Heart talk: Emotional inner speech increases heart rate

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Abstract

In this pre-registered study, we investigated whether emotional inner speech influences heart rate. Participants were asked to engage in 3-minute sessions of: positive inner speech, negative inner speech, or inner counting while their heart rate was monitored. Participants were lying on a bed and asked to remain still. Motion tracking was applied to control for body movement. Median heart rate across each inner speech session was analyzed and a significant difference was found between emotional inner speech and inner counting. No difference between positive and negative inner speech was observed. Post-hoc analyses investigated the relationship between movement and heart rate and found an effect with a peak lag of approximately 14 seconds. Removing these effects did not change the effect of emotional inner speech. Additional analyses showed that heart rate and respiration rate were linked. Including respiration rate as a covariate did not alter the effect of emotion.

Keywords: inner speech; emotion; heart rate; motion tracking

Introduction

It has previously been documented how inner speech/self-talk is more or less omnipresent in human consciousness (Alderson-Day & Fernyhough, 2015; Nedergaard, et al., 2021) and may play a causal role in attention and self-control (Nedergaard, et al., 2023a; Nedergaard, et al., 2023b; Wallentin & Nedergaard, 2023). Here, we investigate how self-talk may be involved in evoking emotion and/or in emotional control (Fritsch & Jekauc, 2020). This has important implications for theories on psychological stress, rumination and depression (Lawrence & Schwartz-Mette, 2019).

Inner speech is often linked to mood (Alderson-Day, et al., 2018; Ellis, 1962; Morin, et al., 2011). The causal relationship between the two, however, has not been established. Does negative mood impact inner speech, or does negative inner speech cause depressed mood? One way of thinking about the difference is in terms of physiology. It may be the case that mood is simply a physiological state and that inner speech is a consequence of being in such a state. Alternatively, mood could be considered a mental phenomenon and physiological state the consequence. A textbook controversy in cognitive neuroscience (e.g. Gazzaniga, et al., 2019; Ward, 2020) involves the question put forward by William James and Carl Lange of whether changes in bodily arousal, such as changes in heart rate, lead to changes in experienced emotions (James, 1884; Lange, 1885) or whether experienced emotions lead to physiological reactions (Cannon, 1927). A famous study by Schachter and Singer (1962) investigated this by injecting participants with the stress hormone adrenaline. This led to changes in heart rate, blood pressure, and respiration. The drug by itself was found not to lead to self-reported experiences of emotion, contrary to the James-Lange theory. Participants, on the other hand, did self-report an exaggerated emotion in the presence of an appropriate context.

The present study investigated the same controversy from the opposite angle. If inner speech has a causal effect on mood, one would expect it also to have a causal effect on physiological parameters related to stress and mood. We therefore set out to test if inner speech and especially emotional inner speech can be found to change heart rate.

Among the sources of changes in heart rate are signals from the sympathetic (SNS) and the parasympathetic (PNS) parts of the autonomic nervous system (ANS) (Johnson, 2013; Malik, et al., 1996; Malpas, 2002; Stauss, 2003). The SNS response involves the neurotransmitter adrenaline to be released, which binds to beta-adrenergic receptors on the heart and causes an increase in heart rate and blood pressure (Coote & Chauhan, 2016; Hoffman & Lefkowitz, 1982). This is thought to happen at the 0.1 Hz time scale, i.e. around 6-10 s cycles (Stauss, 2003). The PNS counteracts the effects of the SNS, promoting relaxation and recovery. The

In L. K. Samuelson, S. L. Frank, M. Toneva, A. Mackey, & E. Hazeltine (Eds.), *Proceedings of the 46th Annual Conference of the Cognitive Science Society.*

neurotransmitter acetylcholine is released by the vagus nerve, which binds to muscarinic receptors on the heart, leading to a decrease in heart rate and a decrease in the force of contraction (Malik, et al., 1996). This effect can happen at a slightly faster time scale (2.5-6.0 s cycle length in humans) and is also influenced by respiratory rhythm (Stauss, 2003).

The autonomic nervous system is, as the name indicates, thought to be relatively independent of cognition (Clarke & Jacyna, 1987). There is, however, evidence that some people are able to change their heart rate at will (Carroll & Whellock, 1980; De Pascalis, et al., 1991; Delizonna, et al., 2009; Levenson & Ditto, 1981; McFarland, 1975; Whitehead, et al., 1977). One study (De Pascalis, et al., 1991) asked participants about the strategy used for increasing heart rate and found that 52 percent used "thoughts about motor activity, anxiety, fear, anger or aggressive ideas" while 41 percent used "self-instruction about the physiological responses to be controlled." Both of these strategies could be argued to involve inner speech, but the immediate effects of inner speech on heart rate and the relationship with emotional valence has not been investigated.

Questionnaires probing the different varieties of inner speech (Alderson-Day, et al., 2018) have found that positive and negative inner speech group in two different experiential factors, P: The positive/regulatory factor and E: The evaluative/critical factor. Positive inner speech is grouped with speech used for calming the speaker down, while negative inner speech is involved in evoking self-criticism and anxiety. Based on this, we hypothesized that negative inner speech would be more efficient at evoking sympathetic responses, while positive inner speech would do so to a lesser extent, perhaps even evoking a parasympathetic response which would decrease heart rate relative to negative speech and potentially relative to a non-emotional control condition. There is thus reason to think that emotional inner speech could affect heart rate and moreover that positive and negative inner speech might do so differently.

Hypotheses

The larger aim preregistered before this study is to investigate whether cognitive processes can influence "autonomic" bodily processes. The hypothesis is that it can. Specifically, we hypothesize that:

1) emotional inner speech can induce increased heart rate compared to non-emotional inner speech (inner counting) in the absence of any physical movement.
2) emotional inner speech with negative valence will increase heart rate to a larger extent than inner speech with positive valence, in the absence of any physical movement.

Methods

Pre-registration

The experimental protocol and analysis strategy was pre-registered: https://osf.io/br4zt

Experimental procedure

Participants were placed on a daybed and rested for 10 minutes prior to the experiment, while they were instructed and sensors were positioned on their body (Figure 1). Participants were instructed to talk internally to themselves in three different conditions, each lasting 180 seconds per trial:

- C: Neutral control condition (internal counting)
- P: Positive self-encouraging self-talk
- N: Negative self-degrading self-talk

Participants were lying on a bed with eyes closed during trials and eyes open in the brief intervals between trials. Instructions to begin and end trials were given using beeps played using PsychoPy (Peirce, et al., 2019). Participants wore headphones to attenuate disruption from external noise (Figure 1).

The experiment consisted of two sessions, each with four trials. One session consists of two P/N and two C trials, e.g.:

- Session 1: C-P-C-P (or P-C-P-C),
- Session 2: N-C-N-C (or C-N-C-N),

The same types of emotional inner speech trials were always grouped in the same session to minimize the risk of misunderstanding instructions and keep cognitive load due to experimental complexity to a minimum. The order of sessions and conditions was pseudo-randomized across participants to ensure an equal distribution of all possible combinations. Each trial was separated by a 10 s break.

In order to allow for individual differences, participants were not given specific instructions about how to talk to themselves but told to use the words and content that they would find appropriately negative or positive. A couple of examples were provided by the experimenter for inspiration, but it was stressed that participants could say whatever they decided and that they would not be questioned about the content. Participants were further instructed that inner speech could be in whatever language the participant preferred and that repeating the same thoughts was allowed. During inner counting trials, participants were instructed to count at approximately one Hz (i.e. one count per second).

All participants were debriefed after the experiment to ensure that their emotional level had returned to normal.

Heartrate measurement

Heart rate was measured with a BIOPAC BioNomadix system (1000 Hz sample rate), which was connected to three electrodes placed on the skin of the participant; one on each collarbone and one on the lowest rib (Figure 1). Using beat-to-beat intervals, the data was converted to heart rate (beats per minute – bpm.) and down-sampled to 1 Hz. We then calculated median heart rate per 180 s. trial.

Movement control

Physical motion was measured using Qualisys motion tracking (100 Hz sample rate) with sensors on each foot,

hand, cheek, and one sensor on the abdomen and chin, totaling 8 sensors (Figure 1).

Figure 1: Physical movement was monitored with Qualisys motion capture using eight sensors on feet, hands, cheeks, abdomen and chin.

Accumulated movement (absolute displacement in any direction) was calculated for each trial. Trials with movement 3 standard deviations above the mean on any sensor was discarded, in accordance with our preregistered analysis plan. We excluded all data from a participant if all trials from a particular condition (positive/negative/neutral) were excluded.

Participants

Based on power analyses, we aimed at 60 participants in our preregistration. Due to technical problems, we were forced to increase the number. 67 participants completed the experiment with a complete heart rate dataset. 66 remained with an intact PsychoPy script with condition logs (41 female, 25 male; median age: 24 years; age range: 19-75 years). 51 participants also had complete motion capture data. After excluding participants due to excessive movement, according to our pre-registered protocol, 48 participants remained. Gender was not deemed significant and was not considered in analyses (Wallentin, 2009, 2020).

Preregistered analyses

Each participant ended up with 8 median heart rate measures (2xPositive, 2xNegative, 4xControl, measured in bpm.). The data were analyzed using mixed-effects modeling (library(lmerTest) Bates, et al., 2014; Kuznetsova, et al., 2017) in RStudio with the following pseudo code:

HeartRate~Emotional/NonEmotional+Positive/Negative +TrialNumber + (1|participant)

The effect of the Emotional(N+P)-NonEmotional(C) contrast tested Hypothesis 1. The effect of the Positive(P)-Negative(N) contrast tested Hypothesis 2.

Post-hoc analyses

Adding random slope The effect of time in the experiment is likely to be different for different participants. We therefore included an additional analysis where trial number was added as a random slope to the regression.

Testing full sample Technical challenges with the motion capture system made it necessary to exclude 15 participants in order to be able to conduct the preregistered analysis. To test if this changed the results, we also performed the analysis on the full heart rate sample.

Testing motion/heart rate correlation We also investigated the relationship between the observed physical motion and heart rate (Figure 2).

Regressing out low frequencies and motion To remove slow drift in the heart rate signal from increased relaxation along the experiment (e.g. see Figure 3 top), we additionally high-pass filtered the heart rate data using a 400 s Butterworth filter and in an attempt to remove additional effects of motion, we regressed out motion effects using a -40 to 40 s. time lag for all motion sensors and repeated the analyses to test if this changed the results (Figure 3).

Testing respiration rate/heart rate correlation Respiration rate has been found to influence heart rate variability (Song & Lehrer, 2003) and heart rate (Ahmed, et al., 1982; De Pascalis, et al., 1984; Marc, et al., 2017). On a subset of the participants, it was possible to calculate respiration rate based on abdominal movement (Hideo & Jun, 2012). We used this to test the relationship between respiration rate and heart rate and to test if changes in respiration rate could potentially explain away changes in heart rate.

Results

The preregistered analysis (n=48) yielded a significant effect of emotional inner speech versus inner counting (coef: 0.86 (±0.29), t(312)=2.97, p<0.005, η_p^2=0.03) in support of hypothesis 1. No difference between positive and negative inner speech was observed (coef: -0.14 (±0.41), t(312)=-0.35, p>0.05), failing to support hypothesis 2.

Adding trial number as a random slope replicated the effect of emotional inner speech (p<0.001, η_p^2=0.06), and again failed to show an effect of positive versus negative inner speech (p>0.05).

Repeating the analysis on the full sample (n=64) replicated the effect of emotional inner speech versus counting (coef: 0.49 (±0.24), t(441)=2.07, p<0.05, η_p^2=0.01). No difference between positive and negative inner speech was observed (coef: 0.04 (±0.33), t(441)=0.14, p>0.05). Including trial number as random slope on the full sample replicated the effect, but with higher t-value for effect of emotion (η_p^2 = 0.03).

Median respiration rate was found to be predictive of median heart rate (n=48, coef: 0.24 (±0.01), t(336)=2.6, p<0.01). However, when including respiration rate in the linear regression as a covariate for explaining heart rate, the

emotional condition remained significant (n=47, coef: 0.74 (±0.21), t(256)=3.5, p<0.001, η_p^2=0.05), along with respiration rate (n=47, coef: 0.21 (±0.06), t(291)=3.4, p<0.001, η_p^2=0.04).

We time-shifted the motion data (absolute displacement) at 1 s intervals from -40 to 40 s. Investigating the cross-correlation between the motion displacement for individual markers and the high-pass filtered heart rate at different temporal lags revealed that movement resulted in an increase in heart rate which peaked 14 seconds after movement (i.e. a peak correlation at 14 s time shift, see Figure 2)

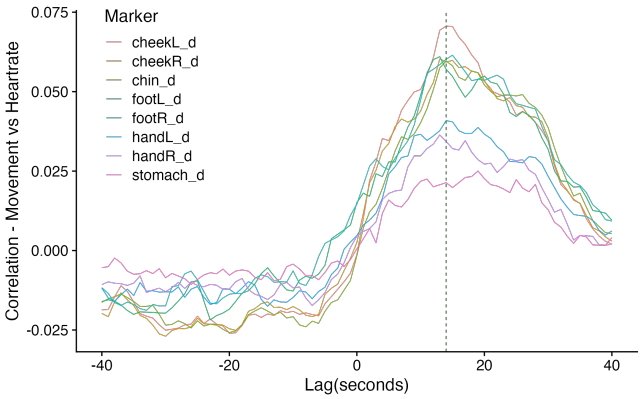

Figure 2: Cross-correlation analysis between high-pass filtered heart rate data and movement (displacement in any direction) at different time lags revealed that body movement causes a heart rate increase peaking with an average 14 s delay (dashed line).

The motion data from all 8 sensors at each lag were therefore used to regress out additional motion effects from the heart rate data (Figure 3). For each time lag we used the motion data as predictors of the heart rate signal in a linear regression. The residuals of this analysis were used in the regression for the next time lag, etc., before summary statistics were again obtained.

After filtering, the data were subjected to a linear mixed-effects regression, again showing an effect of emotion vs counting (coef: 0.077 (±0.02), t(334)=3.88, p<0.001, η_p^2=0.04) and no effect of emotional valence (p>0.05). Note the change in regression coefficient is due to data normalization (see difference in y-axes on Figure 3).

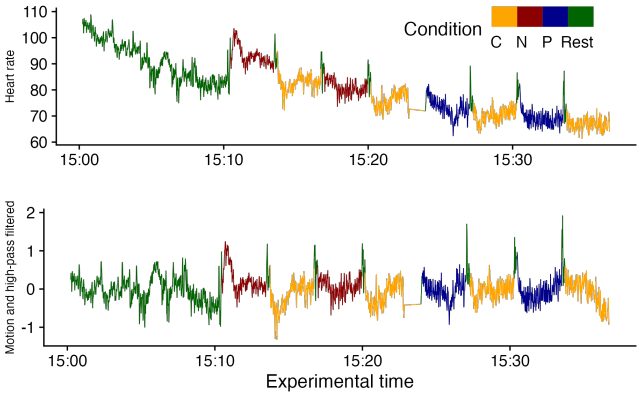

Figure 3: Single participant example. Top: Raw heart rate data. Bottom: z-score normalized, movement and high-pass filtered data. Conditions. C: Counting - Neutral condition; N: Negative inner speech; P: Positive inner speech; Rest: Initial 10 min. rest + 10 s. between trials.

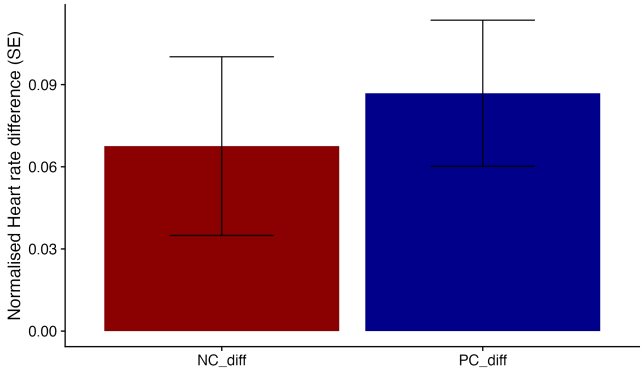

Figure 4: Normalized heart rate differences across participants. NC_diff: Negative inner speech minus Inner counting. PC_diff: Positive inner speech minus Inner counting. Note both are different from zero, but not different from each other. SE: Standard error of mean of the normalized differences.

Discussion

Both our preregistered analysis and additional post-hoc analyses showed a significant effect of emotional inner speech on heart rate relative to inner counting in support of Hypothesis 1. The effect size was small (η_p^2≈0.03). No significant difference between positive and negative inner speech was found (Figure 4), contrary to Hypothesis 2.

Robustness tests and power issues

Subsequent post-hoc analyses that were intended to test the robustness of the findings all gave the same results. These analyses included adding a random slope to the model, regressing out slow drift and motion effects, testing the full

sample from before motion tracking analysis, and also taking effects of respiration into account. The statistical effect thus seems to be small, but robust.

Our preregistered power analysis resulted in a sampling plan of 60 participants. The final analysis only included 48 participants, due to a number of technical issues with the motion capture system. However, when repeating the analysis with all 64 participants, we got the same results. This indicates that the lack of a significant difference between positive and negative inner speech is not due power issues caused by deviations from our sampling plan, and that if there is a difference between the two conditions, it is much smaller than expected and would thus require a larger or different experiment to locate. Nominally, we see positive inner speech yielding a (non-significant) larger heart rate effect than negative inner speech (Figure 4). This makes it less likely that an effect of negative inner speech is hiding in the data and would be observable with a larger study.

Small effect size

Emotional inner speech was found to be associated with an increased heart rate of approximately one beat per minute on average across the 180 s. trials compared to inner counting. This is arguably not a lot for a system which will increase with 10-20 beats per minute by just standing up, and the effect size measure (η_p^2) also indicates that the effect size is in the "small" domain. However, given that heart rate increase is a supposedly autonomic response to a need for oxygen in the body and brain, which happens for many other reasons than emotion (e.g. standing), it is not surprising that the effect is small.

Motor and auditory imagery

As mentioned in the introduction, people have reported to use motor imagery for consciously increasing heart rate (De Pascalis, et al., 1991). This raises the possibility that the imagined speech movements in themselves could be the cause of the observed heart rate effects in the present study. Speaking against this is the fact that the control condition (inner counting) also contained imagined articulation and auditory imagery as well.

Arousal

The difference between neutral and emotional inner speech may thus be linked to arousal. Both positive and negative inner speech may yield an increased sympathetic nervous system response, which includes increased heart rate and heart rate variability (Wallentin, et al., 2011).

Contrary to our hypothesis, negative inner speech did not cause a higher degree of arousal level than positive inner speech, as indexed by heart rate. This speaks against the idea that positive inner speech should have a relaxing effect on the cardiac system. It does not rule out that certain kinds of positive inner speech may have this effect, but the types used by our participants in the absence of any detailed instructions, did not yield this result. On the contrary, we found an increase in heart rate also with positive inner speech relative to counting. This leads us to stress that that there is nothing inherently unhealthy in having your heart rate affected by emotional inner speech or any other arousing activities. In fact, increased arousal may be a positive effect of inner speech and may be one of the causes of the observed beneficial effects of inner speech on boring or straining tasks requiring attention (Nedergaard, et al., 2023a; Wallentin & Nedergaard, 2023). However, inner speech may become a problem if its use is continuously associated with evoked physiological stress responses in the body, e.g. through rumination (Watkins & Roberts, 2020).

An immediate consequence of our findings, if they replicate, would be that if one wants to decrease the autonomic response from negative inner speech as a form of intervention, then replacing it with inner counting will have stronger effect than trying to use positive inner speech. Counting to ten as an anger management strategy could thus turn out to be efficient (Bloxham & Gentry, 2010). There is a need for a renewed focus on self-talk strategies for mood regulation (Fritsch & Jekauc, 2020; Kross, et al., 2014) and control (Nedergaard, et al., 2023b; Wallentin & Nedergaard, 2023).

We cannot rule out, however, that the increased heart rate for emotional inner speech could partly be a result of differences in task difficulty between counting and the more free task of producing an elaborate inner monologue. Task difficulty has previously been found to increase heart rate (Gellatly & Meyer, 1992). Additional experiments attempting to control task difficulty differences between the active and the control condition are warranted.

Body movements

Overt movements are a source of noise in experiments like the present. The nature of the relationship between small body movements and heart rate responses is difficult to find in the literature. We found a transfer between body movement and heart rate increase with a time to peak of 14 seconds, consistent across sensors (Figure 2). Previous investigations of this relationship primarily come from sleep research. Here, one study found that leg movements lead to an increase in heart rate with a time to peak of 6 seconds (Winkelman, 1999). Another study found that heart rate increases preceded movement (Townsend, et al., 1975). One can imagine effects during sleep, e.g. pain, which first cause the heart rate to go up and subsequently lead to movement. Our study suggests that the transfer function in awake, supine participants could be different from the ones observed during sleep and that the effect primarily goes from movement to heart rate and not the other way. There may, however, be individual differences. Investigating this further and controlling for it would be an important part of future studies of the relationship between inner speech and physiology.

Given the short (10 second) breaks between trials in our study, it is possible that some effects of movement during the breaks could spill into the following trials. However, this should not be different for the different conditions, and

furthermore, such an effect would probably only be present in the beginning of the 180 second trial periods (given the 14 s lag) and be eliminated by the use of the median heart rate rather than the mean. In one of the post hoc analyses, we also regressed out all measured effects of movement, and this did not affect the heart rate effect of emotional inner speech. Our 8 motion sensors, however, placed on feet, hands, abdomen and the face, may not have captured all existing motion. It thus remains a possibility that the effect of emotional inner speech could be caused by unmeasured movement. However, we deem this to be unlikely, as it is difficult to move body parts without it simultaneously being picked up by the sensors on the extremities, the torso and the head.

Respiration

We replicated the well-known link between respiration rate and heart rate (Ahmed, et al., 1982; De Pascalis, et al., 1984; Marc, et al., 2017). We found a positive relationship between the two variables, but including respiration rate as a variable in our linear model did not change the effect of emotional inner speech on heart rate. Participants were not instructed to breathe in any particular way, and it has been observed that inner speech changes respiration (McGuigan, 1970; Rounds & Poffenberger, 1931), often in a manner analogous to overt speech where the outflow of air interferes with respiration (Chapell, 1994; Conrad & Schönle, 1979). It may thus be that unmodelled parts of respiration differences can account for some of the heart rate effect of inner speech. One way to control for this would be to conduct the experiment in shorter trials during breath hold or to control for respiration rate in statistical analyses.

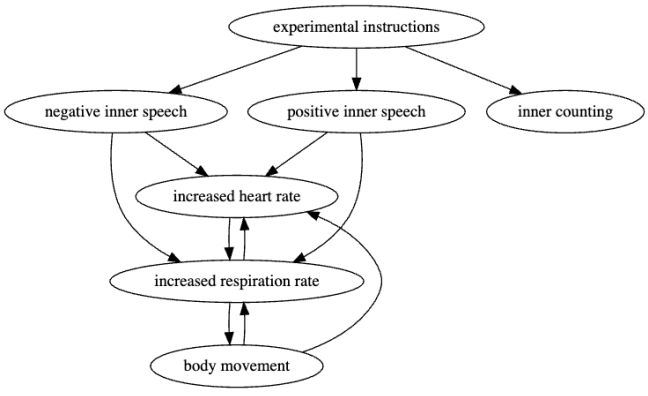

Figure 5: Simplified causal graph of experimental setup and possible effects.

Our findings are compatible with a simplified causal model (Figure 5) in which the experimental instructions cause participants to enter into the three experimental conditions. The two emotional conditions then cause heart rate and respiration rate to increase, which have a bidirectional link with each other. Body movements are not directly caused by the experiment but may nevertheless occur without known causes. These also influence heart rate and respiration rate. Respiration, however, also involves body movement, especially of the abdomen (Hideo & Jun, 2012), making the causal link bidirectional. Testing the validity of this causal model is beyond the present report. An alternative is that one would find bidirectional links between the sympathetic nervous system and emotional inner speech under more ecologically valid conditions, such that physiological arousal, in the right context, as observed by Schachter and Singer (1962), can lead to changes in the content of inner speech. The path between emotional inner speech and increased heart rate may thus be complex, but our study does not allow for testing more dynamic models. Further studies are needed.

Conclusion

Our study found support for the hypothesis that emotional inner speech yields a sympathetic nervous system response, but not support for the hypothesis that this was specific to negative emotional inner speech.

Acknowledgments

This study was funded by a seed grant from the Interacting Minds Centre at Aarhus University.

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