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ESTABLISHING THE VALIDITY OF A NOVEL PASSIVE STRESS TASK

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Abstract

Laboratory tasks used to elicit a cardiovascular stress response in the laboratory can involve either active or passive coping. However, in previous work, passive stress tasks often incorporate a distinct physical stress element, such as the handgrip or cold pressor task, meaning observed changes in cardiovascular parameters may be the result of the physical element of the stressor rather than truly reflecting psychological stress. The present study aimed to establish the validity of a psychological passive stressor; one more analogous to active tasks than those previously employed in laboratory studies. Twenty-six young, healthy adults completed a speech task in the laboratory following a resting baseline period. Twelve months later, they were invited back to the laboratory and watched the video recording of their speech. Analyses confirmed that while both tasks elicited significant SBP and DBP change (all *p*s < .001), only the active task was associated with HR and CO reactivity (both *p*s < .001), while only the passive task was associated with TPR reactivity ($p = .028$). Furthermore, the passive stressor was associated with a mixed hemodynamic profile, whereas the active stressor was associated with a clear myocardial profile. This study confirms that watching a video recording of oneself complete a speech task is associated with a more vascular response profile, a response associated with passive coping contexts.

1. **Introduction**

Active and passive coping contexts are characterized by the opportunity afforded to individuals to influence performance or the outcomes of a situation (e.g., Obrist, 1981; Schneiderman & McCabe, 1989). Active stress tasks, such as speech or mental arithmetic tasks, require active engagement and participants can alter their performance on the task. In contrast, passive tasks require that participants simply endure the task; they cannot change the results of the task (Sherwood, Dolan, & Light, 1990). Passive tasks can further be considered as physical or psychological stressors. For example, within the cardiovascular reactivity literature a common passive stressor is the cold pressor task (e.g., Brindle, Whittaker, Bibbey, Carroll, & Ginty, 2017; Tuomisto, Majahalme, Kahonen, Fredrikson, & Turjanmaa, 2005; Vella & Friedman, 2007). However, even though employed as a psychological stressor, this task involves physical changes, and might better be regarded as a physical stressor. Psychological passive stress tasks usually involve viewing distressing film clips or images. For example, asking participants to view negative images (e.g., Kim & Hamann, 2012), humorous film-clips (e.g., Tuomisto et al., 2005), surgical film-clips (e.g., Gross, 1998a; Zakowski, Cohen, Hall, Wollman, & Baum, 1994), or aversive film-clips (e.g., Dunn, Billotti, Murphy, & Dalgleish, 2009; Hermans et al., 2011). Other studies employ such measures, but attempt to increase the relevance of the situation by asking participants to immerse themselves within the image or film-clip by adopting an eye-witness perspective or imaging themselves as part of the situation (e.g., Kinner, Het, & Wolf, 2014; van Marle, Hermans, Qin, & Fernandez, 2010).

It is posited that active and passive tasks elicit different patterns of cardiovascular responding due to preferential activation of beta- and alpha-adrenergic receptors, respectively (see; Schneiderman & McCabe, 1989). Active tasks are characterized by greater beta-adrenergic activation, indexed by increased blood pressure, heart rate (HR) and cardiac

output (CO). In contrast, passive tasks appear to elicit less beta-receptor activation compared to active tasks, and more alpha-adrenergic activity, resulting in greater vascular tone (indexed by increases in total peripheral resistance [TPR]). As a result, blood pressure increases to passive tasks are accompanied by lower CO and elevated TPR (Bolli, Amann, Hulthen, Kiowski, & Buhler, 1981; Hurwitz et al., 1993; Obrist, 1981; Obrist, Light, McCubbin, Hutcheson, & Hoffer, 1979; Saab et al., 1993; Sherwood, Allen, Obrist, & Langer, 1986). These patterns of responding are indicative of stimulus-response specificity and have been termed as a challenge-oriented response (active coping) or a threat-oriented response (passive coping), respectively (for a review see; Schneiderman & McCabe, 1989). Indeed, a myriad of research has demonstrated that active tasks have greater effects on blood pressure, CO and HR, while passive tasks have a greater influence on TPR (e.g., Winzer et al., 1999). Speech tasks have been found to elicit greater systolic blood pressure (SBP) and diastolic blood pressure (DBP) reactivity relative to the cold pressor task and viewing film-clips depicting stressful life events (Nyklicek, Bosch, & Amerongen, 2005). Similarly, mental arithmetic tasks elicit greater SBP, DBP, and HR reactivity compared to watching surgical film-clips (Patterson et al., 1994) and relative to the cold pressor task (Isowa, Ohira, & Murashima, 2004). In particular, the cold pressor task has highlighted the prominent role of vascular responding in driving blood pressure responses during passive tasks; characterized by little-to-no change in CO and heightened TPR (Saab et al., 1992; Saab et al., 1993). However, this is confounded by the vascular changes elicited by submerging one's hand in ice-cold water.

More recently, research has extended these findings and examined the underlying hemodynamic profile elicited by active and passive stressors, through the application of the Hemodynamic Profile-Compensation Deficit (HP-CD) model (Gregg, Matyas, & James, 2002). This orthogonal model computes continuous measures for HP and CD, accounting for the reciprocal relationship between CO and TPR. HP refers to the way in which CO and TPR

compensate (whether CO predominates, TPR predominates or both respond equally) and CD refers to the degree to which they compensate (an index of blood pressure change; an increase, decrease, or no change). By computing continuous measures for HP and CD, this model extends research which previously categorised people into either vascular or myocardial responders and excluded individuals who did not fit these groups. The validity of this model has been confirmed by a number of studies (e.g., Howard, Hughes, & James, 2011; Hughes, Howard, James, & Higgins, 2011; James & Gregg, 2004; James, Gregg, Matyas, Hughes, & Howard, 2012; O'Leary, Howard, Hughes, & James, 2013; Ottaviani, Shapiro, Goldstein, James, & Weiss, 2006; Ottaviani, Shapiro, Goldstein, & Mills, 2007).

Using this model, active tasks have been found to elicit both a myocardial response and a mixed hemodynamic response (blood pressure driven by synergistic increases in TPR and CO). For example, mental arithmetic tasks have been reported to elicit a myocardial response profile (Gregg et al., 2002). In contrast, physical passive tasks such as the handgrip stressor (Ottaviani et al., 2006) and the cold pressor task (Gregg et al., 2002) elicit a vascular response pattern.

A number of weakness exist with the types of passive stress tasks employed by previous studies. For example, due to the nature of some physical passive stressors, such as the cold pressor task, it is likely that the vascular stress response observed is due to vasoconstriction associated with immersing a body part in cold water; rather than a psychological stress response. Exposure of skin to cold temperatures has been shown to trigger a thermoregulatory reflex leading to increased TPR (Epstein, Stampfer, Beiser, Goldstein, & Braunwald, 1969). Arguably, the observed changes in TPR responding may be a result of physical, rather than psychological stress-related mechanisms.

Although passive viewing of film-clips and imagery has been associated with changes in affect (Gross, 1998b; Hermans et al., 2011; Lang, Bradley, & Cuthbert, 2008; Shiota &

Levenson, 2009; van Marle et al., 2010), such tasks may not create a coping context representative of real-life stressful situations. Film-clips lack dimensions inherent in day-to-day emotion-eliciting situations, such as motivational, social and evaluative aspects (Aldao, 2013), thus limiting our ability to draw accurate conclusions about the cardiovascular response patterns elicited during passive stress. Furthermore, reported changes in affect may be a result of response bias. For example, if participants viewed a sad film-clip and subsequently were asked to rate the degree they feel "sad" participants may report the emotions they expect the experimenter wants them to feel, rather than reporting the actual intensity of emotions felt.

To overcome such limitations, a handful of studies have designed and implemented more novel psychological passive tasks. Typically, the level of engagement, or control, participants have over the task is manipulated by employing an active version of the stress task followed by a passive version of the task. For example, Sherwood et al. (1990) asked participants to complete a reaction time task in pairs; quicker reaction times resulted in a greater monetary bonus. Each participant took part in both the active phase (making the reaction time response), and the passive phase (watching a team-mate make the response). Similar to previous research, the active phase of this task elicited greater SBP, DBP, HR, and CO reactivity in comparison to the passive phase. As expected, the active task was associated with mean decreases in TPR, while the passive task was associated with a mean increase in TPR.

Another methodology involves video-recording participants during an active task, such as singing song lyrics or a speech task, and later asking participants to view this video-recording (Harris, 2001; Hartley, Ginsburg, & Heffner, 1999; Schwerdtfeger & Rosenkaimer, 2011; Soye & O'Súilleabháin, 2019). A notable limitation with studies employing this paradigm is that the active and passive tasks are not directly compared (e.g., Harris, 2001;

Hartley et al., 1999; Schwerdtfeger & Rosenkaimer, 2011). For example, Schwerdtfeger and Rosenkaimer (2011) reported that both the act of completing, and viewing, the speech task elicited increases in SBP, DBP, and HR; however, this study did not compare the magnitude of reactivity elicited by each task. While Soye and O'Súilleabháin (2019), compared SBP, DBP, and HR responses to the speech and video elements of the paradigm (finding the active phase elicited greater SBP, DBP, and HR reactivity), this study did not report CO or TPR responses nor did it apply the HP-CD computational model to examine the underlying hemodynamic profile of responding. Hartley et al. (1999) suggested anticipated evaluation may influence cardiovascular responding during the video-viewing paradigm; participants who were informed the video-clip was going to be evaluated afterwards exhibited greater DBP and TPR responding, while viewing the video, compared to participants informed the clip would be deleted.

The potential confound of evaluation is also inherent in the studies employing the more typical active speech task/passive video-viewing task paradigm. Within these studies participants watched the video-clip with the experimenter observing (Schwerdtfeger & Rosenkaimer, 2011; Soye & O'Súilleabháin, 2019), or with the experimenter and confederates present (Harris, 2001). Therefore, it is unclear if the reported findings are a result of social-evaluation apprehension, or due to viewing the clip of the speech task.

Although this novel psychological passive stressor attempts to address issues inherent in other passive tasks, it also has limitations. First, most research employing this paradigm does not compare the profile of physiological responding elicited by the active and passive versions of these tasks. Second, the focus of these studies is on blood pressure responses (SBP and DBP), rather than the parameters which underlie blood pressure responding (CO and TPR). Third, the cardiovascular response to the video-viewing task may be confounded by the presence of the experimenter. If this passive task is found to elicit increases in blood

pressure driven by more vascular mechanisms this would indicate this task elicits a passive coping context. Furthermore, this task is not compounded by physical changes such as those elicited by the cold pressor task, and is more socially-relevant than simply viewing negative imagery, thus offering researchers a more ecologically valid method to study the effects of passive coping on the cardiovascular system.

The current study therefore sought to address limitations with previous passive stressors and test the validity of the passive stress task reported upon by Soye and O'Súilleabháin (2019); a passive task more analogous to active tasks than those previously used in the laboratory. This research examines the effects of two laboratory stressors on cardiovascular responding; completing a speech task and viewing the video-recording of the speech task, but *without* the experimenter also watching it. Participants first completed a speech task, then a year later were invited back into laboratory to complete the passive stress task; employing a within-subjects design. Previous research has demonstrated the stability of CVR to stress across testing sessions separated by weeks, months (S. Cohen et al., 2000; Ginty, Gianaros, Derbyshire, Phillips, & Carroll, 2013; Kamarck, Jennings, Stewart, & Eddy, 1993) and years (Hassellund, Flaa, Sandvik, Kjeldsen, & Rostrup, 2010; Matthews, Woodall, & Stoney, 1990; Sherwood et al., 1997). It is hypothesized that each task will elicit a distinctive cardiovascular response. Both tasks are expected to elicit increases in blood pressure; however, it is hypothesized that this increase will be greater in response to the active task. The active task is expected to elicit greater HR and CO reactivity, while the passive task will elicit greater TPR reactivity, indicative of a vascular response. The HP-CD model will be applied to examine the underlying hemodynamic response to each task. It is anticipated that the active task will elicit a myocardial or mixed response profile, while the passive task will elicit a vascular response profile. This report describes the design of this passive stress task and seeks to test the validity of the task in terms of (a) the task eliciting a

physiological stress response, and (b) the task eliciting a pattern of autonomic activity distinct from the active stressor.

2. Method

2.1. Design

The current study employed a within-subject design. The within-subjects variables were phase (baseline, task) and task (active task, passive task). The dependent variables were self-reported affect and cardiovascular parameters; SBP, DBP, HR, CO, and TPR.

2.2. Participants

A total of 26 participants completed both laboratory procedures. One participant had resting blood pressure that could be classified as hypertensive, (SBP/DBP > 140/90 mmHg), according to the guidelines for hypertension provided by the British Society of Hypertension and the European Society of Hypertension, so was excluded from all analyses. The present results report on a final sample of 25 participants (17 females, 8 males), aged 18-25 years (M = 20.40, SD = 1.66). Participants were non-smokers, reported good health, no history of cardiovascular disease and were not taking medication known to affect blood pressure. Ethical approval was obtained from the institutional research ethics committee. All participation was voluntary, participants signed an informed consent form and could withdraw from the study at any time. Participants were required to be between 18-25 years of age, non-smokers, and prior to participation refrain from consuming caffeinated products for six hours and alcohol for 12 hours. Such restrictions were employed due to the influence of caffeine (e.g., Hartley et al., 2000; James & Richardson, 1991; Savoca et al., 2005), smoking (e.g., Cruickshank, Neil-Dwyer, Dorrance, Hayes, & Patel, 1989; Ginty et al., 2014; James & Richardson, 1991; Saladini et al., 2016) and alcohol (e.g., McFadden, Brensinger, Berlin, & Townsend, 2005; Potter, Watson, Skan, & Beevers, 1986) on blood pressure.

A priori power analyses were conducted using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) to establish sample size requirements to detect large and medium effects for each statistical analysis, using an alpha of .05, and power of .80. For the 2 × 2 repeated measures ANOVAs a sample size of 12 was needed to detect a large effect (partial $\eta^2 = .64$) and a sample of 22 was needed to detect a medium effect (partial $\eta^2 = .25$). For the repeated measures *t*-tests, and the one-samples *t*-tests, in order to detect a large effect ($d = .80$) a sample size of 12 was needed, and to detect a medium effect ($d = .50$) a sample of 27 was required. As noted, 25 participants completed both visits, meaning the sample had sufficient power to detect medium to large effects.

2.3. Materials and Apparatus

2.3.1. Cardiovascular Assessment

Cardiovascular parameters (SBP, DBP, HR, CO, and TPR) were measured non-invasively using the Finometer PRO (Finapres Medical Systems BV, BT Arnhem, The Netherlands). The Finometer provides beat-to-beat measures of blood pressure and hemodynamic monitoring, based on the volume-clamp method (Peňaz, 1973). A finger cuff is attached to participant's middle finger. Inside the cuff is an infrared transmission plethysmograph which maintains the arterial walls at a set diameter. The Finometer has been used extensively in cardiovascular and clinical research; and meets the validation criteria of the Association for the Advancement of Medical Instrumentation and the revised protocol of the British Hypertension Society (Schutte, Huisman, Van Rooyen, Oosthuizen, & Jerling, 2003). CO and TPR indicators are provided based on the validated Modelflow modelling method (Wesseling, De Wit, Van der Hoeven, Van Goudoever, & Settels, 1995; Wesseling, Jansen, Settels, & Schreuder, 1993).

2.3.2. Measures of affect.

2.3.2.1. Positive Negative Affect Schedule (PANAS). The PANAS (Watson, Clark, & Tellegen, 1988) is a 20-item scale measuring state positive and negative affect. This measure asks participants to indicate the extent to which they agree with a number of statements from 1 (*very slightly/not at all*) to 5 (*extremely*). Scores are summed for each subscale providing a total positive affect (PA) score and total negative effect (NA) score. The PANAS demonstrated good internal reliability at baseline and post task for both visits, Cronbach's α was ≥ .68 for each scale.

2.3.2.2. Self-report questionnaires. Participants were asked, pre- and post task to rate on ten-point Likert scale from 1 (*not at all*) to 10 (*extremely*) the extent to which they felt stressed or anxious.

2.3.3. Stressor tasks.

2.3.3.1. Active stress task. A speech task was employed, using standardized presentation of word stimuli. A block of forty words were presented on Superlab (Version 2.02; Cedrus Corporation; San Pedro, CA). For the present study, forty neutral-emotion words, and forty negative-words, were chosen from the Affective Norms for English Words (ANEW; Bradley & Lang, 1999); a set of words rated in terms of valence (negative/positive emotions elicited) and arousal (high, low).Words were selected using previously reported cut-offs for arousal and valence; words were deemed negative if they had an arousal value greater than 6.00 and a valence value less than 4.00, and neutral if the arousal value was less than 5.45 and the valence value was between 4.00 and 6.00 (Scott, O'Donnell, Leuthold, & Sereno, 2009). This format of speech task has previously been shown to reliably elicit activity of the cardiovascular system (e.g., Hughes & Callinan, 2007; O'Súilleabháin, Howard, & Hughes, 2018).

2.3.3.2. Passive stress task. The video of the participant completing the speech task was used as a passive stress task. This builds on paradigms previously developed, where participants were filmed completing an active task and later shown this film-clip to elicit a passive coping context (Harris, 2001; Hartley et al., 1999; Soye & O'Súilleabháin, 2019).

2.4. Procedure

On both visits, all participants visited the laboratory at an assigned time slot between 8:30am and 2pm to minimize the impact of diurnal changes to blood pressure. On both visits, participants were greeted by the researcher and seated at a desk with a personal computer screen, separated from the experimenter by a partition. Demographic information was recorded, and participants completed a 20-minute acclimatization period. During this time, participants completed a series of psychometric scales and were given neutral reading material (the *National Geographic* magazine). Participants were then connected to the Finometer PRO. The Finometer cuff was attached to the middle finger of the non-dominant hand. After a 2-minute recording period, a return-to-flow systolic calibration was performed. Resting cardiovascular measures were taken during an official 10-minute baseline period while participants completed the affective scales (the PANAS, and self-reported stress and anxiety). Again, reading material was provided to lower the risk of potential boredom and/or rumination arousal, employing the Vanilla resting baseline as recommended by Jennings, Kamarck, Stewart, Eddy, and Johnson (1992). Following this period on visit 1, participants were verbally informed that words would appear on the computer screen, and to talk about each word for as long as possible. The task lasted for five minutes. Participants were randomly presented from either a block of forty neutral-emotion or negative-emotion words preselected from the ANEW[1]. The experimenter evaluated when to change the word

[1]There were no differences between participants who spoke about negative words (n = 13) and neutral words (n =12) in terms of CVR to the task, self-reported affect, and performance (in terms of number of words spoken about in 5-minutes), all p's > .176, .207, and .643, respectively.

presented; after three seconds of silence (as measured by a stopwatch), or repetition. Participants were instructed to look at the camcorder as much as possible during the task as the recorded video-clips would later be analysed for body language; this instruction was to heighten the socio-evaluative aspects of task. After the task, participants completed the affective measures again. On visit 2, following a 20-minute acclimatization period and 10-minute baseline, participants were instead verbally informed that the video-clip would be played on the screen in front of them and to simply watch it. They then viewed themselves completing the task from visit 1 on-screen for five minutes. Again, on visit 2 participants filled out the affective rating scales at baseline, and immediately following the video-clip task. Following completion of the laboratory session at both visits, participants were debriefed and thanked for their participation.

3. Results

3.1. Overview of Analyses

The mean values for each cardiovascular parameter (SBP, DBP, HR, CO, and TPR) during the baseline preceding each task (active and passive), and during each task (active and passive) were calculated and are presented in Table 1. Internal consistency for each cardiovascular variable was excellent with Cronbach's $\alpha > .89$. TPR values did not meet the assumption of normality, even after treatment of outliers. TPR values 2*SD* above and below the mean were transformed to the 2*SD* above/below the mean to improve normality; however, neither this transformation or the removal of outliers nor achieved normality. Therefore, outliers were retained and non-parametric tests were employed when conducting analyses involving TPR values.

HP and CD values for each task were calculated using the computational method proposed by (Gregg et al., 2002). One-samples *t*-tests were conducted to examine HP and CD changes in response to the active and passive task. HP values were not normally distributed,

therefore HP scores 2*SD* above and below the mean were transformed to the 2*SD* above/below the mean to improve normality. This resulted in two adjusted HP scores for both tasks. Likewise, CD scores in response to the passive task were not normally distributed, one CD score was transformed to 2*SD* above the mean to improve normality.

Effect sizes for correlations are presented as *r*, with values of .10, .30, and .50 taken to be indicative of small, medium, and large effect sizes respectively (J. Cohen, 1988). Effect sizes for repeated measures and one-sample *t*-tests were calculated using G*Power, and are presented as *d*, with values of .20, .50, and .80 indicative of small, medium, and large effect sizes. Effect sizes are presented as partial η^2 for ANOVA analyses with values of .04, .25, and .64 taken to demonstrate small, medium, and large effects, respectively (J. Cohen, 1992).

3.2. Preliminary Analyses

Independent samples *t*-tests confirmed there were no significant differences between participants who returned to complete visit 2 (the passive task), and who did not, in terms of age ($p = .918$), SBP, DBP, HR, or CO reactivity (all ps > .150), and self-reported affect (all ps > .058) during the speech task (visit 1). Full results are reported in Table 2. Nor was there a difference in TPR reactivity, $z = -0.30$, $p = .765$, $r = .04$, or in sex between these groups, $\chi^2 = 2.09$, $p = .149$, $w = .091$. Previous research has demonstrated that individuals with blunted, that is, lower CVR to stress tasks, tend not to return for follow-up laboratory visits (Ginty, Brindle, & Carroll, 2015); this was not the case with the current sample.

"INSERT TABLE 1 ABOUT HERE"

"INSERT TABLE 2 ABOUT HERE"

3.3. Psychological Experience of the Stress Task

A series of 2 × 2 repeated-measures ANOVAs were conducted. There was a main effect of task on NA, $F(1, 23) = 8.52$, $p = .008$, partial $\eta^2 = .270$. Participants reported more NA after the active task, compared to the passive task. Means and standard deviations for all self-report data are presented in Table 1. There was no main effect of task on PA, stress, or anxiety (all p's > .152), nor was there any main effect of phase on self-reported affect (all p's > .068). There was also no Phase × Task interaction effects, all p's > .088. Full results of all ANOVA analyses are reported in Table 3. The current findings suggest that there was no difference between the two tasks in terms of self-reported anxiety, stress, and PA, however the active task elicited greater NA than the passive task.

"INSERT TABLE 3 ABOUT HERE"

3.4. Resting cardiovascular assessment

Repeated measures t-tests found significant differences in resting SBP and DBP between the active and passive baselines, SBP; $t(24) = 3.35$, $p = .003$, $d = 0.67$, 95% CI [2.07, 8.69], DBP; $t(24) = 4.17$, $p < .001$, $d = 0.83$, [2.94, 8.72]. SBP and DBP were significantly lower during the baseline preceding the passive task (the second laboratory visit). Means and standard deviations are reported in Table 1.

3.5. Cardiovascular reactivity

A series of 2 × 2 within-subjects ANOVAs were conducted to compare reactivity between the active and passive tasks. The first within-subjects factor was task type; active and passive. The second within-subjects factor was phase; baseline and task. ANOVA confirmed main effects for phase on SBP, $F(1, 24) = 42.64$, $p < .001$, partial $\eta^2 = .64$, DBP, $F(1, 24) = 60.97$, $p < .001$, partial $\eta^2 = .72$, HR, $F(1, 24) = 17.76$, $p < .001$, partial $\eta^2 = .71$, CO, $F(1, 24) = 16.55$, $p < .001$, partial $\eta^2 = .41$, and TPR, $F(1, 24) = 4.89$, $p < .001$, partial η^2

= .17. Figure 1 demonstrates that, overall, cardiovascular parameters increased from baseline to task, with the exception of lower HR during the passive task. Means and *SD*s are presented in Table 1.

There were main effects for task type on SBP, $F(1, 24) = 29.27$, $p < .001$, partial $\eta^2 = .55$, and DBP, $F(1, 24) = 32.91$, $p < .001$, partial $\eta^2 = .58$. As can be seen in Figure 1, across the two phases of the tasks, SBP and DBP levels were higher during the active task. There were no main effects for task type on HR, CO, or TPR (all *p*s > .068).

There were task type × phase interaction effects on SBP, $F(1, 24) = 29.27$, $p < .001$, partial $\eta^2 = .55$, DBP, $F(1, 24) = 35.95$, $p < .001$, partial $\eta^2 = .60$, HR, $F(1, 24) = 57.55$, $p < .001$, partial $\eta^2 = .71$, CO, $F(1, 24) = 9.57$, $p = .005$, partial $\eta^2 = .298$, but not TPR, $F(1, 24) = .03$, $p = .858$.

"INSERT FIGURE 1 ABOUT HERE"

3.6. Comparison of Active and Passive Task

As the factorial ANOVAs did not allow direct comparison of patterns of reactivity associated with passive and active tasks, it was decided to examine each task separately, using paired samples *t*-tests.

3.6.1. Active task. The active task resulted in an increase in SBP, $t(24) = -7.10$, $p < .001$, $d = 1.42$, [-19.19, -10.55], DBP $t(24) = -8.34$, $p < .001$, $d = 1.67$, [-11.75, -7.09], HR $t(24) = -6.89$, $p < .001$, $d = 1.08$, [-7.41, -3.99] and CO $t(24) = -4.36$, $p < .001$, $d = 0.79$, [-0.97, -0.35] from baseline to task. Means and standard deviations are presented in Table 1. There was no change in TPR (*Md* = 0.86) to task (*Md* = 0.88), $z = -1.51$, $p = .132$, $r = .21$. SBP, DBP, HR, and CO all increased from baseline to task in response to the active task; however, the active task had no effect on TPR.

3.6.2. Passive task. The passive task resulted in a significant increase in SBP, $t(24) = -3.69$, $p = .001$, $d = 0.74$, [-7.95, -2.25] and DBP, $t(24) = -3.91$, $p = .001$, $d = 0.79$, [-4.47, -

1.38] from baseline to task. TPR also significantly increased from baseline (*Md* = 0.84) to task (*Md* = 0.87), *z* = -2.19, *p* = .028, *r* = .31. There were no significant changes in HR, *t*(24) = 1.64, *p* = .114, *d* = 0.33, [-0.25, 2.15] or CO, *t*(24) = -1.05, *p* = .306, *d* = 0.21, [-0.32, 0.11]. Means and standard deviations are presented in Table 1. The passive task elicited increases in SBP, DBP and TPR, but had no effect on HR or CO.

3.6.3. Magnitude of reactivity. Reactivity scores were calculated separately for each task by subtracting baseline cardiovascular values from task values. A series of repeated measures *t*-tests, using the computed reactivity scores, indicated that SBP, DBP, HR, and CO reactivity to the *active* task was significantly greater than reactivity to the *passive* task: SBP, *t*(24) = 5.43, *p* <.001, *d* = 1.09, [6.05, 13.49]; DBP, *t*(24) = 5.99, *p* < .001, *d* = 1.20, [4.26, 8.73]; HR, *t*(24) = 7.59, *p* < .001, *d* = 1.08, [4.84, 8.46]; CO, *t*(24) = 3.21, *p* = .004, *d* = 0.79, [0.21, 0.96]. There was no difference in TPR reactivity elicited by the tasks, *z* = -.87, *p* = .375.

3.7. Hemodynamic profile

One-samples *t*-tests indicated that both tasks elicited significant CD changes: passive task, *t*(24) = 2.92, *p* = .007, *d* = 0.58, [0.00, 0.02]; active task; *t*(24) = 9.19, *p* < .001, *d* = 1.84, [0.03, 0.04]. In terms of HP, the passive task did not elicit significant HP change, which is indicative of a mixed response profile, *t*(24) = 1.07, *p* = .297, *d* = 0.21, [-0.01, 0.03]. In contrast, the active task elicited a myocardial response profile, *t*(24) = -2.00, *p* = .029 (one-tailed), *d* = 0.40, [-0.04, 0.00], indicated by the negative *t*-value. A one-tailed *t*-test confirmed that HP values were significantly higher than during the active task, *t*(24) = 1.78, *p* = .044, confirming that the active task was associated with a myocardial profile. Means and standard deviations are reported in Table 1.

4. Discussion

The present study confirmed that the passive stress-task employed was successful in eliciting a physiological stress response. Watching the video-recording resulted in observable increases in blood pressure; changes driven by an increase in TPR responding (with little or no change in CO). This hemodynamic response pattern is suggestive of a more vascular response. While this pattern of responding is consistent with reported cardiovascular responses to other, more traditional, passive stressors such as the cold pressor task and film-clip stimuli (e.g., Hurwitz et al., 1993; Saab et al., 1993; Sherwood et al., 1990; Winzer et al., 1999), the current task controls for potential confounds associated with earlier operationalisations of passive stress. Furthermore, this response pattern aligns with the hypothesis that blood pressure responses during passive tasks are driven primarily by alpha-adrenergic sympathetic activation and less beta-adrenergic activation (Obrist, 1981). In contrast, the active task elicited a cardiovascular response marked by cardiac activation with observable increases in blood pressure, HR, and CO, with little or no change in TPR.

A comparison of the magnitude of cardiovascular reactivity elicited by each task confirmed that the active task was associated with greater SBP, DBP, HR, and CO reactivity relative to the passive stress task. Research comparing active and passive stressors consistently report active tasks elicit greater cardiovascular reactivity than passive tasks; strengthening the validity of the current passive stress task (e.g., Nyklicek et al., 2005; Patterson et al., 1994; Soye & O'Súilleabháin, 2019). Notably, the active and passive tasks did not differ in terms of task appraisals, with the exception of NA. There was no difference in the degree of PA, self-reported stress, or anxiety elicited; highlighting that participants experienced similar levels of stress during each task.

We extended past research by applying the computational HP-CD model proposed by Gregg et al. (2002) to examine the underlying hemodynamic response profile. As expected,

the active task elicited a myocardial hemodynamic profile, thought to indicate a challenge-oriented response (Mendes, Reis, Seery, & Blascovich, 2003; Tomaka, Blascovich, Kibler, & Ernst, 1997). We expected the passive task to elicit a vascular response profile; however, a mixed hemodynamic profile was observed. It is worth noting that not much research has examined cardiovascular responding during passive stress using the HP-CD model. Although a vascular response was noted in three studies (Gregg et al., 2002; James & Gregg, 2004; Ottaviani et al., 2006), in the latter only women demonstrated a vascular response; men had a mixed hemodynamic profile. Furthermore, two of these studies used passive tasks that were physical in nature, not psychological. It is possible that task differences led to the observed responses. Future research should employ *psychological* passive tasks, such as the current stressor, in order to elucidate this relationship.

It must be acknowledged that the sample size of the current study is small ($N = 25$). Therefore, the sample size should be taken into consideration when interpreting the reported results. However, due to the implementation of a repeated measures design the statistical tests employed had sufficient power to detect large effects and, to a degree, detect medium effects. Furthermore, the observed cardiovascular responses were in the hypothesised directions. It is also worth noting that a similar sample size has been employed by past research comparing active and passive stressors (20-30 participants; Bosch et al., 2001; Harrison et al., 2000; Hubert & de Jong-Meyer, 1991; Hurwitz et al., 1993; Isowa et al., 2004; Lovallo et al., 1985; Saab et al., 1992; Winzer et al., 1999). Future research is needed to replicate the validity of this novel stress task in a larger sample and include a more diverse sample in terms of ethnicity and age. In particular, the observed results for self-reported affect need to be replicated. Typically, past research has observed medium effect sizes for an increase in self-reported stress (Gallagher, O'Riordan, McMahon, & Creaven, 2018; McMahon, Creaven, & Gallagher, 2019) and anxiety (Lu, Wang, & Hughes, 2016; Lu, Wang, & You, 2016) in

response to speech tasks. However, in the present study the passive task had no effect on self-reported stress or anxiety. Perhaps due to the sample size the present study was underpowered to detect such effects, should they exist; future research should address this potential issue.

It must also be noted that the order of tasks could not be counterbalanced. Viewing the video-clip of the speech task was dependent on the speech being recorded beforehand. Therefore, we cannot claim the results were not influenced by order effects. A potential way to counterbalance the task order is to ask participants to watch video-clips of someone else completing the same task. Future research could test if watching someone else complete the task elicits a passive coping context or is it the self-relevance of the stressor that elicits the cardiovascular stress response observed. Furthermore, in the present study participants completed the task alone. While this overcomes a limitation with past research, where the experimenter and/or confederates were present (Harris, 2001; Schwerdtfeger & Rosenkaimer, 2011; Soye & O'Súilleabháin, 2019), it does not allow us to determine the influence, if any, social evaluation has on the affective and physiological experience of the task. Future research can address this by employing an additional condition; an active speech task, a passive viewing phase (alone), and a passive viewing phase (with the experimenter), in order to examine this relationship.

The observed pattern of results demonstrates the utility of using this paradigm to elicit a passive coping context. First, the current passive stress task was successful in eliciting a notable cardiovascular stress response. Furthermore, participants' psychological experience of the task (in terms of PA, perceived stress, and anxiety) was no different to their experience during a well-established active stress task (Hughes & Callinan, 2007; O'Súilleabháin et al., 2018). Second, the task elicited a pattern of cardiovascular responding consistent with other more traditional passive stress tasks. Blood pressure increased, and this increase was driven

by vascular, rather than cardiac responding. This extends research previously employing this paradigm (Schwerdtfeger & Rosenkaimer, 2011; Soye & O'Súilleabháin, 2019). Third, in the present study participants watched the task alone so any demonstrable changes in cardiovascular responding were a result of watching the video-clip, not a result of evaluative apprehension; a confound with previous studies employing this method. Fourth, examination of the underlying hemodynamic profile of the stressors was somewhat consistent with past literature; the active task elicited a myocardial profile and the passive task a mixed response.

Most importantly, the current passive task offers several advantages over previous tasks considered to elicit passive coping. The psychological nature of the stressor ensures that observed differences in physiological responses were due to changes in cognitive and motivational states, rather than external physical stimuli (such as during the cold pressor task). Likewise, the task presented participants with a more socially relevant experience compared to watching an excerpt from a film or viewing negative imagery. The self-relevant aspect of the task strengthens the ecological valid of the coping context, beyond that provided by previous passive stressors. The presented passive stressor is relatively easy to employ and has been shown to effectively perturb the cardiovascular system even when used in the same laboratory session as the speech task (e.g., Soye & O'Súilleabháin, 2019). Future studies should consider employing this paradigm; it overcomes limitations with previous passive stressors and is associated with a cardiovascular response pattern consistent with other passive stress tasks.

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Table 1.

Mean (with SDs) cardiovascular variables, and self-reported affect, during baseline and task

	Passive Task				Active Task			
	Baseline		Task		Baseline		Task	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
SBP (mmHg)	115.35	9.42	120.44*	13.99	120.73	10.30	135.59*	18.88
DBP (mmHg)	66.96	5.78	69.88*	8.32	72.79	7.08	82.21*	10.46
HR (bpm)	78.98	13.78	78.03	13.74	78.20	12.23	83.90*	12.88
CO (lpm)	6.53	1.62	6.64	1.83	6.29	1.74	6.95*	2.21
TPR (pru)	0.85	0.16	0.88*	0.16	0.95	0.26	0.99	0.32
HP			0.01	0.04			-0.02	0.05
CD			0.01	0.02			0.04	0.02
NA	13.04	3.20	13.32	3.45	13.80	3.99	14.56	4.37
PA	28.00	7.45	27.64	8.56	30.24	6.23	28.23	7.54
Stress	3.28	1.99	3.32	2.06	3.16	1.77	4.20	2.31
Anxiety	3.24	2.11	3.32	2.25	3.16	1.72	4.24	2.20

Note. * denotes a significant increase from baseline to task (*ps* < .05)

Table 2.

Examination of differences between individuals who completed both tasks, and just the first task

	t	*df*	*p*	Lower CI	Upper CI
Age	0.10	46	.918	-0.87	0.77
SBP Reactivity	1.07	46	.289	-9.88	3.01
DBP Reactivity	0.86	46	.393	-4.72	1.89
HR Reactivity	0.14	46	.890	-2.60	2.26
CO Reactivity	1.47	46	.150	-0.64	0.10
HP	0.69	46	.495	-0.02	0.04
CD	0.82	46	.417	-0.02	0.01
Stress baseline	-0.79	46	.431	-1.18	0.51
Anxiety baseline	0.37	46	.711	-0.92	1.33
PA baseline	0.99	46	.329	-2.17	6.33
NA baseline	0.38	46	.707	-2.16	3.16
Stress post task	1.95	46	.058	-2.46	0.04
Anxiety post task	0.55	46	.587	-1.75	1.00
PA post task	0.60	46	.549	-3.21	5.96
NA post task	0.61	46	.544	-2.01	3.76

Table 3.

Results of 2 × 2 ANOVAs for self-reported affect

	Main effects for phase			Main effects for task			Phase × Task Interaction effects		
	F	η_p^2	*p*	*F*	η_p^2	*p*	*F*	η_p^2	*p*
NA	0.58	.024	.456	8.52	.270	.008*	0.18	.008	.672
PA	2.12	.084	.159	0.84	.035	.369	0.55	.023	.466
Stress	2.04	.078	.167	2.19	.083	.152	2.27	.087	.145
Anxiety	3.65	.132	.068	1.95	.075	.176	3.16	.116	.088

Note. * denotes a significant *p* value (< .05).