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ORIGINAL CONTRIBUTION

Taking a closer look: autonomic dysregulation in socially anxious children

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Abstract Previous research on autonomic characteristics of social anxiety in children and adolescents has produced highly inconsistent results which may partially be due to task differences and a limited breadth of autonomic measurement. Here we investigated a sample of high (HSA) and low socially anxious (LSA) children, aged 10–12 years before, during and after a standardized evaluated speech task while acquiring a broad set of autonomic and experiential measures. During baseline, we found evidence for tonically higher sympathetic autonomic activity in HSA children, indicated by higher low frequency heart rate variability (LF) and a trend for higher LF to high frequency heart rate variability ratios (LF/HF). In response to the speech task, HSA children showed blunted cardiac responding evidenced by slower increase and delayed recovery of heart rate and a similar significant trend on LF/HF values. Self-reported anxiety, by contrast, showed enhanced reactivity from baseline to anticipation in the HSA compared to the LSA group. The results suggest a restricted cardiac flexibility in HSA children and illustrate that broad autonomic assessment during a well-structured, naturalistic task may improve our understanding of the autonomic physiology of socially anxious children. The results have implications for current theories of social anxiety.

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Introduction

Bodily symptoms of anxiety play a key role in cognitive maintenance models of social phobia (SP; [1]). These models assume that during social evaluative stress, individuals with SP focus on their bodily symptoms of anxiety because they worry that visibility of such physiological symptoms to others is interpreted as social failure (e.g. "If others see how much I blush, I will embarrass myself"). As a result of this focused attention, physiological symptoms are overestimated. Furthermore, this attentional focus on physiological arousal might aggravate social fears, because it limits the attentional resources available for adequate coping in social interactions [1]. While the Clark and Wells Model was initially conceptualized to explain the maintenance of clinical SP, research has indicated that similar processes are present in subclinical samples, i.e. individuals with high levels of social fears (HSA) but no clinical diagnosis (reviewed below). Interestingly, these subclinical–clinical HSA groups seem to show a qualitatively similar psychopathology as full blown SP but with less severity [2, 3], which has led researchers to speculate that various forms of social anxiety can be understood as different parts of a social anxiety continuum with similar qualities in psychopathology (see the continuum model of social anxiety; [4]).

Social anxiety during childhood and youth

To understand the cognitive and biological factors that contribute to development and/or aggravation of social

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fears, the study of HSA and SP children is crucial, since most cases of social anxiety first arise during childhood and become chronic through youth [5]. Further, besides its role as a risk factor for SP, HSA in this age group can have a dramatic negative impact on the social-academic life of the affected children [6]. Importantly, research on adults with social anxiety cannot be simply extrapolated to childhood and youth, due to their strong cognitive, emotional, and physiological development [7, 8].

However, supporting the assumptions of the Clark and Wells model, previous studies in HSA/SP children have replicated adult findings showing that they overestimate the strength of their own physiological symptoms (blushing, sweating or a racing heart) during social evaluative stress, and are over-concerned about the visibility of these symptoms to others [9–11]. Still, an obvious but currently open question is, whether true physiological dysfunctions are present in social anxiety during childhood or whether only their representation in the experiential system is altered. Thus, the question arises whether socially anxious children show objective alteration in the regulation of the autonomic nervous system (ANS), which is central in regulation of the organisms coping with stress and negative emotion [12]?

Previous research on autonomic function in social anxiety during childhood and youth

Several studies have investigated the autonomic responses to social stress in HSA or SP children and adolescents. However, results are highly divergent, showing either higher autonomic reactivity [13, 14], lower autonomic reactivity along with a tonic sympathetic hyperarousal [15, 16] or no significant differences in socially anxious groups [9, 10, 17, 18]. For example, Anderson and Hope [9] exposed a large sample of adolescents with SP, aged 13 to 17 years, and a healthy control (HC) group to two anxiety provoking tasks, while actual physiological arousal (heart rate [HR], blood pressure) and perceived arousal were measured. Results showed that SP adolescents perceived their bodily arousal as stronger but did not differ from HCs in actual physiological arousal. The authors conclude that a biased perception of physiological symptoms but not actual autonomic differences may characterize adolescents with SP.

Taking findings as this one as a starting point, we [15] used refined psychophysiological measurements comprising a broad array of sympathetic and parasympathetic activity measures in SP and healthy control (HC) children while these underwent the Trier Social Stress Test for Children (TSST-C; [19]). During baseline, SP children showed higher HR levels, lower levels of respiratory sinus arrhythmia (RSA or HF)—a measure for parasympathetic vagal control of the heart [20], and higher levels of sympathetic activity than HCs. Further, during reactivity to the TSST-C, SP children showed a decreased RSA reactivity to the TSST-C, and a delayed HR recovery from the stressor when compared to HC children. Drawing on the concept of autonomic flexibility [21], we explain these findings as revealing a restricted autonomic flexibility within socially anxious children. According to this concept, healthy autonomic functioning is characterized by the ability to respond quickly and flexibly to environmental demands—which is primarily maintained by the parasympathetic branch through the nervus vagus because its effect on heart rate change is especially swift. Anxiety disorders, as proposed by this approach, relate to a tonic autonomic hyperarousal during rest, which then results in a blunted autonomic reactivity and a delayed recovery (see also the neuro-visceral integration model by Thayer and Lane [21]).

Limitations of previous research

What factors may explain the substantial differences in empirical findings in the field? First, most studies with null results on autonomic characteristics of HSA and SP children have assessed only a limited number of autonomic parameters, possibly failing to fully capture all relevant autonomic changes. Heart rate (HR), for example, is frequently measured but this parameter is regulated jointly by both sympathetic (SNS) and parasympathetic (PNS) nervous system branches. Thus, HR alone and cannot speak to a dysregulation of one autonomic branch or the other. Moreover, the same HR level can be the result of several different combinations of PNS and SNS activity that would can go unnoticed if not decomposed [22]. Second, autonomic assessment in most previous studies was temporally relatively coarse (e.g. measured only during baseline and stress) and did not uniformly include anticipation and recovery phases [9, 17]. However, particularly during anticipatory processing socially anxious individuals may show autonomic differences from non-anxious controls as suggested by recent research on HSA adults [23]. In addition, a recent meta-analysis by Chida and Hamer [29] showed that elevated trait anxiety is often associated with a prolonged autonomic recovery from stressful events, which corresponds well with previous findings in SP children samples suggesting a slower autonomic recovery in clinical social anxiety [15, 16]. Hence, a more detailed temporal assessment including anticipatory and recovery phases seems to be promising to uncover autonomic disturbances in social anxiety.

The current study

Thus, the aim of our study was to investigate autonomic characteristics in children with high social anxiety, while taking some of the limitations of previous research into account. We assessed autonomic functioning in a sample of 10–12 years old children with either high (HSA) and low levels (LSA) of social anxiety during baseline, anticipation, an evaluated speech, and recovery. In our study a broad array of physiological parameters for sympathetic and parasympathetic activation was measured. To fully capture autonomic sympathetic activation, we separately measured sympathetic activity in different organ-specific domains innervated by the sympathetic nervous system (heart, blood vessels and skin) as suggested by previous research [24]. Further, in adults and children with elevated trait anxiety, it has been found that these samples are often characterized by a tonic sympathetic hyperarousal, relative to parasympathetic control (for an overview, see [25]). For the assessment of this sympatho-vagal balance, the calculation of physiological measures that relate sympathetic and parasympathetic activation to each other (e.g. LF/HF ratios; [26]) may be sensitive, thus we calculated indices for sympatho-vagal balance. Based on previous results, we expected HSA children to evidence: (1) a sympathetically modulated hyperarousal during baseline along disturbances in sympatho-vagal balance [15, 25, 27] and (2) a delayed autonomic recovery [15, 28, 29]. Due to the heterogeneity of previous research [9, 10, 15, 16], no specific predictions were made for reactivity to the stress task.

Method

Participants

A total of 108 families responded to advertisements in local newspapers and information flyers in schools offering €35 for participation in a study on shy and non-shy children. After having received detailed written information about the aim and procedure of the study, and the Social Anxiety Scale for Children revised (SASC-R; [30]), families mailed back the questionnaire and the informed consent form for participation. A total of 40 children were assigned to either the HSA or LSA group ($n = 20$ each). This was done according to previous research [31] based on the cut-off scores on the SASC-R suggested by La Greca and Stone [30]: HSA: $\geq$54 for males, $\geq$50 for females; LSA: $\leq$40 for males and $\leq$36 for females. These cut-off scores relate to social anxiety found in clinical and non-clinical samples. Exclusion criteria for all children included medical conditions that might affect the physiological systems investigated (e.g. cardiovascular disease, asthma) or the use of medication that could alter physiological responses (e.g. methylphenidate). Furthermore, to limit other psychopathological influences (e.g. ADHD), children who were reported by their parents to have received a lifetime diagnosis of a mental disorder (e.g., through a medical practitioner) were excluded. Participant characteristics can be found in Table 1. The current study was approved by the local ethics committee.

Table 1 Participant characteristics

	HSA ($n = 20$)	LSA ($n = 20$)	Statistics
Age	11.0 (0.76)	11.1 (0.83)	$t(38) = 0.39$, n.s.
Female (%)	60	45	$\chi^2(1) = 0.91$, n.s.
Elementary schoo (%)	15	15	$\chi^2(1) = 0.00$, n.s.
BMI-SDS	0.39 (1.03)	0.30 (1.04)	$t(38) = 0.22$, n.s.
SASC-R	61.4 (7.64)	30.6 (8.75)	$t(38) = 11.84$***
CDI	15.1 (8.92)	10.3 (12.46)	$t(38) = 0.69$, n.s.
CBCL	64.0 (8.62)	49.7 (8.60)	$t(38) = 5.26$***
Externalizing	56.1 (11.01)	52.6 (8.70)	$t(38) = 1.31$, n.s.
Internalizing	68.9 (9.47)	50.9 (7.64)	$t(38) = 6.62$***

CBCL Child behavior checklist (t values), *CDI* child depression inventory, *HSA* high social anxiety group, *LSA* low social anxiety group, *BMI-SDS* body mass index-standard deviation scores, *SASC-R* Social Anxiety Scale for children-revised

*** $p < 0.01$

Psychometric and experimental measures

The Social Anxiety Scale for children-revised (SASC-R; [30])

The SASC-R is a self-report measure which assesses trait social anxiety symptoms in children with total scores ranging from 18 to 90. The SASC-R has satisfactory test–retest reliability and internal consistency [32]. Children respond to each item using a 5-point Likert-type scale ranging from 1 (not at all) to 5 (all the time). The scale is moderately correlated with general measures of anxiety, self-perceptions of social confidence, teacher ratings of anxiety withdrawal, and peer nominations of popularity [33]. Internal consistency in our sample was excellent ($\alpha = 0.928$).

The Child Depression Inventory (CDI; [34])

The CDI assesses the cognitive, affective, and behavioral symptoms of depression in childhood. Total scores range from 0 to 52 with a recommended cut-off score at 19. Internal consistency and test–retest reliability estimates are acceptable and the CDI shows good discriminant and

convergent validity [35]. In the current sample, the CDI showed good internal consistency ($\alpha = 0.800$).

The Child Behavior Checklist (CBCL; [36])

The CBCL is a parent-report measure for emotional and behavioral problems in children and adolescents. It includes various DSM-oriented syndromes and competence scales grouped into internalizing or externalizing scales. The CBCL has shown good levels of internal consistency, test–retest reliability and acceptable convergent validity (e.g. [36]). Since we did not include a structured diagnostic interview for the assessment of mental disorders in children, the CBCL was used to measure levels of psychopathology within the sample. Both internalizing and externalizing scales showed excellent internal consistency ($\alpha = 0.922$, and $\alpha = 0.888$, respectively).

Psychophysiological measures

All psychophysiological channels were simultaneously recorded at 400 Hz using the Varioport-II system (Becker Meditec, Karlsruhe, Germany). Data analysis was performed using ANSLAB software [37]. Since measured sympathetic autonomic activation can differ depending on the organ in which it is assessed (e.g. heart or blood vessels, see also [24]), we broadly assessed sympathetic arousal in three different domains (cardiac, vascular, and electrodermal sympathetic activation). Hence, in addition to HR (in beats per minute [bpm]), five parameters for vascular and electrodermal sympathetic activity were calculated, which were integrated into separate indices for sympathetic activity, to increase reliability [38]: Vascular sympathetic index (VSI; $\alpha = 0.859$): pulse wave transit time, and pulse wave amplitude; electrodermal sympathetic index (ESI, $\alpha = 0.913$): skin conductance level, number of non-specific skin conductance fluctuations, and amplitude of non-specific skin conductance fluctuations. To further calculate a measure for cardiac sympathetic activation, which may capture specifically sympathetic influences on HR, spectral density of R–R interval fluctuations was summed over the low frequency band (LF 0.04–0.10 Hz), which is assumed to reflect primarily cardiac sympathetic influences [39, 40].

We examined the individual respiratory rates to see if they lay outside the 9–30 cycles/min range (0.15–0.5 Hz). As this was never the case, RSA was quantified by the natural logarithm of the summed Welch power spectral density of the inter-beat-interval (measured in milliseconds between two consecutive R-waves in the ECG) in this range, corresponding to the high-frequency (HF) range of heart rate variability for children in the studied age range [41]. LF/HF ratios were calculated as a measure of cardiac sympatho-vagal balance [26]. Since RSA can be influenced by respiration, independent of vagal control [20], respiration rate and tidal volume were measured using two linear transducer respiration belts around the thorax and the abdomen of the participating children, which were then compared between the groups using ANOVAs (see statistical analyses). Further, we calculated the transfer function between respiratory channels and RSA/HF [42]. Respiratory channels were calibrated against a fixed volume of 350 ml.

Procedure

After arriving at the laboratory, children were given 30 min to habituate to the laboratory setting during which they filled out several questionnaires and played a card game. Children were then guided to the sound-attenuated, temperature-controlled experimental room, seated in a comfortable armchair, and equipped with electrodes. During a 5-min baseline phase, children watched a relaxing video clip, which included non-stimulating nature scenes [11, 43]. Prior to the following recounting task, children listened to a 9-min short story (anticipation phase). Before the beginning of the anticipation phase, all children were instructed that they would have to recount the story for 3 min in front of a panel of two unknown adults (task period). To further increase the social-evaluative character of the task, children were told that other children their age would rate their performance from a video tape recorded during the task [44]. Members of the panel were trained to show a standardized and distanced non-verbal behavior during the task. To equate groups on speaking duration, the panel gave a standardized verbal reminder, if children finished early with their recounting (given after 10 s without speaking: "There is still time left, please continue with your story" c.f. [16]).[1] After the completion of the task, a 5 min recovery phase followed during which the children again watched a relaxing video clip. Self-reported anxiety ratings (0 no anxiety, 10 extreme anxiety) were obtained at four times during the task (after baseline, anticipation, speech, and recovery).

Data reduction and statistical analyses

To protect against spurious findings and alpha-inflation, a multivariate analysis of variance (MANOVA) including all seven primary measures (anxiety, HR, ESI, VSI, LF, HF,

[1] There was no difference between the groups in the amount of received reminders by the panel ($p > .465$).

LF/HF) preceded univariate analyses for individual measures. This MANOVA (and follow-up univariate ANOVAs) included the factors Group (LSA, HSA) and Phase (baseline, anticipation, task, and recovery) to determine global group differences on the primary measures. Effects involving Group were then decomposed by measure and by phase in univariate, repeated measures ANOVAs for the individual measures. Further, we statistically compared the two groups on respiratory parameters to assess respiratory influences on RSA.

To differentiate between tonic state and phasic reactivity, baseline scores for each of the primary measures was subtracted from the following experimental phases [15]. Independent sample t tests compared groups on baseline scores. In case of significant global ANOVA effects involving group, we calculated difference scores of successive measurement points, which were then compared between the two groups, using independent sample t tests. There was robust reactivity on all measures to the task ($ps < 0.001$), thus we report significant effects of Phase only when they interacted with Group. Partial eta square (η^2) is reported for significant ANOVA/MANOVA findings, Cohen's d for significant t tests. A power analysis showed sufficient statistical power (calculated as $1 - \beta$ probability = 0.77–0.99) for medium to large main effects and interactions, as reported by previous studies in the field [14–16].

Results

Participant characteristics and psychometrics

As shown in Table 1, groups where successfully matched on age, gender, education, and BMI—standard deviation scores calculated according to national norms (measured by meter and scale). Children of the HSA group showed significantly higher levels on the SASC-R and the internalizing subscale of the CBCL. Groups did not differ either on CDI scores, or on the CBCL externalizing subscale.

General analysis of primary measures

Figure 1 shows the baseline and difference scores for all primary measures, which approximately fitted the normal distribution ($ps > 0.231$). The MANOVA on the combined primary measures revealed a significant Group (HSA, LSA) × Phase (baseline, anticipation, task, recovery) interaction, $F(3,18) = 3.95$, $p = 0.029$, $\eta^2 = 0.094$, cubic contrast, $p = 0.008$, but no main effect of Group, $F < 1.00$. Hence, this global Group × Phase interaction was decomposed by follow-up ANOVAs.

Baseline

During baseline period, the HSA group showed borderline significant higher LF levels, $t(38) = 1.96$, $p = 0.057$, $d = 0.74$, and higher LF/HF ratios, $t(38) = 2.49$, $p = 0.018$, $d = 0.84$. There were no significant group differences on any other measures during baseline, $ts < 1.32$, $ps > 0.197$.

Reactivity and recovery

Self-reported anxiety

The 2 (Group) × 4 (Phase: baseline, anticipation, task, recovery) ANOVA showed a significant main effect of Group, $F(1,38) = 9.94$, $p = .003$, $\eta^2 = 0.207$, and a Group × Phase interaction, $F(3,76) = 3.98$, $p = 0.018$, $\eta^2 = .095$, cubic Group × Phase contrast ($p = 0.014$). Post hoc paired t tests revealed that HSA children had a higher increase from baseline to anticipation compared to LSA children, $t(38) = 2.75$, $p = 0.010$, $d = 0.88$.

HR

For HR, the ANOVA revealed a significant Group × Phase (baseline, anticipation, task, recovery) interaction, $F(3,114) = 3.81$, $p = 0.033$, $\eta^2 = 0.091$, cubic group × phase contrast ($p = 0.008$), but no main effect of group, $F < 1.00$. Post hoc t tests revealed that HSA children had a lower reactivity from anticipation to the stress task, $t(38) = 2.56$, $p = 0.014$, $d = 0.81$, and a slower recovery than LSA children, $t(38) = 2.28$, $p = 0.028$, $d = 0.73$.[2]

Sympathetic and parasympathetic activation

On the cardiac sympathetic measure (LF scores), analyses showed a main effect of group, $F(1,38) = 5.67$, $p = 0.022$, $\eta^2 = 0.130$, indicating a lower cardiac sympathetic arousal in HSA children relative to baseline values, but no other main effects of group or group × phase interactions on the sympathetic measures (ESI, VSI, LF scores), $ps > 0.164$. Further, no significant main effects of group or group × phase interactions emerged on parasympathetic activity (HF), $Fs < 1.88$, $ps > 0.135$.

[2] Exploratory analyses revealed that HR reactivity (anticipation to task) and recovery (task to recovery) were significantly negatively related to SASC scores in the sample ($r = -0.394$, $p = 0.012$; $r = -0.411$, $p = 0.008$), but neither to CBCL internalizing/externalizing scales nor to CDI scores.

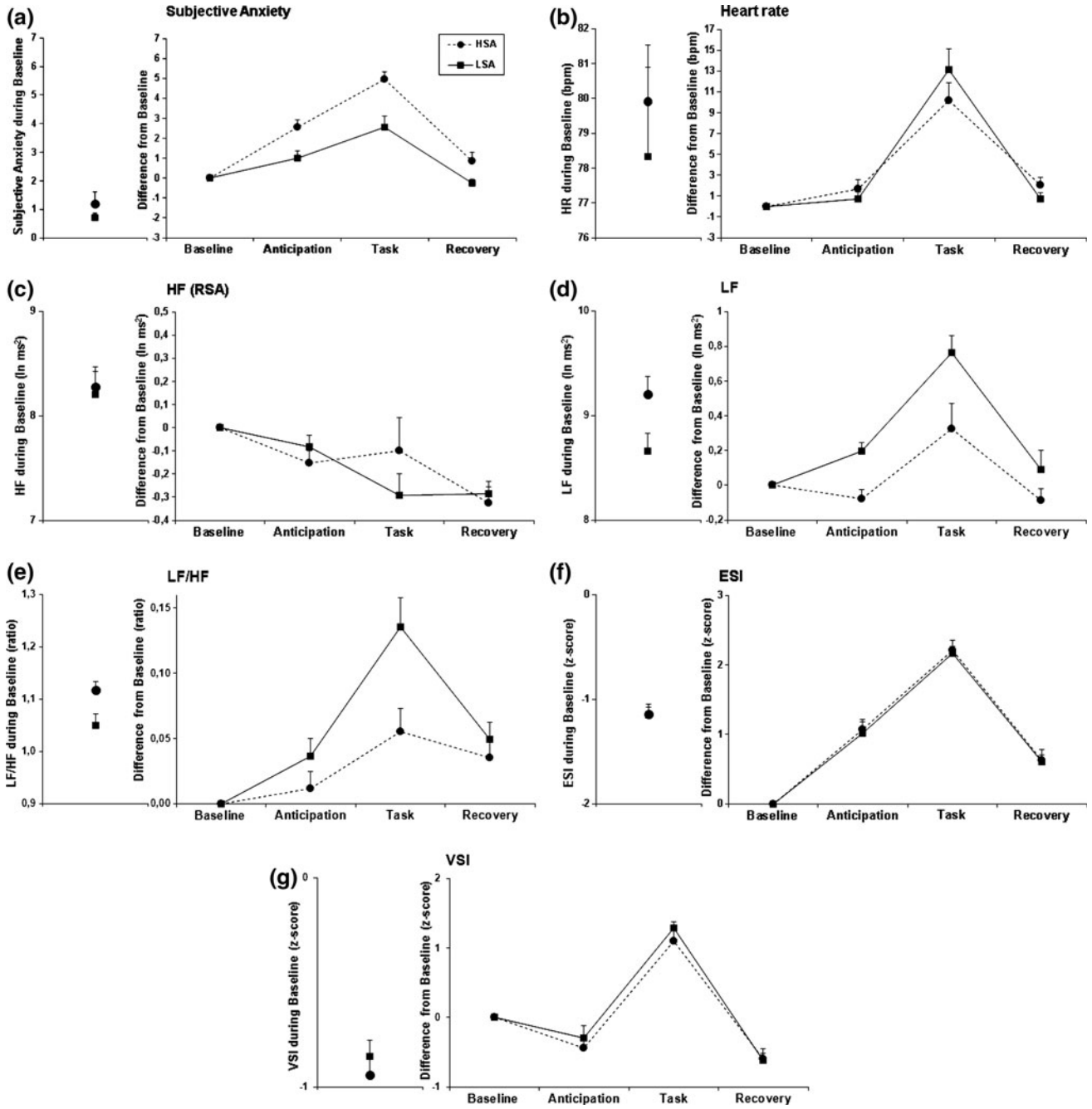

Fig. 1 Baseline and difference scores on all measures. *Error bars* are SEMS

Respiratory influences on RSA/HF

There were no effects of group or group × phase interactions during reactivity and recovery on respiratory parameters, $ps > 0.090$. In addition, no significant effects involving group emerged for the transfer function between RSA and respiratory channels $ps < 0.134$.

Cardiac sympatho-vagal balance

For LF/HF ratios, the ANOVA revealed no main effect of group, $F(1,38) = 2.89$, $p = 0.972$, but a significant group × phase interaction, $F(3,114) = 3.83$, $p = 0.023$, $\eta^2 = 0.091$, quadratic contrast ($p = 0.027$). Post hoc t tests on difference scores revealed that HSA children had a borderline significant lower increase in LF/HF ratios from

anticipation to the stress task, $t(38) = 2.00$, $p = 0.052$, $d = 0.64$, and a borderline significant slowed recovery, $t(38) = 1.96$, $p = 0.057$, $d = 0.62$.

Discussion

Research on autonomic functioning in childhood social anxiety has produced highly divergent findings [9, 10, 15, 17], which may be due to differences in experimental designs and autonomic assessment. While addressing some of the limitations of previous research (narrow assessment of the ANS and relatively coarse temporal assessment), we here investigated autonomic and experiential responding prior, during and after a social stress task in a sample of high (HSA) and low socially anxious (LSA) children. Relative to LSA children, we expected the HSA group to evidence: (1) a sympathetically modulated hyperarousal during baseline along with disturbances in sympatho-vagal balance, and (2) a delayed autonomic recovery. Due to the heterogeneity of previous research [9, 10, 15, 16], no specific predictions were made for reactivity to the stress task.

Beside a higher increase in subjective anxiety from baseline to anticipation in anxious children, the HSA group showed a trend toward an imbalance between sympathetic and parasympathetic autonomic branches at baseline, partially confirming our first hypothesis: borderline significant higher baseline levels of LF and significantly higher LF/HF ratios suggested higher cardiac sympathetic dominance in relation to parasympathetic control [25, 26, 45]. In line with hypothesis two, HSA children showed a blunted HR reactivity and recovery from the speech task, which was accompanied by a trend toward a weaker increase of LF/HF ratios in HSA children to the stress task, and a trend toward a blunted LF/HF decrease during recovery.

Blunted cardiac responding in childhood social anxiety?

The finding in HSA children of a blunted HR reactivity and recovery fits with several previous findings in adults and children with various anxiety disorders. For example in a larger meta-analysis by Chida and Hamer [29] on 761 studies, the authors found that high-trait anxiety in adults was related to a blunted HR reactivity and recovery when facing experimental stressors, which was also stable when only studies with high methodological standards were included. Recently, a blunted HR reactivity was also found in a sample of adult SP patients when compared to HC controls [46]. Despite this strong evidence, so far this pattern has rarely been described in children with social anxiety. Interestingly, while several other studies on autonomic characteristics in childhood social anxiety failed to find differences in HR reactivity to social stress [9, 10, 14, 18], our analyses revealed a decreased HR rise from anticipation to stress in our HSA children. Notably, these previous studies did not include separate anticipatory and recovery phases and might thus have missed this difference. Supporting this idea, exploratory analyses of our own data showed no differences in HR reactivity when simple baseline-stress differences were compared between the groups. Put differently, our detailed temporal assessment revealed substantial anticipatory responding in anxious individuals, which represents the starting point for an additional increase during actual stress. A different explanation for the blunted autonomic responding in our HSA children could be that the anticipation phase was necessary to start to build up physiological activation and the stress task further added to it [23]. Thus, the length of the stressor in our study may effectively have been longer when compared to other studies. Future research should strive to disentangle anticipatory from stress-induced responses, e.g. by presenting anticipated and unanticipated stress tasks.

Restricted autonomic flexibility and motivational theories

On a more general level, a blunted HR reactivity and recovery correspond well with theoretical models proposing that anxiety disorders and elevated trait anxiety are associated with a restricted autonomic flexibility [21, 25]. According to this approach, anxious individuals show a tonic hyperarousal during rest which then leads to a blunted and inflexible reactivity during coping with environmental demands. Further, the ANS is assumed to be less effective in the down regulation of physiological arousal in the aftermath of stressful events, resulting in a prolonged autonomic recovery/arousal [15, 28]. One has to note that while these concepts emphasize the central role of the parasympathetic branch for a restricted autonomic flexibility, our finding of a statistical trend toward a blunted LF/HF reactivity and recovery could suggest that a lower HR flexibility in our HSA children was driven by both branches of the ANS.

A different approach beside the autonomic flexibility model to explain a blunted HR reactivity in anxious individuals relates to motivational theories [47]. According to these theories, task difficulty and success importance are the two major determinants of energy investment in instrumental behavior. Consequently, organismic energy expenditure should be proportional to a behavior's difficulty as long as success is possible and justified. If a behavior is either too difficult or the necessary amount of energy for its execution exceeds the justified level, energy

investment is low, guided by the resource conservation principle [48]. Supporting these assumptions, Richter et al. [49] report that individuals' cardiac reactivity (blood pressure, pre-ejection period) to a mental stress task increased with increasing task difficulty but decreased when the task became impossible. For autonomic reactivity in childhood social anxiety, it is conceivable that cardiac reactivity also depends on how children appraise their chances of success in a social performance task. Possibly, HSA children in our study judged their the chance of success in the upcoming social performance task as minimal due to high task difficulty (performance task in front of two unknown adults, videotaping of their performance, evaluation by other unknown children, monitoring of the physiological responses) and strong negative self-beliefs [50]. Differences in social tasks and in perceived task difficulty could also explain divergent findings in different studies on autonomic reactivity childhood social anxiety. Hence, future studies may want to manipulate social task difficulty and measure expected performance in socially anxious children, to evaluate if motivational theories can help to explain a blunted autonomic reactivity in childhood social anxiety.

Social anxiety and social phobia: on one continuum or qualitatively different?

Since we did not assess the presence of mental disorders in diagnostic interviews in our sample, we cannot rule out the possibility that some of our HSA children may have fulfilled the criteria of SP or a different mental disorder. This limitation has to be kept in mind when comparing the present results with those obtained in children/youth with full-blown SP in our previous studies: baseline HR, for example, was elevated in previous studies on SP children relative to controls [15, 16] but not in our present sample HSA children (under otherwise comparable conditions regarding demographics, tasks, measurements). Further, in SP children, we recently found a lower blunted RSA reactivity to social stress, while in the current study, no such effect was found. Differences in severity of social anxiety between the samples may explain this: although children in the current study were selected for self-reported social anxiety considered to be clinically relevant [30], it is possible that social anxiety in our sample relates to a broader range of both clinical and subclinical social anxiety (see also the continuum model of social anxiety; [4]). This is possible when samples are selected based solely on questionnaire measures [51]. Hence, full-blown SP in children may have a more deviant parasympathetic response profile suggesting that activity of this branch of the nervous system may change as social fears become more severe. In addition, the pattern of elevated baseline HR in SP was absent in HSA. This leads us to speculate that anticipatory stress increases as socially anxious children progress into SP. Ultimately, longitudinal research would be needed to confirm such hypotheses.

Implications for cognitive models of social anxiety, limitations and future research

Cognitive models of SP propose that socially anxious individuals overestimate the strength of their bodily arousal during social situations, which is thought to play a key role in the maintenance of social fears [1]. However, beside such a perception bias, these models also propose that socially anxious individuals may also show an additive autonomic hyperreactivity to social stress, which can lead to increased blushing, a racing heart, or sweating [1, p. 74]. While previous studies have quite consistently found a perception bias for bodily arousal in children and adolescents with social anxiety already [9, 11], there is less evidence for such an autonomic hyperreactivity in these samples. Still, there is growing evidence for a tonic hyperarousal in socially anxious children, including our own results [15, 16], and it may be conceivable that during social evaluative stress, socially anxious children become more aware of this chronic arousal, as a result of a shift in attentional deployment [1], which then leads to a biased perception and interpretation of bodily anxiety symptoms.

One important limitation of the current study is the small sample size and the moderate statistical power. Related to this, some of the effects on autonomic parameters (e.g. LF/HF ratios) were significant only on a trend level. Hence, our results need replication in larger samples and future studies should control for other important confounds such as puberty status or cognitive ability to evaluate the reliability of our results. Further, one might ask how specific our findings are for social anxiety and to what degree they also apply to samples of children with other anxiety problems. Answering this question is difficult because social stress is considered a specific anxiety provocation for socially anxious children and not for children with specific phobias or separation anxiety disorder. An interesting future approach in the field of childhood anxiety may be the use of mental imagery techniques which can help to induce anxiety specific to one but not the other disorder with comparable intensity and duration. In adults, such methods have produced interesting results, showing differences in autonomic profiles between subgroups of anxiety [52]. In addition, sympathetic differences emerged only on cardiac sympathetic parameters, but not on measures for electrodermal or vascular sympathetic activity (ESI and VSI index). While LF is frequently used

as an indicator for sympathetic activity [25, 26, 40, 45], the validity of LF scores has been questioned by some researchers [53]. Nonetheless, our results of a blunted LF reactivity in our HSA children fits well with the blunted HR reactivity in the same group, which may support this measure as an index of cardiac sympathetic activation. Still, future studies need to replicate our results to evaluate their reliability in larger samples of HSA children, using additional and well-validated measures for cardiac sympathetic activity such as pre-ejection period. Further, we used a laboratory social performance situation in front of adults but it remains uncertain if our results also hold for social interactions with same aged peers—one of the most feared situations in childhood social anxiety [6]. We tried to ameliorate this by instructing subjects that their video tapes would be examined by same age peers. In addition, previous research has shown that physiological abnormalities, which emerge in laboratory studies, do not necessarily hold during ambulatory assessment [54]. Consequently, an important future direction would be to investigate sympathetic and parasympathetic changes in children with social anxiety during their everyday life, including during social threat situations.

Beside these limitations, we believe that our results are important since they show that social anxiety in children is associated with subtle disturbances in the ANS that become evident when jointly considering SNS and PNS and by closely tracking anticipation, recovery, and stress reactivity. Without such measures and task features, null findings might be obtained, giving the impression that social anxiety is simply a cognitive phenomenon.

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

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