations have shown a significant variance in balance performance and anxiety levels (Era et al., 2006; Haywood & Getchel, 2001; Keogh & Sugden, 1985). However, it is unclear whether the comorbidity between balance and anxiety disorders in clinical populations also exists in the normative range of the population. Some studies support the notion that even a low level of balance dysfunction correlates with elevated anxiety. Thus, patients who have mild vertiginous symptoms may score high on anxiety scales (Alvord, 1991). In Bart, Hajami, and Bar-Haim’s (2007) study, they found that among a normative sample of children who were in kindergarten, those who had poor motor skills displayed elevated anxiety and withdrawn behavior when they reached first grade, compared with their better coordinated peers. In addition, a community survey based on questionnaire data revealed that anxiety prevails in individuals who have reduced balance skills (Yardley & Redfern, 2001). If supported by further studies, these findings suggest that individuals with a subclinical balance deficiency or subclinical anxiety may suffer from comorbid deficiency. By establishing the concept of comorbidity in a subclinical population, the issue of anxiety can be brought to the focus of occupational therapists. Clinicians would be able to assert whether improved balance as a result of physical therapy is associated with a reduction in anxiety.

The present study tested the association between balance and anxiety in university students. In line with the notion of comorbidity between balance and anxiety disorders, we predicted that poor balance performance would correlate with high trait and state anxiety. We assessed balance skills by scoring the participants’ performance on a challenging stability platform. We assessed trait anxiety using the Spielberger State–Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Also, we assessed fear provoked by the challenging balance task (i.e., state anxiety) by scoring the amplitude of auditory startle responses (ASRs).

**Method**

*Participants*

Participants were 20 undergraduate students (8 men, 12 women) who were attending an Israeli university and who had either high- or low-anxiety scores. Participants’ ages ranged from 22 to 29 years ($M = 23.9$ years, $SD = 4.7$ years). At the beginning of the academic year, we administered the STAI (Spielberger et al.,
1983) to 230 undergraduate students who were attending Tel Aviv University and between the ages of 22 and 29 years. Of the 230 students, we randomly selected 10 participants (3 men, 7 women) from those who obtained trait anxiety scores in the top quartile of the sample’s distribution and 10 (5 men, 5 women) from those who scored in the bottom quartile. The 10 participants who obtained trait anxiety scores in the top quartile made up the high trait anxiety group, and the 10 participants who obtained trait anxiety scores in the bottom quartile made up the low trait anxiety group, with $M_s \pm SDs = 56.0 \pm 4.4$ and $24.2 \pm 1.8$, respectively, $t(18) = 21.1, p < .0001$. None of the participants had experience in balance activities (e.g., skateboarding, surfing, snowboarding, waterskiing, gymnastics).

Measures

STAI. The STAI subscale (Spielberger et al., 1983) of trait anxiety consists of 20 items. The summed score provides an index of trait anxiety and higher scores represent a higher level of anxiety. The STAI demonstrated sound reliability and validity (Spielberger et al.).

Balance performance. We measured balance performance by using the stabilometer apparatus, which was a 65- × 105-cm platform resting across an axle (Lafayette 16030; Keogh & Sugden, 1985; see Figure 1). Each trial started with the left side of the platform on the ground. Participants experienced wobbly and stable positions (i.e., trials) in alternate order. For the wobbly position, we asked the participants to place their feet on either side of the platform’s center and informed them that their task was to balance the platform in a horizontal position. For the stable position, we asked the participants to position their feet safely to the right of the platform’s center. In this position, the platform was tilted 15° from the floor with its proximal edge leaning firmly on the floor, thus not requiring any balancing effort. The stabilometer was set to a threshold level of a 5° deviation from the fully horizontal position, and its binary output was stored using the CED 1401 interface. We analyzed balance performance offline as duration of horizontal position, defined as time period with deviation from the platform of less than 5° from the fully horizontal position. The stabilometer test has exhibited strong reliability and validity (Murray, 1982; Wulf, Weigelt, Poulter, & McNevin, 2003).

ASRs. ASRs are motor responses triggered by loud tones and mediated by a brainstem reflex circuit (Davis, 2006; Davis, Gendelman, Tischler, & Gendelman, 1982). They are potentiated in animals by states of fear (Davis et al.) and in humans by experimentally induced anticipatory anxiety (Grillon, Falls, Ameli, & Davis, 1994) and aversive states (Smith, Bradley, & Lang, 2005). The increase in ASRs also correlates with the state scale of the STAI (Grillon, Ameli, Foot, & Davis, 1993; Smith et al., 2005). The amygdala output mediates fear-related potentiation of ASR because amygdala stimulation potentiates the ASR, and
the amygdala lesion reduces the potentiation effect (Hitchcock & Davis, 1987; Rosen & Davis, 1988). In human participants, the ASR is frequently monitored as a blink response to a loud tone that is measured by electromyography (EMG) activity of the orbicularis oculi.

In the present study, white noise auditory pulses triggered the ASRs. These pulses were 95 dB SPL (i.e., sound pressure level) in intensity and 50 ms in duration, with instantaneous rise time to maximal loudness (Campden Instruments Limited, Serial Model 530) presented binaurally through headphones. We recorded the ASRs as EMG eye blinks by using a pair of electrodes that we applied to the participants' right orbicularis oculi muscle. The EMG signal was amplified with Grass Instruments bioamplifiers, digitized at 1 kHz, and stored and filtered offline to a 100–500 Hz band. After rectification, we integrated the signal over 40-ms epochs with successive epochs advancing at 1-ms steps. We then presented the result of each epoch at the midpoint of each epoch. The amplitude of the ASR was calculated as the difference between the maximum of the signal in the 150 ms after the auditory pulse onset and the background level averaged during a 150-ms period preceding the onset of the pulse. We controlled
and recorded the stimuli during the experiment by using the CED 1401 interface and performed the offline analysis by using the Spike2 program (version 6.1, Cambridge Electronic Design).

**Procedure**

We invited the selected participants to the laboratory and habituated them to the experiment room. All of the participants read and signed an informed-consent form. The instructions that we gave to the participants described the successive experimental stages (i.e., baseline, training, test). We then attached electrodes to the participants to record their ASRs. The procedure was controlled by the CED 1401 interface.

*Baseline stage.* Figure 2 shows a schematic representation of the baseline and test stages. We engaged the participants in the dynamic balance task on the stabilometer for approximately 5.5 min. This task consisted of four wobbly and four stable trials presented in alternating order and interspersed by variable intertrial intervals (ITIs). The wobbly and stable trials both started with a differential fear-conditioning period of 6 s to induce anticipatory fear in the wobbly trials but not in the stable trials. During the conditioning period, the participants assumed a stable position on the stabilometer (i.e., no balance effort) and were asked to look straight ahead at a board of lights. A red light that was lit for the entire 6 s signaled a wobbly trial: When the red light was turned off and the white light was turned on, the participant started balancing on the stabilometer platform for 20 s until the white light was turned off. In the alternate trials, a green light that was lit for 6 s signaled a stable trial: When the green light was turned off and the white light was turned on, the participant maintained a stable position on the stabilometer platform for 20 s until the while light was turned off.

This schedule of events conformed to a differential fear-conditioning procedure in which the red light served as (CS+), the conditioned stimulus followed by unconditioned stimulus (US), which predicted the aversive wobbly position, and the green light served as (CS–), the conditioned stimulus not followed by unconditioned stimulus, which predicted the stable state. The turning off of the white light signaled the start of an ITI, during which participants were required to either attain a stable position on the stabilometer after a wobbly trial or maintain a stable position on the stabilometer after a stable trial in preparation for the next trial.

We recorded the ASRs to determine whether the discriminative conditioning procedure induced a state of fear in response to seeing the red light, which signaled the wobbly trials. Note that we recorded all of the ASRs only when the participants were in a stable position on the stabilometer. Thus, the ASR recordings were not confounded by motor movements associated with the balance task. Before the baseline stage, three ASR pulses were recorded to ensure
a rapid habituation process (10–30-s ITIs). We then recorded the ASRs on the background of the red CS+ and green CS– lights (4.5 s after the light became lit) in two wobbly trials and in two stable trials. To reduce the contingency between the CSs and the aversive loud auditory pulse used to trigger the ASRs, we recorded two additional ASRs during the ITI while the participants waited for the next trial.

**Training stage.** In the training stage, the participants practiced dynamic balance on the stabilometer (i.e., a continuous wobbly state) for 3 min.

**Test stage.** The test stage, which was an exact replica of the baseline stage, served to test the effects of balance training on balance performance and acquired state anxiety that the ASRs measured.
Results

Balance Performance

We analyzed balance performance, scored as a percentage of time in a horizontal position on the stabilometer, by using analysis of variance (ANOVA) with group (i.e., low vs. high trait anxiety) as a between-participants factor and stage (i.e., baseline vs. test) and trial (i.e., four wobbly trials at each stage) as within-participant factors. The data analysis revealed better balance performance in low-anxiety participants versus high-anxiety participants, $F(1, 18) = 6.2, p < .03$ (see Figure 3). A significant stage effect confirmed improvement in balance performance during the test stage versus the baseline stage, $F(1, 18) = 93.2, p < .001$. The improvement of the low-anxiety participants exceeded that of the high-anxiety participants, and that improvement was confirmed by a significant Stage × Group interaction, $F(1, 18) = 9.3, p < .01$. Separate ANOVAs for the baseline stage revealed a significant improvement of balance performance across trials, $F(3, 54) = 7.0, p < .01$. However, the ANOVAs did not reveal a group effect, $F(1, 18) = 0.8, p > .05$. Separate ANOVAs for the test stage revealed that the low-anxiety participants performed better than the high-anxiety participants did, $F(1, 18) = 12.2, p < .01$. In addition, only the low-anxiety participants improved across trials, and that finding was confirmed by a significant Trial × Group interaction, $F(3, 54) = 5.1, p = .01$.

![Graph showing balance performance](image)

**FIGURE 3.** Balance performance scored as percentage of time in a horizontal position on the stabilometer ($M ± SEM$) in low anxiety and high-anxiety participants. Data shown for the four wobbly trials during the baseline and test stages.
To further demonstrate the relation between balance and anxiety, we regrouped participants on the basis of their average balance performance during the test stage. Using the criterion of at least 70% of the time in a horizontal position (approximately the average performance at the test stage), we defined 12 participants as high-balance performers and 8 participants who scored lower than this limit as low-balance performers. Table 1 presents the surplus of high-balance performers among the low-anxiety sample and the surplus of low-balance performers among the high-anxiety sample, $\chi^2(1, N = 10) = 7.5$, $p < .01$, two-sided.

ASR Reactivity

We analyzed the ASR amplitude on the habituation trials using ANOVAs with group (i.e., low vs. high trait anxiety) as a between-participants factor and stage (i.e., baseline vs. test) and ASR sequence (i.e., three successive ASRs in each stage) as within-participant factors. Habituation of ASR was observed before the baseline but not before the test stage and supported by a significant Sequence $\times$ Stage interaction, $F(2, 36) = 4.4$, $p < .02$. In effect, the habituation resulted in significantly lower ASR amplitudes before the test versus baseline stage, $F(1, 18) = 32.4$, $p < .001$. Group effect and the interactions with group were not significant ($p > .05$).

We analyzed the ASR amplitude during the conditioning procedure using ANOVAs with group (i.e., low vs. high trait anxiety) as a between-participants factor and stage (i.e., baseline vs. test), trial type (i.e., wobbly vs. stable), and ASR sequence (i.e., two ASRs for each trial type) as within-participant factors. Trial type had no effect on ASR amplitude, $F(1, 18) = .003$, $p > .05$; therefore, we excluded this variable from the analysis. Figure 4 shows that during the baseline stage, the ASR amplitude was greater in the high-anxiety participants versus low-anxiety participants, but because of a more pronounced decrease in amplitude across the stages, the high-anxiety participants did not differ from the low-anxiety participants during the test stage. These changes were supported by a significant decrease in the ASR amplitude in the test stage versus baseline.

| TABLE 1. Distribution of Participants Across High and Low Levels of Balance Performance and Across High and Low Levels of Trait Anxiety |
|---------------------------------|-----------------|------------------|-----------------|-----------------|
| Trait anxiety                   | High            | Total            | Low             | Total           |
|                                 | Men  | Women | Total | Men  | Women | Total |
| High                            | 1    | 2     | 3     | 2    | 5     | 7     |
| Low                             | 4    | 5     | 9     | 1    | 0     | 1     |
stage, $F(1, 18) = 9.8, p < .01$, and a significant Stage × Group interaction, $F(1, 18) = 4.4, p < .05$.

**Discussion**

Clinical and experimental literatures provide evidence for the comorbidity between balance and anxiety disorders. The present study tested whether a similar correlation between balance and anxiety could be demonstrated in a normal sample. We screened participants for low trait anxiety versus high trait anxiety on the basis of the STAI (Spielberger et al., 1983). The balance performance on the stability platform for the high trait anxiety participants was inferior to that of low trait anxiety participants. This finding suggests the coexistence of inferior balance and elevated anxiety in the normal population. As in other studies, the present findings provide no indication of the causal relations between the two variables. Also, the findings do not reveal whether there is a possible mediation by a third variable.

Our primary question concerned whether high trait anxiety is associated with compromised balance performance. The dynamic balance task was difficult for all of the participants. On average, during the baseline stage, they were able to balance the platform for less than 50% of the given time. Confronting this challenge, the participants showed a gradual improvement in balance performance across the successive trials of the baseline and test stages. Because of this
performance profile, the groups could differ at the initial level of performance or in the rate of subsequent improvement. Results showed a similar balance performance by the two groups during the baseline stage. However, the low-anxiety participants improved significantly during the test stage. Thus, in their encounter with a novel balance challenge, the two groups shared a comparable level of balance skills. However, the high trait anxiety participants did not seem to improve from repeated trials of the challenging balance exercise.

A related question concerned whether the challenging wobbly task triggered a higher state–fear response in the high trait anxiety participants versus the low trait anxiety participants. We reasoned that the participants may have perceived the balance task as posing certain physical risks. If the high trait anxiety participants experienced difficulty in negotiating the stability platform, then they may have experienced greater fear while waiting for the more risky task (when the red light was lit prior to the wobbly condition). In the present study, we assessed state–fear response by measuring ASR amplitude during the successive stages of the balance task. The two groups showed similar amplitudes of ASR responses during the habituation period. Group differences emerged during the baseline stage with higher ASRs in the high trait anxiety participants. During the subsequent test stage, the ASR amplitude decreased, particularly in the high-anxiety group, to the extent that the two groups showed a similar level of ASR responses. These findings suggest that the high trait anxiety participants responded with increased ASRs only to the initial encounters with the challenging balance situation.

The finding of potentiated ASRs in high trait anxiety participants cannot be attributed to group differences in the motor performance itself because ASRs were monitored exclusively when participants were in a stable position on the stabilometer. In addition, it is unlikely that the potentiated ASRs in the high trait anxiety group are outcomes of differential fear conditioning to wobbly conditions versus stable conditions on the stabilometer. Indeed, the ASRs recorded on the background of a red light CS+ signaling the approaching challenging wobbly condition did not differ significantly from ASRs recorded on the background of a green light CS− signaling a stable condition. A possible explanation is that the potentiated ASRs in high trait anxiety participants were caused by a rapid conditioning of fear response to the context of a wobbly condition rather than response to any specific stimulus.

This interpretation is in line with findings of a previous study that tested ASRs in a paradigm involving the anticipation of electric shocks by healthy volunteers (Grillon et al., 1993). Participants were grouped by the state–fear portion of the STAI (Spielberger et al., 1983). The amplitude of fear-potentiated ASRs, but not baseline ASRs, was greater in the high state anxiety group compared with the low state anxiety group. Thus, fear-potentiated ASRs can be considered a reliable index of state-related anxiety. According to this interpretation, the potentiated ASRs in the present study may reflect higher contextual fear in the
high trait anxiety participants versus the low trait anxiety participants. In other words, the high trait anxiety participants may have been prone to respond with high contextual fear when facing novel challenging balance situations.

By considering the involvement of contextual fear, researchers can foresee both negative and positive clinical effects in an encounter with a balance challenge on individuals with low-balance skills. The known negative consequence is that in the real world, the emergence of contextual fear may induce avoidance of situations that challenge balance skills. The positive consequence is that even brief training in balance skills, although only marginally effective in terms of the balance performance, may reduce contextual fear to almost normal levels.

Cumulatively, the present findings indicate that participants with high trait anxiety experienced elevated fear during the initial encounters with balance-challenging episodes and lingered in balance learning compared with participants with low trait anxiety. This suggests that the clinical entity designated as a comorbidity of balance and anxiety disorders has an analogous representation in the normal population in the form of reduced balance skills in high trait anxiety participants. Because of the small size of the present sample, further validation of the results obtained in our study is needed. However, the behavioral findings that we obtained aligned with our prediction; therefore, the small sample size might reflect the robustness of the effects.

However, it is important to note that the present study did not resolve the question of causality of the balance–anxiety comorbidity. On one hand, it is possible that high anxiety interferes with sensory-motor learning, and interference would explain the flat learning curve of the trait anxiety participants on the stabilometer. Such speculation is supported by the higher ASRs displayed by the high anxious group relative to the low anxious group in the initial baseline stage of learning. On the other hand, it is possible that a specific balance-learning dysfunction slows the learning of participants in the high trait anxiety group. On the basis of the theoretical predictions stated in Erez et al.'s (2004) study, we are currently testing whether the improvement in balance is associated with an amelioration of the anxiety symptoms. Verification of the causality of the balance–anxiety comorbidity will require (a) specific treatment of either the balance or the anxiety and (b) simultaneous monitoring of the consequences of the treatment on the other factor.

AUTHOR NOTES

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