

Cardiovascular indicators of disgust

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Abstract

A bradycardia and an increasing parasympathetic activity are often recommended as characteristic physiological disgust reactions. However, findings concerning the influence of disgust on heart rate and autonomic control are heterogenous. Apart from this, only a few studies examined cardiovascular reactions to disgust, besides heart rate.

The aim of this study is a differentiated description of cardiovascular reactions going along with disgust using impedance cardiography. Moreover, it will be surveyed if different cardiovascular responses are associated with content-specific disgust-inductions.

One-hundred subjects watched three films: A neutral film (screensaver), a filmclip showing an amputation of the upper extremity and a filmclip displaying a person who is vomiting. The latter films are regarded as disease- and food-related disgust stimuli respectively, representing two superior disgust domains.

Subjective, electrodermal and cardiovascular reactions to these films were compared using Repeated Measures ANOVAs.

Strong subjective, electrodermal and cardiovascular reactions towards the filmclips with disgusting content were observed. The cardiovascular reactions of the disease- and food-related disgust stimuli differed in subjective and physiological parameters. Thus, a decrease in heart rate could only be observed as a response to disease-related disgust-induction. The observed differences are discussed as an endorsement for a domain-specific organisation of disgust reactions.

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1. Introduction

Emotions are commonly linked with concurrent subjective, behavioral and physiological changes (e.g. Scherer, 1993). The basic emotion of disgust is assessed by self-descriptions (feelings of dislike and nausea) and behavioral expressions (alienation, characteristic mimic response). Although only very few studies on the physiological basis of this emotion in the peripheral nervous system have been published, a bradycardia or a heightened parasympathetic activity is often recommended as a physiological marker of disgust. Woody and Teachman (2000) propose a parasympathetically mediated bradycardia as part of a definition of disgust. Levenson et al. (1990) state a rise

in vagal activity during the experience of disgust in the same way, because of the strong association with a decreasing heart rate and nausea. Furthermore, Rozin et al. (1999) suppose a general parasympathetic organisation of disgust during which the sympathetic activation is constant or decreasing.

With reference to the assumption that the experience of disgust is dominated by the parasympathetic nervous system, several points remain unclear. It can be observed that disgust-induction is accompanied by consistent increases in electrodermal activity (Gross, 1998; Gross and Levenson, 1993; Johnsen et al., 1995; Lang et al., 1993; Levenson et al., 1990; Schienle et al., 2001), which indicates an increased sympathetic activity. After an induction of disgust, Demaree et al. (2006) found no significant changes in respiratory sinus arrhythmia, which is an indicator of parasympathetic activity. Findings concerning the influence of disgust on heart rate, which is mediated by the sympathetic and by the parasympathetic

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system, are heterogenous. The majority of studies found evidence for a decrease in heart rate (Gross, 1998; Gross and Levenson, 1993; Johnsen et al., 1995), while in some studies heart rate was constant (Ekman et al., 1983; Lang et al., 1993; Levenson et al., 1990) and some studies even observed an increase in heart rate (Alaoui-Ismaili et al., 1997; Vernet-Maury et al., 1999; Vrana, 1994; Schienle et al., 2001). In addition, the effects of the sympathetic and parasympathetic nervous system are to a large extent antagonistic. However, from a functional point of view their effect on the different vegetative organs is more of a working “hand in hand” rather than “against each other” (Birbaumer and Schmidt, 1999, p. 152). Thus it has to be considered, whether the question of sympathetic or parasympathetic dominance can be replaced by the question of their interaction (see Berntson et al., 1994). In this vein, Jänig (2003) argues that ideas about a functional antagonistic organisation of both sub-systems as proposed by Rozin et al. (1999) are untenable, since both systems are co-activated in most contexts (Stemmler, 2004).

Although parasympathetic activation as a reaction to disgust has not been documented so far (Woody and Teachman, 2000), heart rate seems to be a distinguishing component among the physiological correlates in experiencing disgust. Levenson et al. (1990, 1991) conducted a series of experiments with different methods of disgust-induction (imagination, mimic instruction). They showed that a light decrease of heart rate or no change in heart rate could differentiate disgust significantly from other negative emotions. Stark et al. (2005) found a significant negative correlation between average heart rate and the intensity of disgust stimuli.

It seems reasonable to examine cardiovascular reactions of disgust in more detail, because of the relevance of heart rate changes during the experience of disgust. These investigations can lead to a better understanding of the physiological correlates of this basic emotion. Findings on cardiovascular parameter changes following disgust-induction are rare besides heart rate. To our knowledge, only Schienle et al. (2001) assessed blood pressure changes and found no change in systolic and diastolic blood pressure after inducing disgust.

In order to be able to examine cardiovascular disgust reactions in depth, impedance cardiography, which has never been used for the examination of disgust reactions until now, seems to be the method of choice. Its advantages are that, on the one hand, a rather accurate measure of time for the blood transport's dynamic is gained, on the other hand, statements about the transported amount of blood can be made. Thus relative changes within a subject can precisely be described non-invasively (Schandry, 1989).

Since no direct noninvasive assessment of sympathetic and parasympathetic cardiac efferent activity exists, only indirect indices of autonomic activity are on-hand. Changes in these indices are no direct reflections of sympathetic and parasympathetic activations, since the cardiovascular system and in particular heart rate is influenced by both of them. Cardiovascular changes associated with sympathetic and parasympathetic activations could also occur as a result of other underlying physiological mechanisms (see Bradley, 2000). Nevertheless,

some conclusions can be drawn about sympathetic and parasympathetic activations of the cardiovascular system with these parameters. In this regard a heightened sympathetic activity is linked to an increasing heart rate, myocardial contractility and arterial blood pressure. A heightened parasympathetic activity is associated with a decreased heart rate and a heightened heart rate variability (Birbaumer and Schmidt, 1999; Brownley et al., 2000).

Furthermore, estimations of stroke volume, cardiac output and total peripheral resistance allow us to make statements about the hemodynamic basis of blood pressure changes and patterns of cardiovascular reactivity. It is possible, however, that the cardiovascular reactions to experimentally induced disgust do not display a distinct pattern but rather a common psychophysiological answer to a certain type of stressor. Schneiderman and McCabe (1989) point out two reaction patterns, which are often found in experimental settings: a) *active-coping-patterns* and b) *passive-coping-patterns*. Active-coping-patterns can often be found with stressors, which cause a defensive behaviour. The cardiovascular response of an active-coping-pattern is characterized by increases in arterial blood pressure (particularly systolic blood pressure), heart rate, cardiac output and myocardial contractility as well as decreases in total peripheral resistance. Passive-coping-patterns are associated with stimuli which demand attention to an extent that makes active-coping of the organism impossible (e.g. Cold-Pressor-Test). The cardiovascular response associated with a passive-coping-pattern is increased arterial blood pressure (particularly diastolic blood pressure) and total peripheral resistance as well as a decreased heart rate. It thus has to be tested whether the observed cardiovascular changes correspond to the reaction patterns described by Schneiderman and McCabe (1989) or to an explicitly different emotion-specific pattern.

Examining physiological changes after disgust-induction in more detail, raises questions about disgust being a homogenous phenomenon. The reason for the heterogenous findings concerning the relationship between heart rate and disgust is unclear. Gerlach et al. (2006) trace those heterogenous heart rate reactions when experiencing disgust to the stimuli used. They state that a decrease in heart rate can only be observed when stimuli are used which show blood and physical injuries.

Thus it is possible that different reactions of heart rate depend on the type of disgust-inducing stimuli. Although Rozin et al. (1994) were able to show that, depending on the respective stimulus (food-related stimuli, body boundary violations), mimic disgust reactions are different, those aspects have been neglected so far. Similarly, Olatunji et al. (2005) conclude that disgust is not a homogenous construct on a subjective level. They showed that a two factor model is superior to a single factor model as a result of analysing subjective items of the Disgust scale (Haidt et al., 1994) with confirmatory factor analysis. They suggest core and animal-nature disgust according to Rozin et al. (1999) as superior factors. Another conception of disgust types from Schienle et al. (2002) differentiates between hygiene-related and food-related disgust. Yartz and Hawk (2002) distinguished between disgust related to blood and disgust of a different nature.

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