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# Cardiac-sympathetic contractility and neural alpha-band power: cross-modal collaboration during approach-avoidance conflict

Abbreviated title: Contractility-alpha interactions in approach-avoidance

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**Abstract**

As evidence mounts that the cardiac-sympathetic nervous system reacts to challenging cognitive settings, we ask if these responses are epiphenomenal companions or if there is evidence suggesting a more intertwined role of this system with cognitive function. Healthy male and female human participants performed an approach-avoidance paradigm, trading off monetary reward for painful electric shock, while we recorded simultaneous electroencephalographic (EEG) and cardiac-sympathetic signals. Participants were reward sensitive, but also experienced approach-avoidance "conflict" when the subjective appeal of the reward was near equivalent to the revulsion of the cost. Drift-diffusion model parameters suggested that participants managed conflict in part by integrating larger volumes of evidence into choices (wider decision boundaries). Late alpha-band (neural) dynamics were consistent with widening decision boundaries serving to combat reward-sensitivity and spread attention more fairly to all dimensions of available information. Independently, wider boundaries were also associated with cardiac "contractility" (an index of sympathetically mediated positive inotropy). We also saw evidence of conflict-specific "collaboration" between the neural and cardiac-sympathetic signals. In states of high conflict, the alignment (i.e., product) of alpha dynamics and contractility were associated with a further widening of the boundary, independent of either signal's singular association. Cross-trial coherence analyses provided additional evidence that the autonomic systems controlling cardiac-sympathetics might influence the assessment of information streams during conflict by disrupting or overriding reward processing. We conclude that cardiac-sympathetic control might play a

critical role, in collaboration with cognitive processes, during the approach-avoidance conflict in humans.

## Significance statement

Complex behavior likely involves coordination across multiple branches of the human nervous system. We know much of how cortical systems of the brain adapt to cognitive challenges. In parallel, we are beginning to understand that autonomic mediated responses in peripheral organ (cardiac-sympathetic) systems might also play an adaptive role in cognition, particularly complex decisions. We probed if such signals have separate or collaborative associations with behavior, using computational models of decision behavior, brain (electroencephalography) and cardiac-sympathetic (contractility) data. Our evidence suggests that these systems might work together, as humans attend to all available information when resolving particularly conflicting decisions. The cardiac-sympathetic system may be part of a coordinated response that helps balance the human tendency to overly focus on rewards.

## Introduction

Our nervous system and coupled body evolved together to be flexibly responsive, allowing rapid and often anticipatory changes to meet a broad array of cognitive and physical challenges presented in dynamic environments. Decades of research in cognitive neuroscience have characterized flexible cognitive mechanisms and underlying cortical systems for preserving goal-directed function when external circumstances change. Meanwhile, autonomic reactivity in peripheral organ systems, such as the cardiac-sympathetic branch, is well documented in tasks requiring momentary goal-directed changes in mental and physical exertion, showing appropriate reactivity (just enough, just in time) to tasks at hand (Richter et al., 2008; Richter et al., 2016; Stump et al., 2023). More recent evidence extends cardiac-sympathetic reactivity to complex cognitive

challenges such as value-based decision making (Dundon et al., 2020, 2021). However, it remains unclear whether these peripheral responses are independent of cortically mediated cognition or if the regulatory systems controlling these responses are more integrally involved in cognitive processes. A crucial next step is to therefore pinpoint the specific cognitive mechanisms that the cardiac-sympathetic system aligns with.

Emerging event-related evidence suggests cardiac-sympathetic reactivity might be particularly relevant in value-based situations that involve some manner of "conflict" and where decisions

must incorporate negative information or costs (Ogden et al., 2019; Dundon et al., 2020; Dundon et al., 2021). While such a reactivity profile could be epiphenomenal, it is also consistent with a broader literature showing sympathetic involvement when humans face increasing uncertainty (Palacios-Filardo and Mellor, 2019) and difficulty (Richter et al., 2008), or a requirement to explore alternative goal-relevant stimuli (Aston-Jones and Cohen, 2005) with a specific emphasis on incorporating negative information (Garrett et al., 2018). Together, these findings suggest cardiac-sympathetic reactivity reflects a process that may be centrally generated and part of a coordinated response that helps balance the human tendency to overly focus on rewards (Garrett et al., 2014; Sharot and Garrett, 2016; Pedersen et al., 2021). However, to date, no study has tracked the computational-behavioral and neural processes relevant for value-based conflict and reward sensitivity, and thereafter probed whether reactivity in cardiac-sympathetics is independently or collaboratively associated with behavior or cortical activity.

In the present work we therefore use a modified version of the approach-avoidance paradigm (Champion, 1961; Elliot and Thrash, 2002). This paradigm creates states of high "conflict" when the appeal of a reward is near equivalent to the revulsion of a cost (Figures 1A–B). It can also identify sensitivity toward a particular value dimension, such as reward sensitivity (Volz et al., 2017; Shapiro and Grafton, 2020; Pedersen et al., 2021; Figure 1C). We configured the paradigm to additionally record how neural perceptual signals measured by electroencephalography (EEG) track reward and cost information, specifically sensory gain (steady-state visually evoked potentials; SS; Pfurtscheller and Aranibar, 1977; Galloway, 1990; Müller et al., 1998; Müller et al., 2006; Gulbinaite et al., 2019) and goal-directed attention (spatially responsive dynamics in the alpha band; Foxe and Snyder, 2011; Klimesch, 2012; Wang et al., 2016). We simultaneously recorded beat-by-beat estimates of contractility (inotropy), which is primarily mediated by noradrenergic sympathetic drive (after adjusting for heart and respiratory rate) and associated

with cardiac reactivity to challenge (Lewis et al., 1974; Light, 1985; Linden, 1985; Newlin and Levenson, 1979; Sherwood et al., 1986, 1990; Callister et al., 1992). To further decompose behavior, and extract fine-grained assays of behavior to correlate with physiology signals, we fitted the drift-diffusion model (DDM) to choice and response time (RT) data (Figure 1D). Initially considered in perceptual contexts (Usher and McClelland, 2001; Ratcliff and McKoon, 2008; Forstmann et al., 2016), parameters from the DDM are an increasingly useful tool for disambiguating the underlying reasons for lengthier RT in more complex value-based contexts (Peters and D'Esposito, 2020; Shahar et al., 2019; Ballard and McClure, 2019; Colas, 2017; Fontanesi et al., 2019; Dundon et al., 2023; Figure 1D).

[Figure 1 here]

Our primary aim was to establish how human participants respond to conflict at the computational-behavioral level. We thereafter tested if cardiac-sympathetics are associated with relevant DDM parameters in a manner that suggests redundancy (i.e., epiphenomenal), independent function or collaboration (i.e., interaction) with perceptual neural signals. In particular, we examined if cardiac-sympathetics aligned with neural processes associated with reward sensitivity, i.e., increased gain of or attention toward either cost information or more symmetric processing of reward and cost.



## Materials and Methods

We recorded continuous multi-channel electroencephalography and cardiac-sympathetic physiology (combined electrocardiography and impedance cardiography) while human participants made approach-avoidance choices regarding offers that varied trial-by-trial in reward and in cost. Each "take-it-or-leave-it" trial offer gradually presented a monetary reward ranging in value from $0.01 to $1.50 and a shock cost ranging in value from minimal to near maximum bearable pain (see trial schematic in Figure 2A). We configured the paradigm to additionally record how EEG signals track reward and cost information, both in terms of sensory gain (steady-state visually evoked potentials; Figure 3A) and goal-directed attention (spatially responsive dynamics in the alpha band; Figure 3B). We additionally divided neural assays into early and late time windows, to capture the dynamics of conflict as decisions unfold, given recent evidence that they might be time-sensitive Shapiro and Grafton (2020).

*Participants*

We recruited an initial sample of 33 human participants, via both word-of-mouth and an online participant recruitment portal operated by the University of California, Santa Barbara (UCSB). We removed six participants from all analyses: One subject accepted more than 90% of offers, two participants' EEG data had an artefact in more than 50% of epochs, and one further subject satisfied both screening criteria. In addition, we removed two participants due to excessive noise in their impedance cardiography data. We accordingly report findings from a final sample of 27 participants. This group had a mean (standard deviation) age of 21.4 (3.3), and 17 were female. All participants were right-handed and attested to no history of cardiovascular or related diseases. Subject remuneration was $20 per hour base rate, with a bonus payment determined by their approach-avoidance behavior, which approximately corresponded to an additional $13.50 per subject. All testing took place during a single session in a quiet, dimly lit experimental suite and

all procedures received approval from the Institutional Review Board at UCSB. Participants provided informed written consent, prior to participating.

*Approach-avoidance paradigm*

*Overview.* We used a modified version of the approach-avoidance task previously employed in nonhuman primate (Amemori and Graybiel, 2012, 2015) and human (Volz et al., 2017; Shapiro and Grafton, 2020; Dundon et al., 2021) experiments. The main modification was the incorporation of reward and cost stimuli with different frequency flicker rates and spatial positioning to facilitate identification of specific cortical activity. Stimuli also appeared gradually on each trial, to facilitate identification of early and late responses. (O'Connell et al., 2012). Similar to prior studies, participants approached or avoided varying levels of monetary reward paired with varying levels of painful electric shock, in trial-by-trial "take-both-or-leave-both" offers. Participants made a total of 352 approach-avoidance choices (split into eight blocks of 44). Their head position was fixed by an adjustable chin and forehead rest, to maintain a viewing distance of 57 cm from the stimulus presentation screen: an ASUS VS278 monitor, viewing area 60 cm width by 33.5 cm height, refresh rate of 240 Hz (inter-frame interval=.004 s). We advised participants to move their bodies as little as possible, to prevent motion-related confounds entering the physiology recordings.

*Trial structure.* Each approach-avoidance trial gradually presented an offer to participants, in which two bars communicating the level of reward and cost slowly appeared. Responses were recorded with button press. The trial schematic is depicted in Figure 2A. During each trial, participants fixated their eyes on a central point ($RGB_{[min=0,max=1]}$=[.750 .750 .750]; diameter=0.221°). The background color remained black ($RGB_{[min=0,max=1]}$=[0 0 0]) at all times,

except for payout trials (see below). Offers comprised four sequential events: (i) baseline, (ii) offer onset, (iii) offer offset and (iv) feedback. (i) Each offer initiated with a baseline period with a duration between 420 and 540 frames (inclusive) drawn with discrete uniform probability on each trial (approx. 1.75 s to 2.25 s). Baseline onset was signified by the immediate appearance of two vertically oriented rectangular dot arrays, each spanning 7.30° width by 27.8° height, comprised of 79 columns and 322 rows of dots (dot diameter=.056°), with centroids positioned at a horizontal eccentricity +/- 3.75° from the central fixation point. (ii) Following baseline, during offer onset, the offer bars gradually communicated the magnitude of the offer's value dimensions, with one bar communicating the level of offered reward and the other bar communicating the level of incurred shock. We drew a different offer on each trial from a two-dimensional decision (reward-by-shock) space with uniform probability, and communicated the magnitude of each dimension by gradually filling in an area of both bars with a relevant offer color (khaki or blue; one color per offer bar). Specifically, contiguous rows of dots, equally portioned above and below the centroid of each offer bar, gradually changed into one of two offer colors. The number of rows changing into an offer color indicated the magnitude offered in that dimension, i.e., the offered reward (no rows: $0.01, to all rows: $1.50) and the offered shock (no rows: minimum pain, to all rows: maximum bearable pain—see "costs" section below). Counterbalanced across participants, reward and shock mapped onto one color for the entire experiment, while color laterality was determined with 0.50 uniform probability before each trial. Offer onset duration was four seconds, with color change controlled by reward and shock gradients that respectively changed dot colors from baseline grey ($RGB_{[min=0,max=1]}$=[.375 .375 .375]) to peak offer color (khaki, $RGB_{[min=0,max=1]}$=[.632 .586 .351]; blue, $RGB_{[min=0,max=1]}$=[.328 .616 .375]) at even step sizes in RGB space over the offer onset frames (n=960). (iii) Following peak onset, during offer offset, offer colors gradually returned to baseline grey over a two-second period. We instructed participants to respond as soon as they decided whether to approach or avoid an offer, with a time limit of the end of offer offset.

Participants registered their responses by pressing z with the index finger of their left hand, or m with the index finger of their right hand. The mapping of z and m onto approach and avoidance responses was fixed for each block of 44 trials, determined prior to the block with 0.50 uniform probability. (iv) Following offer offset, participants observed a feedback screen for one second (nine seconds for payout trials, see below). As depicted in the lower portion of Figure 2A, a "successful" response, i.e., a single response executed during offer onset or offset, led to a confirmation feedback screen containing a snapshot image of the offer (at peak offer colors), accompanied by a written confirmation of their choice. If participants responded more than once, responded during the baseline (pre-onset), or failed to execute any response before the final frame of offer offset, a warning appeared on the screen indicating the relevant error, along with the lateralized response prompts (lower portion of Figure 2A). We stored all error trials and reissued them to participants after the eighth block, to ensure that errors could not be a strategy to circumvent specific offers. All feedback screens (successful, error or payout (below)) also included prompts to remind participants which colors mapped onto the different reward dimensions (indicated with a lightning bolt (shock) or dollar symbol (reward) overlaying the relevant bar), and which button response mapped onto which choice for the given block. This screen was also displayed prior to each block. Finally, offer bars flickered throughout each trial, either left: 12 Hz, right: 13.33 Hz or vice versa, determined with 0.50 uniform probability. We presented all stimuli with customized scripts in MATLAB (Version 2018a, The Mathworks Inc., Natick, MA, USA, https://www.mathworks.com/products/matlab.html) using functions from PsychToolbox-3 (Brainard, 1997; Pelli, 1997; Kleiner et al., 2007). Successfully registered choices were coded either 1 (approach) or 0 (avoid), and response time (RT) was the time (in seconds) between offer onset and choice execution.

*Costs:* Shocks offered to each participant were calibrated a priori in order to range in pain from an individualized subjective minimum to near-maximum level. We administered the costs with cutaneous electrical stimulation (1 s duration; f=100 Hz; λ=2 ms), via two electrodes on the back of the hand. We used a constant current stimulator and train generator (respectively, models DS7A and DG2A, Digitimer, Great Britain), and modulated pain via voltage. We used an identical calibration procedure to previous studies (Shapiro and Grafton, 2020; Dundon et al., 2021). That is, we started 1 mV and gradually increased voltage until participants reported (1) a perception of the shock, then (2) when the voltage began to cause discomfort, and then finally (3) when the voltage caused unbearable pain. Once a level of unbearable pain was reported, we asked them to confirm that this was the maximum pain they could tolerate. This prompt usually spurred participants to accept a further increase in voltage. Once they confirmed reaching an unbearable level of pain, we administered 14 sample shocks, ranging in voltage between (2) and (3) above, and participants reported the level of pain on a scale of 0 to 10. We repeated this entire procedure twice to account for habituation. Shocks offered to each participant then ranged from a lower bound of (2) above to an upper bound of the second estimate of (3) above. We also fitted a sigmoid function to the second set of pain ratings, to first estimate shocks of 0.05, 0.25, 0.50, 0.75, and 0.95 intensity (as per their individualized scales) to provide as sample shocks. We also estimated where their 0.80 level of pain was and excluded trials offering pain equal or greater to this level from payout trials (see below).

*Payout trials*. For safety reasons, we did not administer any electric shocks during the testing session while participants wore physiology electrodes. We instead postponed payout on an infrequent subset of trials, in line with previous work recording physiology signals during similar approach-avoidance paradigms (Shapiro and Grafton, 2020; Dundon et al., 2021). Prior to testing, we selected eighteen "payout trials" (5.11%) with uniform probability across the entire set of offers,

provided the offered shock was below 80% of a subject's maximum pain level. The baseline, onset and offset sequence of payout trials were identical to non-payout trials. However, during the feedback section of payout trials (extended from one to nine seconds), the screen changed from black to red ($RGB_{[min=0,max=1]}$=[1 0 0]; left lower portion of Figure 1C) and participants learned that the monetary reward and shock from that offer would be administered following the testing session (were that offer approached) or that the values would have been administered (were that offer avoided). If participants made a response error during a payout trial, they instead saw the error feedback screen, and the payout trial was added to the list of trials to be reissued. We instructed participants that payout trials could not be predicted before registering a response and to treat each offer as a potential payout trial. Previous work reports that payout trials do not affect behavior on subsequent choices (Shapiro and Grafton, 2020; Dundon et al., 2021).

*Paradigm configuration for perceptual signals.* We configured the paradigm to record how neural perceptual signals measured by EEG track reward and cost information, both in terms of sensory gain (steady-state visually evoked potentials; Figure 3A) and goal-directed attention (spatially responsive dynamics in the alpha band; Figure 3B). The former was achieved by flickering the offer bars throughout each trial, one at 12 Hz, the other at 13.33 Hz. This meant that each trial had a unique frequency "tag" associated with reward and cost. For the latter, we lateralized the reward and cost stimuli to exploit spatially responsive alpha dynamics. For the specific filtering procedure in each case, see the *Neural recordings* section below.

*Behavioral analyses*

*Overview.* We performed initial behavioral analyses to evaluate whether participants were reward sensitive in addition to whether they confronted approach-avoidance "conflict" in established

regions of decision space when performing our task. To conform with previous studies, we used a logistic framework employing maximum-likelihood methods to compute subjective value and all related measurements. All other modeling and statistical tests were performed using hierarchical Bayesian models, in which posteriors were sampled and model fits computed using a combination of HDDM (Wiecki et al., 2013) and pymc3 functions (Salvatier et al., 2016) in Python.

*Logistic choice models.* We used a logistic framework previously reported (Shapiro and Grafton, 2020; Dundon et al., 2021). This framework fits two-dimensional logistic models separately to each individual subject's set of choices (Y), modeling p(approach) as a function of the corresponding set of rewards (X1) and shock costs (X2) offered, with a standard logit function, i.e.:

$$Y=\text{logit}^{-1}(b_0+b_1*X1+b_2*X2). \quad \text{[Eq. 1]}$$

We estimated the maximum likelihood parameters of each participant's model using the mnrfit function in MATLAB after first normalizing X1 and X2 to z-score ranges within participants.

*Reward sensitivity* in approach-avoidance contexts is demonstrated by choices that overweight the reward relative to the offered costs (Figure 1C). In our paradigm we uniformly sampled the reward and cost dimensions, the latter of which was calibrated to span an individualized minimum to maximum (see above section on "Costs" in the section describing the paradigm). We accordingly deduce that participants were reward sensitive within this task context if their logistic

choice coefficients (from Eq. 1) showed a bias toward approach ($b_0>0$) or an overweighting of the reward coefficient ($|b_1|>|b_2|$).

*Approach-avoidance conflict* is highest near the region of decision space where participants are as likely to approach as they are to avoid (Figure 1A). We assessed the level of conflict presented by each trial, accounting for individual differences in subjective valuations, using a previously employed procedure (Shapiro and Grafton, 2020; Dundon et al., 2021). The method uses discrete classification, categorizing trials as either high or low in conflict. For this, from the coefficients of each participant's logistic choice model (from Eq. 1), we first computed the subjective value (the log odds of approach) of each trial as $SV_k=b_0+b_1*x1_k+b_2*x2_k$, where $x1_k$ and $x2_k$ are respectively the reward and cost offered on trial (k), normalized to z-score ranges within participants. In this way SV>0 reflects p(approach)>0.50 and vice-versa. We then classified a trial as high conflict if it was either (a) approached, but where its $SV_k$ was below the median SV of all approached trials, or (b) avoided, but where its $SV_k$ was above the median SV of all avoided trials. These trials are depicted schematically by the fuchsia regions in Figure 1. All remaining trials were classified as low conflict, depicted by the aqua regions in Figure 1. For each trial, its SV and its binary degree of conflict (high vs low) were estimated prior to any screening due to EEG or sympathetic artefact, described in the section below.

We performed additional tests to support that the logistic framework had identified states of high and low conflict. One indication of high conflict is a reduction in choice "consistency". For this, we used a nonparametric assessment of the deviation in choices appearing in bins of the decision space. We divided each participant's decision space into a ten-by-ten grid of equal bins. We enumerated choice consistency for a given bin as the variance in choices across all offers

appearing in it. Choices were numerically assigned x=0 for avoid and x=1 for approach, and variance (V) computed across n trials in each bin as $V_{bin}=\frac{1}{n}\sum_{k=1}^{n}(x_k-\bar{x})$. Positive variance values reflect lower consistency (i.e., different choices registered at different times for similar offers). We compared (between participants) regions identified as high and low conflict (see above) using each participant's average consistency score across all bins in a region. An additional indication of high conflict is lengthier RT which we defined as the time between the onset of the offer and the registration of a response. We accordingly compared (between participants) trials identified as high and low conflict, comparing participants' region specific median RT.

*Computational modeling framework*

*Overview.* Our computational modeling aimed to assess whether states of high conflict shaped parameters associated with choice behavior, and whether key parameters were additionally associated with trial-by-trial fluctuations in neural signals, a cardiac-sympathetic signal or the alignment (interaction) of neural and cardiac signals.

*HDDM.* We decomposed behavior using a Hierarchical-Bayesian extension of the drift-diffusion model (DDM; Figure 1D). The DDM classically proposes that choice and RT data are underscored by a noisy evidence accumulation process that terminates at a decision criterion (boundary). Following an initial nondecision time (t), the decision process begins at starting point (z) and accumulates evidence at rate (v) toward one of two boundaries that determines the choice (in our case, approach (+) or avoid (-)); boundaries are separated by distance (a). These parameters provide a fine-grained assay of behavior, such as likely bias toward one choice (z), how rapidly evidence is integrated during decision formation (v) or the amount of evidence required before a choice is executed (a wider boundary denoting a more conservative criterion). With the

Hierarchical-Bayesian extension (HDDM; Wiecki et al., 2013), the choice and RT data of each trial ($y_t$) form a distribution described by a Wiener diffusion likelihood function (Navarro and Fuss, 2009), parameterized by {a,v,t,z}. These parameter posteriors can be sampled in a static fashion using Bayes Monte Carlo, i.e.:

$$y_k \sim \text{Wiener}(a,v,t,z) \quad \text{[Eq. 2]}$$

A wealth of existing literature has tested hypotheses by additionally fitting the DDM parameters separately for different task conditions (e.g. Ratcliff and Frank, 2012; Wiecki et al., 2013; Bottemanne and Dreher, 2019; reviews in O'Connell et al., 2018; Gupta et al., 2022). Under the above probabilistic framework, parameters {a,v,t,z} can be fitted as a mixture model, to account for different states (s). In such a case, the model assigns $y_k$ to one of two Wiener distributions, depending on trial k's state of conflict (s), i.e.:

$$y_k|(s_k=s) \sim \text{Wiener}(a_s,v_s,t_s,z_s) \quad \text{[Eq. 3]}$$

where $s \in \{0,1\}$, i.e., low or high conflict. Evaluating this mixture extension allows probabilistic inference that DDM parameters are credibly different depending on the state of conflict. This can be evaluated using model fits and/or by testing whether the highest density interval (HDI) of the

group-level posterior for a parameter in low conflict (s=0) minus the posterior for that parameter in high conflict (s=1) does not subtend 0, i.e., $0 \notin HDI([p(P_0|y_0)] - [p(P_1|y_1)])$, where $P \in \{a,v,z,t\}$.

Under the probabilistic framework, parameters {a,v,t,z} can additionally be modeled as a linear combination of continuous predictors, such as trial-by-trial estimates of a neural (e.g., Frank et al., 2015) or cardiac-sympathetic signal, i.e.:

$$y_k \sim \text{Wiener}(a,v,t,z), \text{ where:}$$

$$a = b_{0,a} + b_{1,a} * X_1 , ..., b_{n,a} * X_n$$

$$v = b_{0,v} + b_{1,v} * X_1 , ..., b_{n,v} * X_n$$

$$t = b_{0,t} + b_{1,t} * X_1 , ..., b_{n,t} * X_n$$

$$z = b_{0,z} + b_{1,z} * X_1 , ..., b_{n,z} * X_n$$

[Eq. 4]

Here, $X_1 , ..., X_n$ are vectors of trial-by-trial physiology signals and $b_{1,P} , ..., b_{n,P}$ are their coefficients (for each $P \in \{a,v,t,z\}$). Evaluating this regression extension (either using model fits or by testing if the HDI of group-level posteriors $b_{1,P} , ..., b_{n,P}$ do not contain 0) allows probabilistic inference that DDM parameters are associated with moment-to-moment physiological fluctuations.

Finally, the models described in Eqs. 3 and 4 can be merged into a mixed-regression model, to allow inference about both state-specific parameter estimates and state-specific associations between moment-to-moment physiology and parameters, i.e.:

$$y_k|(s_k=s) \sim \text{Wiener}(a_s, v_s, t_s, z_s), \text{ where:}$$

$$a_s = b_{0,a,s} + b_{1,a,s} * X_{1,s} ,..., b_{n,a,s} * X_{n,s}$$

$$v_s = b_{0,v,s} + b_{1,v,s} * X_{1,s} ,..., b_{n,v,s} * X_{n,s}$$

$$t_s = b_{0,t,s} + b_{1,t,s} * X_{1,s} ,..., b_{n,t,s} * X_{n,s}$$

$$z_s = b_{0,z,s} + b_{1,z,s} * X_{1,s} ,..., b_{n,z,s} * X_{n,s}$$

[Eq. 5]

Here $X_{1,s}$ ,..., $X_{n,s}$ are vectors of trial-by-trial physiology signals in state s. Evaluating this mixture-regression extension allows the inferences of Eqs. 3 and 4. In addition, this model allows probabilistic inference about whether the association between DDM parameters and physiological fluctuations depends on the state of conflict.

In our analyses, we used an iterative approach to narrow down the best combination of physiology variables associated with state-specific parameters of the DDM. We first fitted a "baseline" model. This was the mixture model described in Eq. 3 and it both served as a baseline comparison for later physiology models and probed the static parametric differences between high and low conflict (Figure 2D). We then fitted a series of mixture-regression models using the formula in Eq. 5. These models tested if additionally modeling the state-specific parameters as a linear

combination of a state-specific physiology signal would provide a better-fitting model than the baseline. These "singular" models (Figure 3D) contained a single regressor, i.e.:

$$P_s=b_{0,P,s}+b_{1,P,s}*X_{1,s} \text{ for each } P\in\{a,v,z,t\}$$

[Eq. 6]

where $s\in\{0,1\}$, i.e., low or high conflict, and the single regressor ($X_{1,s}$) was one of the neural variables, or the cardiac-sympathetic variable, described below. We used model fits (Deviance Inference Criterion (DIC); Wiecki et al., 2013) to determine if these singular models were a better fit to the data than the baseline model.

We pre-empt some results here to aid describing the next stage of modeling, i.e., that a number of singular models were superior fits to baseline, including the singular model containing the cardiac-sympathetic assay. We next fitted a series of "cross-modal" mixture-regression models (Figure 3E). These models tested whether the best singular model fit could be improved by extending it to two regressors. In each of these models, one regressor was the cardiac-sympathetic assay and the other regressor was a neural variable (i.e., cross-modal). We restricted the addition of neural variables to only those that had featured in singular models that were superior fits to baseline. A two-regressor "additive" cross-modal model was therefore:

$$P_s=b_{0,P,s}+b_{1,P,s}*X_{1,s}+b_{2,P,s}*X_{2,s} \text{ for each } P\in\{a,v,z,t\}$$

[Eq. 7]

Here s∈{0,1}, i.e., low or high conflict, the first regressor ($X_{1,s}$) was the cardiac-sympathetic assay and the second regressor ($X_{2,s}$) was a neural variable from singular models outperforming baseline. An additional three-regressor "interactive" cross-modal model was:

$$P_s=b_{0,P,s}+b_{1,P,s}*X_{1,s}+b_{2,P,s}*X_{2,s}+b_{3,P,s}*X_{3,s} \text{ for each } P\in\{a,v,z,t\}$$

[Eq. 8]

Here all parameters were the same as in Eq. 7, except now a third regressor ($X_{3,s}$) contained the z-score-normalized dot product of $X_{1,s}$ and $X_{2,s}$, i.e., capturing the correlation over trials between the neural and cardiac variable. In other words, the interactive model additionally allowed inference about the alignment of neural and cardiac signals being associated with DDM parameters. We used model fits DIC scores to determine if any additive (Eq. 7) or interactive (Eq. 8) models were a better fit to the data than the best-fitting singular model (Eq. 6).

We pre-empt some additional results here to aid describing the next stage of modeling, i.e., that a number of cross-modal models were superior fits to the best-fitting singular model. We next fitted a final series of mixture-regression models (Figure 3F). These "complement" models now tested whether the best-fitting cross-modal model could be improved by extending it to incorporate additional neural regressors and regressors of neural-cardiac alignment. The design matrix of these models started with the regressors from the best-fitting cross-modal model, i.e., a neural variable, the cardiac-sympathetic variable and, if applicable, the interaction term. We then tested if the fit could be improved by also including complement (i.e., set difference) regressors from

other cross-modal models. In other words, we combined cross-modal design matrices, removing redundant regressors. We tested complement models by merging the best-fitting cross-modal model with the set difference of the cross-modal models involving all neural variables that passed the singular model stage (i.e., early_attn_rew, late_the, early_SS_sym, late_alpha_sym, late_alpha_rew), using their best-performing cross-modal forms (i.e., whether or not they included interactions). Each "complement" model was therefore:

$$P_s = B^*[X'_{P,s} | X''_{P,s} \setminus X'_{P,s}] \text{ for each } P \in \{a,v,z,t\} \quad \text{[Eq. 9]}$$

where $s \in \{0,1\}$, i.e., low or high conflict, X' is the design matrix of the best-fitting cross-modal model, X'' is the design matrix of an additional cross-modal model (provided it was a superior fit to the best singular model) and $X'' \setminus X'$ describes the set-difference, i.e., removal of any common columns between them. B is a coefficient vector of length corresponding to the resulting concatenated design matrix. We used model fits DIC scores to determine if any complement models (Eq. 9) were a better fit to the data than the best-fitting cross-modal models (Eqs. 7–8).

*Model to discretize neural-cardiac interactions.* We pre-empt some additional results here to aid describing the next stage of modeling, i.e., that one complement model was a superior fit to the best-fitting cross-modal model. This model featured an association between the decision boundary and a dot-product regressor described in the "interactive" model in Eq. 8 (specifically contractility·late $alpha_{sym}$). This association was additionally unique to states of high conflict. To help clarify the underlying dynamics of this seeming three-way interaction, we fitted a model that

discretized these two continuous regressors (high and low contractility and high and low late $alpha_{sym}$) within each participant, and fitted a decision boundary separately for the resulting combination of physiological states, separately again for low and high conflict, creating eight states in total. This model was the same as the baseline model in Eq. 3 but with a different parameter mixture for the decision boundary, i.e.:

$$y_k|(s_k=s,d_k=d)\sim Wiener(a_d,v_s,t_s,z_s) \quad \text{[Eq. 10]}$$

where $s\in\{0,1\}$, i.e., low or high conflict, and $d\in\{000,001,010,011,100,101,110,111\}$, where the digits in each of the eight binary code sequences respectively describe the conflict (low (0) or high (1)), late $alpha_{sym}$ (low (0) or high (1)) and contractility (low (0) or high (1)) states of trial k. The latter two levels were classified using median splits within participants. In this model, we report parameter estimates $a_d$ as a difference measure (Δ boundary) from when d=000.

*Control models for local and global brain activity.* To assess whether cardiac-sympathetics might be a proxy for more general activity levels in the brain (for example, a global or frequency-specific increase in gain), we performed two additional control models for our best-fitting complement model. In each case, we substituted an alternative measure of brain activity for the contractility regressor and the contractility component of any dot-product (interaction) regressors featuring contractility. In the first, we substituted contractility with an estimate of global field power (GFP; Skrandies, 1990), which we computed as the standard deviation across all electrodes in the montage, with data bandpass-filtered from 1 Hz to 40 Hz. In the second, we substituted

contractility with a signal more local to the relevant frequency band in our findings (alpha). For this, we used an estimate of the average alpha power across all electrodes in the montage, with data preprocessed in the same way as our assays in this frequency band (see below). For a single trial-by-trial estimate in each case, we averaged both the GFP measure (using the root-mean-square average) and the global alpha power measure across timepoints in the time window [0 s to 1 s] post offer onset.

In all HDDM models, we sampled both individual and group-level parameters in a hierarchical fashion and report group-level findings. We sampled posteriors 5000 times with Markov-Chain Monte Carlo, using the HDDMRegressor function from the HDDM toolbox (Wiecki et al., 2013) version 0.6.0 in Python 2.7, using default settings for hyper-parameters. We discarded the first 500 samples of each posterior estimate as tuning steps. A single drift rate was fitted using a link function that made it negative on avoid trials and positive on approach trials.

*Alternative model assessment.* For the baseline model, and for the best-fitting singular, cross-modal, and complement models, we additionally report a proxy of RT variance explained by each model using a bin-by-bin regression procedure. This procedure first simulated trial-by-trial RTs using posterior medians of parameters of the models (linearly estimated where relevant using model coefficients and trial-by-trial regressors) with a Wiener-like process. For approach trials, the simulated decision process (x) initiated at time (RT=0) at a starting point in units of the boundary, i.e., $x(RT=0)=z \cdot a$. As RT increased in units of 0.01, x increased with $x=x+0.01v$ until the boundary was reached, i.e., $x>a$. For avoid trials, the process was the same except with v inverted and the process continuing until $x<0$. Finally, nondecision time (t) was added to the resulting RT to arrive at the final simulated value. We next binned observed RTs and correlated

bin-by-bin medians with medians derived from corresponding simulated RTs. We report variance explained from Pearson correlations ($R^2$) for this procedure separately using 10, 15, 20, 25, 30, 35, and 40 bins of RT in the correlation.

*EEG neural recordings - Recording, preprocessing and assays*

*Recording.* Concurrent with the approach-avoidance paradigm, we recorded continuous electroencephalogram (EEG) data from a montage of 63 scalp electrodes (channels) arranged using the International 10-20 system. We sampled the EEG signal at 1000 Hz from each channel, using a BrainAmp MR amplifier (Brain Products, Berlin, Germany). Channel FCz served as the online reference while channel Cz served as the ground. Between blocks, experimenters paused recordings to check electrode impedance (<5 kΩ) and noisy channels.

*EEG preprocessing* used functions available in the EEGLAB toolbox (Delorme and Makeig, 2004). First, each participant's EEG data were downsampled (250 Hz) and hi-pass filtered (<1 Hz) separately for each block. Line noise was removed with an automated function (Mullen, 2012). We merged resulting sets of blockwise data into a single set (one set per subject) and identified noisy channels using an automated function that tested whether data in each channel correlated with those in surrounding channels by a coefficient of at least 0.85 (Kothe and Makeig, 2013). Identified channels were replaced using spherical interpolation. We then re-referenced datasets to the montage average, created epochs spanning from -1 s to +6 s relative to offer onset, and subtracted baseline means (taken from the window -.5 s to 0 s relative to offer onset). We then performed ICA decomposition separately on each subject's resulting epochs, and stored the resulting weights of components that were 95% likely to be ocular or cardiac activity, determined by an automated classifier (Pion-Tonachini et al., 2019). We next imported, downsampled, hi-

pass-filtered and removed line noise from participants' separate blocks of raw data again, as above. Separately for each block, we replaced noisy channels as above and removed ICA components related to ocular and cardiac artefacts. We marked any data point where any channel still exceeded 150 mV (for later rejection) and applied a spatial Laplacian filter across multichannel data at each time point. We then reversed the laterality of electrodes on all trials where reward appeared on the left of the screen, so that each trial "de facto" presented reward on the right and cost on the left. We refer to data at this stage as "preprocessed" data.

*Assay of sensory gain.* To extract timeseries from preprocessed data for steady-state visually evoked potentials relevant for reward ($SS_{rew}$) and cost ($SS_{shk}$) information (Figure 3A), we used rectified and smoothed power timeseries that had been filtered to either 12 or 13.33 Hz, depending on the flicker of reward or cost information for a given trial (note that no specific frequency mapped onto either reward or cost; flickers varied trial-by-trial). We convolved each channel's fast-Fourier-transformed data with trapezoid-shaped bandpass filters ("on" width = .5 Hz, transition bandwidth = 0.25 Hz), centered on 12 Hz or 13.33 Hz before rectifying, smoothing (mean within sliding windows spanning 66 ms) and downsampling inverse-Fourier timeseries to 125 Hz. We also constructed a third dataset, using these exact procedures, but with filters centered on 12.66 Hz (midway between 12 and 13.33 Hz), and subtracted it from $SS_{rew}$ and cost $SS_{shk}$ timeseries to mitigate SS influence from underlying activity in the alpha band. We created epochs spanning from -1 s to +6 s relative to offer onset for the 12 and 13.33 Hz datasets, removing the baseline average value in the 500 ms window prior to offer onset. For trial-by-trial measures, we computed the average power in early ([0 s to 1 s] post offer onset) and late ([1 s to 2 s]) time windows (Figure 3A). Amplitudes were averaged at electrode sites contralateral to the relevant information, i.e., O1 and PO1, or at electrodes O2 and PO2, depending on reward and cost laterality on a given trial (Figure 3A).

*Assay of goal-directed attention.* We also extracted timeseries from preprocessed data of spatially sensitive alpha power, i.e., relevant for reward ($alpha_{rew}$) and cost ($alpha_{shk}$) information (Figure 3B). We first applied notch filters to remove power in SS frequencies (inverse of bandpass filters above), and then convolved each channel's fast-Fourier-transformed data with a trapezoid-shaped bandpass filter ("on" segment spanning 7 Hz to 14 Hz, transition bandwidth = 0.5 Hz). Resulting inverse-Fourier timeseries were rectified, smoothed (mean within sliding windows spanning 66 ms) and we downsampled channels to 125 Hz. We created epochs spanning from -1 s to +6 s relative to offer onset, removing the baseline average value in the 500 ms window prior to offer onset. For trial-by-trial measures, we computed the average power in early ([0 s to 1 s] post offer onset) and late ([1 s to 2 s]) time windows (Figure 3B). Amplitudes were averaged at electrode sites contralateral to the relevant information, i.e., PO and PO7, or at electrodes PO2 and PO8, depending on reward and cost laterality on a given trial (Figure 3B).

*Symmetry timeseries*. In our approach-avoidance paradigm, conflict peaks when an offer's value makes approaching it as appealing as avoiding it, measured as the absolute distance from a decision boundary (SV=0 or p(approach)=0.50; Figure 1). For both the SS and alpha timeseries we also computed a neural proxy of conflict to include in our models, by way of a "symmetry" timeseries, at each timepoint (t); i..e, $SS(t)_{sym}=-1*\log(|SS(t)_{rew}-SS(t)_{rew}|)$; $alpha(t)_{sym}=-1*\log|alpha(t)_{rew}-alpha(t)_{shk}|$). Given the inversion, higher values reflect higher symmetry, or in other words, that more equal power was present in the traces relevant for reward and cost (Figure 3A–B). For trial-by-trial measures, we computed averages in the same manner (early and late windows) as the single traces. To verify that these symmetry metrics tracked conflict, we performed repeated-measures ANOVAs testing whether each participant's symmetry trace was

605 modulated by the state of conflict (low or high), the phase of trials (early vs late), or their
606 interaction.

607

608 *Assays of cognitive control and decision making.* The assays of neural activity described above
609 exploit the modifications (graded onset, frequency-tagged, spatially mapped stimuli) we made to
610 the approach-avoidance paradigm and allow comparison and contrast between early and late
611 perceptual processes during decision making. However, to perform a more complete analysis of
612 neural-cardiac-behavioral relationships, we additionally extracted neural signals traditionally
613 associated with other components that are likely relevant during the approach-avoidance conflict.
614 The first was delta power over posterior parietal sites (Figure 3C). Activity in this frequency range
615 has been linked with DDM-like mechanisms of decision making in perceptual contexts (O'Connell
616 et al., 2012; Harper et al., 2014). For trial-by-trial measures, we computed the average power
617 between (1 and 4 Hz) in early ([0 s to 1 s] post offer onset) and late ([1 s to 2 s]) time windows at
618 electrode sites (CPz and Cz). The other signal we extracted was frontal-midline theta (Figure 3C),
619 classically considered an assay of cognitive control and action regulation (Luu et al., 2004;
620 McLoughlin et al., 2014). For trial-by-trial measures, we computed the average power between (4
621 and 7 Hz) in early ([0 s to 1 s] post offer onset) and late ([1 s to 2 s]) time windows at electrode
622 sites (Fz, F1 and F2).

623

624 *Alpha-phase coherence.* In a final neural-cardiac analysis (as a follow-up on later-reported
625 results), we probed how the state of cardiac-sympathetics (specifically, whether contractility was
626 high or low) was related to the coherence of alpha power across trials, relevant to both the reward
627 and cost information. We re-processed the alpha-band activity contralateral to the reward and
628 cost information, extracting the phase angle of these waveforms over time, i.e., $alpha_{rew}\theta$ and

629 $alpha_{shk}\theta$. If specific events (such as the onset of an offer) evoked temporally consistent
630 fluctuations in alpha power, phase angles would summarize across trials to a coherent sinusoid-
631 like waveform oscillating in and around the alpha-band frequency (7–14 Hz). We averaged across
632 trials for each participant's $alpha_{rew}\theta$ and $alpha_{shk}\theta$ timeseries, separately for trials in states of high
633 and low conflict and separately again for trials in states of high and low contractility (determined
634 by a median split across all of a participant's trials). To compute a summary estimate of early and
635 late coherence across trials, we rectified the resulting participant-average phase waveforms $|\theta|$,
636 and computed the average values across datapoints in early ([0 s to 1 s] post offer onset) and
637 late ([1 s to 2 s]) time windows (Figure 5A).

638

639 *Cardiac-sympathetic recordings - Recording, preprocessing and contractility assay*

640 *Recording.* Concurrent with the approach-avoidance paradigm we also recorded data from
641 combined electrocardiogram (EKG) and impedance cardiogram (ICG) using a total of ten EL500
642 electrodes (BIOPAC, USA). Prior to recording, in a private room, a trained female researcher
643 disinfected the skin at the electrode sites. They gently exfoliated the skin with an abrasive pad
644 (ELPAD, BIOPAC, Inc.), applied NuPrep skin exfoliating gel (ELPREP, BIOPAC, Inc.) to each
645 electrode site (~1-by-1 inch area of skin) and fanned the sites dry. EKG was recorded from one
646 electrode beneath the right collarbone and one beneath the left rib cage. ICG was recorded from
647 eight electrodes: two on each side of the torso and two on each side of the neck. ICG electrodes
648 served as the ground for EKG. All electrodes had a small dab of electrode gel (GEL100, BIOPAC,
649 Inc.). The upper neck and lower torso (outside) electrodes injected a 4mA alternating current into
650 the thoracic cavity at 50 kHz, while the lower neck and upper torso (inner) electrodes were
651 voltage-sensing. We sampled both the EKG and ICG signals at 5000 Hz via carbon fiber leads
652 connected respectively to ECG100C and NICO100C amplifiers, integrated with an MP150 system

(BIOPAC, Inc.). Online, AcqKnowledge software version 4.3 differentiated the raw basal transthoracic impedance (z) ICG data with respect to time (dz/dt) and removed respiratory artifact from the ensuing dz/dt waveform with a high-pass filter (BIOPAC, Inc.). Once seated in the testing suite, we instructed participants to minimize unnecessary movement and vocal sounds to limit disruptions to the physiology signal. Participants completed a nonrecorded resting period to acclimate to the study environment. Between blocks, experimenters paused recordings to check for noise in the EKG and ICG data.

*EKG/ICG preprocessing and contractility assay*. We estimated contractility from the pre-ejection period (PEP). We used a semi-automated software package (MEAP; Cieslak et al., 2018), which uses moving ensemble averages (15-second windows) to help identify the R point of the EKG QRS complex (early systole: initial left-ventricular depolarization) and the B point of the dz/dt waveform from the ICG (mid systole: opening of the aortic valve), for each individual heartbeat (Figure 3D); all heartbeats were manually checked for correct point classification. The time period between these two cardiac events is the pre-ejection period (PEP). This electro-mechanical time interval, covering systolic activity from the initial electrical depolarization of the left ventricle until the opening of the aortic valve, is an index of beta-adrenergic contraction vigor, and is primarily mediated by sympathetic activity (Lewis et al., 1974; Light, 1985; Linden, 1985; Newlin and Levenson, 1979; Sherwood et al., 1986; 1990). Shorter intervals reflect increased contractility (positive inotropy). For trial-by-trial measures, we computed the average PEP value across all heartbeats occurring in the two-second window immediately following offer onset (Figure 3D). These values were log-transformed and then reverse-scored, so that higher values reflect higher contractility.

## Results

We recorded continuous multi-channel electroencephalography and cardiac-sympathetic physiology while 27 human participants performed an approach-avoidance task, trading off monetary reward for electric shock cost (see trial schematic in Figure 2A). On average, participants accepted 68% (σ=12.6%) of offers, and responded with median RT of 1.73 seconds (σ=0.428) relative to offer onset.

### *Behavioral results*

*Logistic choice models.* From two-dimensional logistic models fitted separately to each individual subject's set of choices, modeling p(approach) as a function of an intercept ($b_0$) and the magnitude of monetary reward ($b_1$) and shock cost ($b_2$) offered on each trial, these two continuous value dimensions respectively increased (group-mean $b_1$=6.05; t(26)=9.55; p<0.001, Figure 2B) and decreased (group-mean $b_2$=-4.76; t(26)=-7.65; p<0.001, Figure 2B) the log odds of approach. In other words, participants integrated both reward and a cost into their choices. However these parameters also confirmed that participants were reward sensitive, characterized by a bias toward approach (group-mean $b_0$=4.15; t(26)=6.08; p<0.001, Figure 2B), and an overweighting of reward in the integration of value dimensions (group-mean $|b_1|-|b_2|$=0.987; t(26)=3.59; p<0.001, Figure 2B), consistent with previous studies (Shapiro and Grafton, 2020; Dundon et al., 2021; Pedersen et al., 2021).

[Figure 2 here]

From these logistic models we classified each trial as offering either high or low conflict (see methods). We confirmed that these classifications gave rise to the typical behavioral features of encountering conflict: less consistent choice and lengthier RT. First, we observed in a nonparametric estimate of choice consistency across participants, that there was a higher level of deviation in choices (i.e., different choices registered at different times for similar offers, enumerated with a bin-by-bin variance estimate (V); see methods) in trials identified as high conflict ($V_{choice,high}$=0.071) compared to trials identified as low conflict ($V_{choice,high}$ =0; t(26)=13.23, p<0.001; Figure 2C). Next, we observed across participants in a comparison of median RT, that responses were longer on trials identified as high conflict ($RT_{high}$=2.02 s) compared to trials identified as low conflict ($RT_{low}$=1.66 s; t(26)=12.10, p<0.001; Figure 2C).

*Baseline computational model.* We next fitted a baseline hierarchical Bayesian drift-diffusion model (HDDM; Wiecki et al., 2013), to get a clearer insight into how increased conflict alters the computational parameters associated with choice and RT. This model (described in Eq. 6) fitted distinct group-level DDM parameters {a,v,z,t}, depending on whether participants were making choices on trials in states of low or high conflict. This baseline model revealed that in high conflict, participants displayed a wider decision boundary (a), consistent with seeking more evidence before executing their choices (Figure 2D; Table 2-1). The model also revealed that participants reached this decision boundary by way of a dampened rate of evidence accumulation ((v); Figure 2D; Table 2-1). Starting points (z) in this DDM also suggested an overall bias toward approach in states of both low and high conflict, however this bias was attenuated in high conflict (Figure 2D; Table 2-1). Finally, nondecision time (t) was slightly shorter in high conflict (Figure 2D; Table 2-1).

Summarizing the behavioral and baseline computational results so far, participants were reward sensitive, but also confronted states of subjective "conflict". The parametric-behavioral response to states of high conflict appeared to involve a larger requirement of evidence prior to committing choices, a slower accumulation of evidence toward that criterion and an attenuated bias toward approach behavior.

*Verifying neural symmetry's association with conflict*. Two within-subjects ANOVAs verified that the $SS_{sym}$ ($F(1,26)=8.29$, $p=0.008$) and $alpha_{sym}$ ($F(1,26)=5.82$, $p=0.023$) "symmetry" traces varied with conflict. For alpha, there was also an interaction with trial phase ($F(1,26)=11.9$, $p=0.002$): no significant difference between conflict states early (mean difference=-0.008; SE=0.012; $p_{Tukey}=0.906$), but higher symmetry in high conflict late (mean difference=-0.043; SE=0.012, $p_{Tukey}=0.004$).

*Cross-modal (neural and cardiac-sympathetic) collaborative association with (DDM) parameters*

We next tested if DDM parameters were associated with trial-by-trial physiological fluctuations, and if this association was unique to a specific state (i.e., low vs high conflict). Specifically, we used an iterative modeling approach to find a best-fitting combination of physiological signals associated with DDM parameters, with an emphasis on discovering cross-modal (i.e., neural and cardiac) collaboration. We began this process with a total 17 candidate physiology signals. Neural signals were: steady-state visually evoked potentials relevant for reward ($SS_{rew}$) and cost ($SS_{shk}$) information, in addition to the "symmetry" trace ($SS_{sym}$) enumerating the similarity between these

traces (i.e., the symmetry across the brain) over time (Figure 3A); spatially sensitive alpha power, i.e., relevant for reward ($alpha_{rew}$) and cost ($alpha_{shk}$) information, in addition to a "symmetry" trace ($alpha_{sym}$) enumerating their similarity over time (Figure 3B); frontal-midline theta power (theta) and posterior-parietal delta (delta) power (Figure 3C). For each neural variable, we computed average power in early ([0 s to 1 s] post offer onset) and late ([1 s to 2 s]) time windows, making a total of 16 neural variables for each trial (Figure 3A–C). The 17th physiology signal was an estimate of cardiac contractility (inotropy; Figure 3D). We averaged across a positively scored contractility estimate for each heartbeat registered in the two-second window post offer onset (Figure 3D).

*Singular models.* For the iterative modeling approach we first fitted a series of "singular" models (Eq. 6) that probed whether modeling DDM parameters {a,v,z,t} as a linear combination of a single regressor (i.e., one trial-by-trial physiology signal) would provide a better-fitting model than the baseline model described above. Using Deviance Information Criterion scores (DIC; Wiecki et al., 2013) we observed that seven models provided an improved fit (Figure 3E); these models respectively modeled the DDM parameters as a function of trial-by-trial fluctuations in late $alpha_{sym}$ (-ΔDIC=141.3), late $alpha_{shk}$ (-ΔDIC=129. 8), late $alpha_{rew}$ (-ΔDIC=116.45), contractility (-ΔDIC=39.6), early $SS_{sym}$ (-ΔDIC=8.21), late theta (-ΔDIC=5.54) and early $alpha_{rew}$ (-ΔDIC=2.31). A proxy for RT variance explained for the best fitting singular model is in Figure 3H. We therefore established first that behavioral decompositions from the DDM were associated with both neural and cardiac-sympathetic physiological signals that varied on a trial-by-trial basis, with neural signals predominantly involving the alpha band, and signals measured in the later time window.

[Figure 3 here]

*Cross-modal models.* Singular models revealed associations between DDM parameters and both neural and cardiac-sympathetic signals. We next fitted a series of "cross-modal" models (Eqs. 7–8). These models tested if the singular models containing a neural regressor could be improved by adding contractility as a second regressor (i.e., cross-modal). We restricted cross-modal models to include neural variables from the singular models that were superior fits to baseline. We also tested for evidence of both additive and interactive cross-modal collaboration. For additive collaboration, we tested models with two regressors (the neural variable in question, and contractility), while for interactive collaboration, we added a third interaction or "alignment" regressor which was the normalized product of the neural variable in question and contractility. This made 12 target cross-modal models in total (Figure 3F). We also updated the baseline, and tested if these cross-modal models improved the fit relative to the best-fitting singular model, i.e., which had $alpha_{sym}$ as its sole regressor (dashed line from Figure 3E to 3F). From resulting differences in DIC scores, we observed six cross-modal models providing an improved fit over baseline. All such models involved alpha-band activity recorded in the late window (Figure 3F). The best-fitting model ($-\Delta DIC=29.0$) was additive; it contained $alpha_{shk}$, that is, alpha power relevant for the cost information, alongside contractility, and no third alignment regressor. A proxy for RT variance explained for the best fitting cross-modal model is in Figure 3H

*Complement models.* Our cross-modal models revealed that behavioral features could be modeled by a neural variable relative to cost ($alpha_{shk}$) in addition to cardiac-sympathetic fluctuations (contractility), i.e., side-by-side in the same model. However, given that cross-modal models involving alpha power relevant to reward information ($alpha_{rew}$) and the symmetry of alpha across the brain ($alpha_{sym}$) also provided improved fits (Figure 3F), we ran a final set of

"complement" models to test for their complementary association with DDM parameters (Eq. 9). In other words, we tested if the outright best cross-modal fit (additive $alpha_{shk}$, marked "m1" in Figure 3E) could be improved even further by also including complement (i.e., set difference) parameters from either of the two best-fitting cross-modal models involving $alpha_{rew}$ and $alpha_{sym}$ (each of which was interactive; each marked "m2" in Figure 3F). We again updated the baseline to the best-fitting cross-modal model (dashed line from Figure 3F to 3G). From resulting differences in DIC scores (Figure 3G), we observed both complement models to improve the fit, with substantial improvement in the case of adding set difference parameters involving the interactive cross-modal model with $alpha_{sym.}$ (-ΔDIC=155.3). In other words, the best-fitting complement model modeled DDM parameters not just by $alpha_{shk}$ and contractility, but also by the symmetry of alpha across the brain ($alpha_{sym}$) and the product of $alpha_{sym}$ and contractility. A proxy for RT variance explained for the best fitting complement model is in Figure 3H. Our iterative modeling approach therefore unearthed a set of both neural (exclusively alpha) and cardiac-sympathetic physiological signals associated with parameters of the DDM, and further revealed evidence for interactive cross-modal collaboration (i.e., neural and cardiac-sympathetic alignment).

Inspecting the parameter posteriors of this best-fitting complement model (Figure 4A–D), the majority of associations were with the decision boundary (a). Wider boundaries were accompanied by a fairer spread of attention to all dimensions of available information, though this was not exclusive to states of high conflict. In both low and high-conflict states, there was a negative association between the boundary and alpha relevant to cost ($alpha_{shk}$; Figure 4A; Table 4-1), suggesting greater desynchronization of alpha contralateral to cost information associated with increased boundaries (consistent with more attention being allocated to that information ). Also in both conflict states, there were positive associations between the boundary and the

symmetry of alpha on either side of the brain ($alpha_{sym}$; Figure 4A; Table 4-1). Widening boundaries were therefore not solely associated with deploying attention to cost, but also a more even spread of attention to both channels of information, consistent with a pursuit of greater evidence, but in an additive, i.e., not overriding manner. The relationship between the boundary and cardiac contractility was likewise observed in both states. This (positive) association was consistent with wider boundaries also accompanying increased sympathetic drive (contractility; Figure 4A; Table 4-1). Exclusive to states of high conflict, we identified the interactive aspect of cross-modal collaboration, i.e., neural and cardiac-sympathetic signals aligning with meaningful measures of behavior. That is, in addition to its linear relations with $alpha_{sym}$ and contractility, the boundary (as determined through the DDM) was additionally positively associated with their alignment. Strikingly, this association only occurred in states of high conflict ($alpha_{sym}$*cont.; Figure 4A; Table 4-1). In other words, as participants made choices in high conflict, which was linked with wider decision boundaries, part of this boundary widening was directly related to alignment between a cardiac-sympathetic (positive inotropic) response and the degree of alpha symmetry across the brain. This was the sole evidence of such interactive cross-modal collaboration in our best-fitting model.

[Figure 4 here]

We observed more sparse associations between physiological signals and the remaining DDM parameters. Drift rate (v) was not credibly associated with any signal (Figure 4B; Table 4-1). The starting point (z) was related to alpha related to cost, only in states of high conflict ($alpha_{shk}$; Figure 4C; Table 4-1). This positive association is consistent with a bias toward approach intensifying when less attention is directed at the cost information (i.e., more synchronization of $alpha_{shk}$).

Finally, state-specific associations emerged relating to nondecision time (t). Nondecision time is a constant term included in the DDM to account for early perceptual and motor preparation processes. In low conflict, nondecision time was longer when alpha symmetry decreased, and when alpha relevant to cost increased synchrony ($alpha_{shk}$ and $alpha_{sym}$; Figure 4D; Table 4-1). Conversely, in high conflict, nondecision time was longer solely when $alpha_{sym}$ increased (Figure 4D; Table 4-1).

To help clarify the underlying dynamics of the seeming three-way interaction and the decision boundary (i.e., associations between the decision boundary and combinations of conflict, $alpha_{sym}$ and contractility) we fitted a modified version of the baseline model in Eq. 3. This model discretized trials into eight bins depending on whether these three measures were high or low, and fitted a separate decision boundary for each. Figure 4E and Table 4-2 respectively depict and characterize the resulting parameter posteriors, expressed relative to a baseline (low conflict, low late $alpha_{sym}$ and low contractility). We here see that the decision boundary is credibly widest when both contractility and the symmetry of alpha across the brain is high. This is consistent with the interaction in Figure 4A being driven by the two signals synchronously increasing (i.e., higher contractility alongside higher alpha symmetry) during moments of conflict.

We additionally fitted control models to assess whether cardiac-sympathetics might be a proxy for more general activity levels in the brain, i.e., a global or frequency-specific increase in gain (Figure 4F). In these models, we used the best-fitting complement model, substituting contractility regressors (and interaction regressors featuring contractility) with an estimate of global field power (GFP; Figure 4F) and total alpha power in the brain (whole brain alpha; Figure 4F). Only the model substituting contractility with global field power (GFP) was a slightly better fit (-ΔDIC=5.78).

However, inspecting parameters posteriors for the decision boundary in this control model alongside the contractility complement model (right panels of Figure 4F; Table 4-2) we observed a striking dissociation between the direction of associations. While the boundary is widest for high levels of contractility and $alpha_{sym}$, alignment of GFP and late $alpha_{sym}$ was instead associated with smaller boundaries (parameters marked with arrows in figure 4F). This strengthens the case that contractility might be part of a physiological response to scrutinize evidence in a conflicting situation, in contrast to other brain signals that might instead be associated with an urgency to respond more quickly.

*Inter-trial alpha-phase coherence.* In the above section we observed that alpha dynamics and their alignment with cardiac-sympathetic signals were associated with behavioral parameters during the approach-avoidance conflict. That is, during high-conflict choices, the width of the decision boundary was positively associated with the alignment between contractility and alpha symmetry. We lastly sought additional evidence (outside of computational models) regarding the nature and characteristics of the relationship between contractility and alpha dynamics. For this, we probed how the state of cardiac-sympathetics (specifically, whether contractility was high or low) was associated with coherence of alpha power across trials, separately for alpha power relevant to both the reward and cost information, and separately again for high and low-conflict trials. We extracted the phase angle of these waveforms over time, i.e., $alpha_{rew}\theta$ and $alpha_{shk}\theta$. Figure 4G depicts $alpha_{rew}\theta$ and $alpha_{shk}\theta$ summarized across trials and participants, separately for trials that were above (high contractility) or below (low contractility) a participant's median. Note that the waveforms in Figure 4 only show trials in states of high conflict. Here we observe alpha-like oscillations present in each time series, indicating coherence across trials. However, during the later time window, the sinusoidal patterning in $alpha_{rew}\theta$ (Figure 4G top panel) appears to be greater on low-contractility trials. A three-way within-subjects ANOVA of summarized

rectified phase angles |θ| as a function of the timeseries ($alpha_{rew}θ$, $alpha_{shk}θ$), time window (early, late) and contractility (high, low) confirmed by way of a three-way interaction ($F(1,26)=5.84$, $p=0.023$) that coherence was indeed higher in low contractility ($|θ|=0.117$) vs high contractility ($|θ|=0.091$; $p_{Tukey}=0.012$), only in the $alpha_{rew}θ$ timeseries and only in the late time window (Figure 4H). In addition, the same three-way ANOVA returned no three-way interaction for trials in states of low conflict ($F(1,26)=0.5033$, $p=0.484$). Thus, this additional analysis using both raw data and an alternative means to look at frequency decomposition (coherence vs power) first supports the idea that the relationship between contractility and alpha dynamics is relevant primarily in states of high conflict. In addition, the reduced coherence across trials in alpha power relevant for reward when contractility was high, uniquely observed for high-conflict trials, additionally reveals a potential mechanistic role of for the sympathetic response during conflict. That is, fair assessment of all available information during a high-conflict decision might require disrupting a dominant reward-related signal, and sympathetic systems might contribute to this disruption.

## Discussion

Event-related physiological sciences have laid the foundations to explore cross-modal (i.e., neural and cardiac-sympathetic) collaboration subserving complex value-based behavior. We recorded parallel continuous electroencephalographic and cardiac-sympathetic data to probe associations between cognitive-neural and cardiac-sympathetic responses (contractility) while humans performed a modified version of the approach-avoidance paradigm. Our findings suggest participants were reward sensitive but encountered "conflict" when approach and avoidance presented similar value. Using the drift-diffusion model (DDM), we computationally decomposed

their behavior during conflict, which principally involved a widened decision boundary, consistent with pursuit of more evidence prior to choices. Our best-fitting model of DDM dynamics suggested that regardless of the state (low or high conflict), the boundary increased alongside increased goal-directed attention to both costs and rewards, as well as alongside increased cardiac contractility. However, exclusively in states of high conflict, the alignment of neural and cardiac-sympathetic was associated with additional increase of the boundary width. This association was markedly different from those involving alternative proxy measures of neural gain. Together, these findings offer the first evidence of a potential interactive cross-modal collaboration of neural and cardiac-sympathetic systems during evidence scrutiny in conflicting value-based decisions. Analyses involving cross-trial coherence additionally proposed a putative role for sympathetics, i.e., disrupting the dominance of reward signals.

Our findings suggest that cardiac-sympathetic activity is closely linked with neural processes and specific behavioral parameters during approach-avoidance conflict, indicating that these peripheral responses may be recruited by cognitive processes. Beginning with cardiac-sympathetics, the contractility-boundary relations are broadly consistent with sympathetic reactivity in contexts of increasing uncertainty (Palacios-Filardo and Mellor, 2019) and greater difficulty (Richter et al., 2008). However, our cross-trial coherence findings are the strongest evidence yet that the drivers of sympathetic reactivity might influence dominant reward-signal processing during value-based conflict. Under a value-based framework, such a role would not necessarily conflict with other previous findings associating cardiac indices with the pursuit of reward (Richter et al., 2016). That is, a uniform behavioral policy (i.e., approaching all or avoiding all) for offers presenting high conflict will result in long-term net-negative yields (either from mounting incremental costs incurred or mounting incremental opportunity reward costs eschewed). Optimal behavior should instead try as best as possible to map an efficiently

enumerated net value (i.e., positive or negative subjective value) onto the appropriate action. Across decision-making contexts, humans are usually biased toward more desirable information (Sharot and Garrett, 2016), to the extent that an insensitivity to reward has been reported as a robust computational phenotype of psychiatric conditions such as depression (Garrett et al., 2014; Pedersen et al., 2021). In the present study, and in at least two separately reported human studies using the same task settings (Volz et al., 2017; Shapiro and Grafton 2020), participants consistently overweighted reward when making choices. More recent evidence using transiently disruptive cortical stimulation further proposes that reward sensitivity might not simply reflect impulsivity, but a cortically-mediated model of a person's primary goal in a value-based setting (i.e., capture reward; Rolle et al., 2022). Integrating these findings with our findings under the above value-based framework, it might therefore be physiologically efficient to prioritize reward information, and reserve effortful scrutiny and juxtaposition involving multiple streams of information for moments of conflict. Reward sensitivity also generalizes to dynamic learning tasks, where recent studies report that people learn faster from positive-vs-negative prediction errors (Lefebvre et al., 2017; Garrett and Daw, 2020; Dundon et al., 2020). Consistent with our present findings, this learning asymmetry attenuates (i.e., learning from negative outcomes occurs more rapidly) when sympathetic activity is elevated (Garrett et al., 2018; Dundon et al., 2020), to the extent that sympathetic reactivity even predicts individual participants who adjust their behavior more optimally to declining changes in their environment (Dundon et al., 2020). Whether the neural sources for cardiac-sympathetics serve common mechanisms to resolve uncertainty and address biases across decisions and learning is an exciting avenue of future research.

We additionally observed a collaborative association involving neural dynamics in the alpha band. Broadly considered to reflect inhibition (Jensen and Mazaheri, 2010) and visual spatial attention (Worden et al., 2000), alpha power also shows a correspondingly flexible and goal-directed profile

in cognitive processing. For example, during spatial recall, alpha power can code spatial targets in the absence of external information (MacLean et al, 2019) consistent with post-perceptual goal maintenance. If participants are cued to switch recall to a different memory location after memory arrays disappear, alpha dynamics can likewise switch from encoding the initial target to encoding the new one (van Moorselaar et al., 2018). Alpha power can additionally signal a person's willingness to take future risks (Zhang et al., 2018), suggesting it also responds in more value-based settings. Together, these findings are consistent with our interpretation that late alpha power mediated "fair assessment", i.e., a shift in attention to process additional (cost) information alongside the reward signal information. Interestingly, we observed less association between steady-state visually-evoked potentials (SS) and DDM parameters. This might be due to task requirements. Earlier work implicates SS in coding information relevant for DDM decision boundaries (O'Connell et al, 2012), albeit in tasks requiring perceptual and not value-based decisions. Our task used large visually unambiguous stimuli and created conflict that was value-based (subjective) rather than perceptually driven. Recent human (Zhigalov and Jensen, 2020) and nonhuman (Bastos et al., 2020) work dissociates alpha signals from modulating gain of sensory information, consistent with the idea that these signals have greater relevance for behavioral responses in value-based settings. Our paradigm modifications might also explain the associations we observed principally involving visual attention (alpha) signals over those associated with cognitive control (theta) and decision making (delta). Given the varied possible sites of cortical control for the sympathetics (Dum et al., 2019), future work should not disregard any potential association between these latter signals and sympathetics, and perhaps modify the approach-avoidance paradigm to exploit them more selectively.

We lastly speculate on a network of substrates that might underly behaviorally relevant interaction between the neural (alpha) and cardiac-sympathetic (contractility) signals in states of high conflict.

It is highly likely that our observed neural dynamics in the alpha band were facilitated by noradrenergic (NE) projections from the locus coeruleus in the brainstem (LC; Rajkowski, 1993; Aston-Jones and Cohen, 2005; Joshi and Gold, 2020). The LC-NE system innervates cortical areas involved in orienting attention (e.g., parietal; Foote and Morrison, 1987), responding to arousal (Sara and Bouret, 2012), goal-relevant stimuli, and exploration (Aston-Jones and Cohen, 2005), all of which are likely relevant during moments of conflict. LC-NE can also broadly influence sympathetic activity (Samuels and Szabadi, 2008b). However, when it comes specifically to cardiac activity, evidence from both animal-optogenetic (Wang et al., 2014) and human-imaging (Wood et al., 2017) studies suggest LC-NE influences heart rate via vagal (i.e., parasympathetic) channels, contrasting with our specific cardiac assay—contractility (inotropy)—which primarily tracks beta-adrenergic sympathetic drive to the heart (see discussion in Stump et al., 2023; also see methods for how, in our study, we corrected for influences of heart rate and respiratory cycle). A key subcortical controller of this cardiac-sympathetic response is the rostral ventrolateral medulla in the brainstem (RVLM; Mandal et al., 1990; Shapoval et al., 1991; Kulkarni et al., 2023), which is the primary source of organ-specific sympathetic preganglionic neurons. RVLM principally receives inputs from the cortically modulated hypothalamus (Dum et al., 2019; Kono et al., 2020; Koba et al., 2022). LC has few direct efferent connections with RVLM, although it might communicate indirectly via its projections to the paraventricular nucleus of the hypothalamus. The behavioral changes we observed when neural (alpha) and cardiac-sympathetic (contractility) signals interact may therefore reflect two subcortical nodes (LC-NE and RVLM) activating concurrently. Alternatively, alpha-contractility associated collaboration may ultimately be mediated by interactions at the cortical level.

It is important to note that while our current data offer an important step toward resolving whether complex cognition actively recruits peripheral responses, our findings are correlational, and

should not be taken as evidence of direct mechanistic causality. Future studies incorporating selective modulation of peripheral responses, such as cardiac-specific pharmacological interventions, could further probe the causality and directionality of these interactions. Future studies should also aim to clarify the role of peripheral responses alongside brain functions not examined here, particularly subcortical activity and alternative measures of gain. In addition, while the DDM provides an elegant and intuitive decomposition of decision behavior, it remains a hypothesis of underlying mechanistic function and can potentially carry the risk of over-parameterization (Ratcliff et al., 2016). A more direct paradigm will be needed to replicate and validate our mechanistic interpretations.

*Concluding remarks*

We reveal that fair assessment of all available information (i.e., not just rewards) during a high-conflict decision potentially requires orchestration of both cognitive mechanisms and sympathetic activity. In terms of clinical relevance, autonomic function is vulnerable to neurodegenerative conditions such as Alzheimer's and Parkinson's disease (Samuels and Szabadi, 2008b; Engelender and Isacson, 2017). Future research may therefore test if features of cross-modal collaboration during complex cognition can assist with early detection.



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**Figure 1 Approach-avoidance and drift-diffusion model frameworks**

(A) In the approach-avoidance paradigm participants integrate a reward and a cost in a "take-both-or-leave-both" choice regarding a compound offer. Varying the levels of reward and cost over multiple offers affords a two-dimensional logistic framework that can identify subjective value

(p(approach); red-green gradient) and "conflict" (aqua-fuchsia gradient) across the decision space. Conflict is maximal near a "threshold" (dashed line), i.e., as p(approach) nears 0.50. Four example offers are shown (a–d) that vary in subjective value and conflict.

(B) High conflict (fuchsia) typically makes choices less consistent with lengthier RT.

(C) The slope of the "threshold" characterizes a sensitivity for reward or cost. Fitting the logistic model separately for each participant accounts for such sensitivities prior to enumerating where in decision space they subjectively experience conflict.

(D) The drift-diffusion model assumes choice and RT data can be modeled as a sequential sampling process; following an initial nondecision time (t), the decision process begins at starting point (z) and accumulates evidence at rate (v) toward one of two boundaries that determines the choice (in our case, approach (+) or avoid (-)); boundaries are separated by a distance (a). Parameters provide a fine-grained assay of behavior, such as any bias toward one choice (z), how rapidly evidence is integrated during decision formation (v) or the amount of evidence required before a choice is executed (a wider boundary denoting a more conservative criterion). States of high conflict might impact any or all of these parameters. We depict simulated schematics (n=1000 trials) of singularly changing the drift rate or the boundary separation. In each, we fixed a set of baseline parameters (t=0.30; v=1; a=2; z=0.60), and then increased or decreased v or a by 40%. Note that in each panel, there is a bias toward approach (z>0.50), and identifiably different features in the RT distributions of approach and avoid resulting from the parametric changes. For more in-depth examples, see Ratcliff and McKoon (2008).

**Figure 2 Graded approach-avoidance paradigm reveals fine-grained behavioral responses to conflict**

(A) Participants approached (accept) or avoided (reject) offers pairing varying levels of monetary reward with varying levels of painful electric shock (communicated via size of relevant bar) with a single response during gradual onset of stimuli; see Methods for success, payout and error trials.

(B) Participants integrated reward (rew [$b_1$, Eq. 1]) and cost (shk [$b_2$, Eq. 1]) into choices, with a greater weighting of reward (|rew|-|shk|>0), and a bias toward approach (int [b0, Eq. 1]) indicating reward sensitivity. Error bars are standard error of the mean across parameter estimates for each subject. ***p<0.001, **p<0.01.

(C) Choice consistency ($V_{choice}$) was lower and median response time (med. RT) was longer for states identified (using logistic choice models) as high in conflict. ***p<0.001.

(D) In states of high conflict, participants had a wider boundary (a), had a lower rate of evidence accumulation (v), had less of a bias toward approach (starting point (z)) and had a slightly shorter nondecision time (t). Boundary units are arbitrary "evidence", and drift rate is in units of "evidence" per second; starting point (z) is on a logit scale where positive values (i.e., >0.50) are closer to approach boundary (see caption for Figure 1D). Nondecision time (t) is measured in seconds.

Digitized violin plots contain 400 samples from parameter posterior. Summary data of posteriors and key comparisons are in Table 2-1. Vertical white lines span posterior HDI. *credible Bayesian difference between two parameters (θ1,θ2), i.e., 0∉HDI(D(θ1,θ2)), where D=[p(θ1|X1)−p(θ2|X2)].

**Figure 3 Interactive cross-modal collaboration associated with the decision boundary of the drift-diffusion model (DDM)**

(A) Separate flicker rates applied to reward and cost stimuli afforded capture of steady-state visually evoked potential timeseries for reward ($SS_{rew}$) and cost ($SS_{cst}$). In the "symmetry" timeseries ($SS_{sym}$), higher values reflect greater symmetry (more equal power) between the two SS timeseries (-1*ln|($SS_{rew}$-$SS_{shk}$|)). Timeseries were averaged in early [0 to 1 s] and late [1 s to 2 s] time windows relative to offer onset.

(B) Lateralized stimuli afforded capture of alpha-power timeseries relevant for reward ($alpha_{rew}$) and cost ($alpha_{cst}$). In the "symmetry" timeseries ($alpha_{sym}$), higher values reflect greater symmetry (more equal power) between the two alpha timeseries (-1*ln|($alpha_{rew}$-$alpha_{shk}$|)). Timeseries were averaged in early [0 to 1 s] and late [1 s to 2 s] time windows relative to offer onset.

(C) Posterior parietal delta and frontal-midline theta power. Timeseries were averaged in early [0 to 1 s] and late [1 s to 2 s] time windows relative to offer onset.

(D) The pre-ejection period (PEP) is recorded with combined impedance cardiography (ICG) and electrocardiography (EKG); shorter PEP indicates increased sympathetic beta-adrenergic myocardial contractility. Our contractility estimates, where higher values reflect greater cardiac-sympathetic drive (contractility=-1*ln(PEP)), were averaged across each heartbeat in a [0 to 2 s] time window relative to offer onset.

(E) Singular models for DDM parameters {a,v,z,t} modeled by a single regressor ($x_1$; i.e., either a neural variable or contractility), separately for states of low and high conflict (Eq. 6). Six models improved fits beyond the baseline model in Figure 2D. Fits assessed relative to baseline with improvements in deviance information criterion (-ΔDIC), positive values reflecting better fit. Double-headed (↔) arrow denotes an association that could be negative or positive.

(F) Cross-modal models for DDM parameters {a,v,z,t} modeled by either additive or interactive models winnowed from the fits in Figure 3E. Additive models (empty circles) modeled DDM parameters by a neural variable ($x_1$) in addition to contractility (cont.), separately for states of low and high conflict; 16 regressors in total. Interactive models (circles with crosses) also included a third regressor for the product of the neural signal and contractility [Eqs. 7–8]. Six models improved fits beyond the best-fitting model in Figure 3E.

(G) Complement models (Eq. 9) asked if the fit of the best-fitting cross-modal model (which included $alpha_{shk}$; marked "m1" in Figure 3F) could be improved by adding the complement (i.e., set difference) of cross-modal models using neural variables that passed the singular model

stage, using their best-performing forms (with or without interactions), marked with “m2” in Figure 3F. Each model improved fits beyond the best-fitting model in Figure 3F. In the best overall fitting complement model (marked with *), DDM parameters were modeled by four regressors: $alpha_{shk}$, $alpha_{sym}$, contractility and $alpha_{sym}$*contractility.

(H) Proxy of variance explained ($R^2$) by best fitting baseline, singular, cross-modal, and complement models across varying RT bin sizes. Each trial’s RT was simulated using a Wiener-like process with relevant model parameters and regressors, and $R^2$ values were derived from Pearson correlations between RT bin medians (observed vs simulated).

**Figure 4 Dynamics of the best-fitting complement model**

(A–D) Parameter posteriors from best-fitting (complement) model of DDM parameters. Most neural and cardiac-sympathetic relations involved the decision boundary (a). In both low- and high-conflict states, wider boundaries were related to greater desynchronization of $alpha_{shk}$, greater symmetry in alpha ($alpha_{sym}$) and increased contractility. Unique to states of high conflict, the boundary showed additional positive association with the alignment of cross-modal signals ($alpha_{sym}$*contractility(cont.)). Digitized violin plots contain 400 samples from parameter posterior. Summary data of posteriors are in Table 4-1. Vertical lines span highest density interval (HDI) of coefficient posterior, and are white if HDI does not contain 0 (also marked with *), black otherwise.

(E) Parameter posteriors from a model to discretize the neural and cardiac interactions associated with the decision boundary. Boundary is widest (relative to the baseline, low conflict, low late $alpha_{sym}$ and low contractility—Δ boundary) in high conflict when $alpha_{sym}$ and contractility are both high. + and - symbols respectively reflect high and low (for physiology signals, relative to participant medians). Digitized violin plots contain 400 samples from parameter posterior. Summary data of posteriors and key comparisons are in Table 4-2. Vertical lines span the highest density interval (HDI) of coefficient posterior and, are white if HDI does not contain 0. *** denotes this posterior was credibly larger than all others depicted, i.e., $0 \notin HDI(D(\theta 1, \theta 2))$, where $D=[p(\theta 1|X1)-p(\theta 2|X2)]$ for all possible values of θ2.

(F) Control models substituted proxy measures for local and global brain activity for all regressors featuring contractility in the best-fitting complement model. The model substituting contractility with global field power (GFP) was a slightly better fit. However, inspection of the parameters show opposing associations with the boundary (marked by black arrows). That is, GFP's interaction with $alpha_{sym}$ was associated with a contraction of the decision boundary. Summary data of posteriors for the GFP control model in Table 4-2.

(G) Phase-angle timeseries of alpha contralateral to reward ($alpha_{rew}\theta$ - top) and cost ($alpha_{shk}\theta$ - bottom) in high conflict, averaged across subjects separately for trials that were higher (dark red) or lower (light red) than their median contractility.

(H) Summarizing phase coherence (absolute phase-angle value $|\theta|$) across early and late time windows, we see a three-way interaction whereby late coherence diminishes significantly in high contractility, and only in the alpha timeseries contralateral to reward.