



Does disgust increase parasympathetic activation in individuals with a history of fainting? A psychophysiological analysis of disgust stimuli with and without blood–injection–injury association

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ABSTRACT

People with blood–injection–injury fear can faint when being confronted with blood, injections or injuries. Page (1994) holds that people with blood–injury phobia faint, because they are disgust sensitive and disgust facilitates fainting by eliciting parasympathetic activity. We tested the following two hypotheses: (1) Disgusting pictures elicit more disgust in blood–injection–injury-anxious people with a history of fainting than they do in controls. (2) Disgust causes parasympathetic activation. Subjects were 24 participants with high blood–injection–injury fear and a history of fainting in anxiety relevant situations and 24 subjects with average blood–injection–injury fear and no fainting history. We analyzed self-reported feelings of disgust, anxiety and faintness and reactions in heart rate, skin conductance, blood pressure and respiratory sinus arrhythmia during the confrontation with disgusting pictures with and without blood content. We did not find any evidence that the blood–injection–injury anxious subjects were more disgust sensitive than the control subjects and we also did not find any evidence that disgust elicits parasympathetic activation.

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1. Introduction

In blood–injury phobia irrational and excessive fear is triggered by blood, wounds, syringes and other related stimuli. This may result in avoidance of necessary medical treatment despite negative health consequences. The life–time prevalence is estimated to be about 3.5% (Bienvenu & Eaton, 1998).

Fainting is a common response when individuals are confronted with blood, injuries or syringes (DSM IV-TR; APA, 2000). In individuals with blood–injury phobia, this phenomenon is much more common than it is in individuals who are not afraid of such situations. In a study by Öst (1992) 70% of the blood phobics and 56% of the injection phobics had a history of fainting. Fainting might induce or enforce the fear of blood–injection–injury related stimuli through conditioning. Indeed, fainting is a major reason why blood–injection phobics are afraid of confrontation with their feared stimuli (Page, 1994). However, the mechanisms behind the blood–injury associated fainting response so far are not well understood.

A number of attempts to explain the fainting response in association with blood–injury fears have been proposed. Arguably, two of these hypotheses currently have the strongest scientific merits: the “disgust theory of fainting” as proposed by Page and the “blood loss prevention theory of fainting” as originally proposed by Barlow (1988) and modified by Diehl (2005).

1.1. Disgust theory of fainting

Page (1994) presumes that disgust rather than anxiety is the crucial emotion in blood–injury phobics. Moreover, he assumes that parasympathetic activation is strongly linked to disgust and that elevated disgust sensitivities in blood–injury phobics may partially cause the elevated fainting rate in these subjects. However, heart rate decelerations have only been found when disgust was elicited by stimuli associated with blood and injuries (Gross, 1998; Gross & Levenson, 1993). In contrast, non-injury-related disgusting stimuli tend to elicit heart rate accelerations instead (Alaoui-Ismaili, Robin, Rada, Dittmar, & Vernet-Maury, 1997; Schienle, Stark, & Vaitl, 2001; Vrana, 1994). This pattern of results suggests that the confrontation with blood rather than the emotion disgust is responsible for the vasovagal reactions that are caused by blood–injection or injury stimuli.

In a study by Gerlach et al. (2006) heart rate, blood pressure, skin conductance and respiratory sinus arrhythmia were measured

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during venipuncture and recovery from venipuncture. No evidence for increased parasympathetic activation in blood–injury phobics with a history of fainting was found in comparison to phobics without such a history. Furthermore, phobics without a history of fainting actually tended to report more disgust than phobics with such a history. This finding suggests that disgust may not be as relevant for the fainting response as previously argued. Similarly, Vossbeck-Elsebusch and Gerlach (2012) found that the fainting history or self-reported vasovagal symptoms were not associated with disgust sensitivity in a study of blood donors.

Page's (1994) hypothesis that there is an increased disgust sensitivity in blood–injury phobics is further questioned by studies that failed to find a generally increased disgust sensitivity in blood–injury-phobics (Koch, O'Neill, Sawchuk, & Connolly, 2002; Schienle et al., 2003). However, others found increased disgust sensitivity (Oßwald & Reinecker, 2004; Sawchuk, Lohr, Lee, & Tolin, 1999; Sawchuk, Lohr, Westendorf, Meunier, & Tolin, 2002; Schienle, Schäfer, Walter, Stark, & Vaitl, 2005). Olantunji, Williams, Sawchuk, and Lohr (2006) found evidence for domain specific disgust sensitivity. In their study, individuals with blood–injection–injury anxiety experienced more disgust than controls when they were confronted with animal-related disgusting stimuli, but not when they were confronted with food-related disgusting stimuli (e.g. “core disgust”).

1.2. “Blood loss prevention theory of fainting”

Barlow (1988) suggested that lowering of blood pressure may reduce blood loss in case of injury. The idea of fainting being a phylogenetically old mechanism established as means to prevent blood loss when injured can be criticized due to the observation that during surgeries, commonly blood pressure does not drop before a blood loss of at least 30%. Therefore, Diehl (2005) extended Barlow's original notion by suggesting that some people may have an especially sensitive neurocardiogenic reflex triggered by stimuli much less intense than a 30% blood loss. He assumes this to be an early attempt to support haemostasis before the development of larger blood losses. This notion of an especially sensitive neurocardiogenic reflex in subjects with a fainting history in blood–injection–injury situations is supported by studies looking at their reactions during a tilt-table test (Accurso et al., 2001) and a constrictive pain test (Adler, France, & Ditto, 1991). Furthermore, when humans faint, the Willebrand factor is released resulting in even better coagulation of the blood (Casonato et al., 2003), which supports the prevention of blood loss.

1.3. Mechanisms other than parasympathetic activation associated with fainting

There is evidence that parasympathetic activation alone is not a sufficient cause for the fainting response. Prevention of bradycardia through pacemakers or pharmacological blocking of parasympathetic efferent pathways does not prevent the fainting response (Kosinski, Grubb, & Wolfe, 2004). The loss of sympathetic tone in major blood vessels seems to have at least some relevance for the fainting response as abrupt cessations of sympathetic nerve activation to skeletal muscles have been observed before neurocardiogenic syncopes occur (Wallin & Sundlöff, 1982) and it has been found that the alpha-2-antagonist Yohimbin, which increases sympathetic activity, decreases fainting rates (Hainsworth, 1995). The loss of sympathetic tone counteracts vasoconstriction in the skeletal muscles and an outflow of blood in the skeletal muscles occurs. An amplification of the outflow of blood by an active vasodilation mechanism in the skeletal muscles has consequently been proposed (Dietz et al., 1997; Wieling, Smit, van Steenwijk, van Lieshout, & Karemaker, 1997). Altogether, it is ascertained that mechanisms

other than parasympathetic activation are involved in the neurocardiogenic syncope, but it is still not clear if there is any crucial involvement of parasympathetic activation in blood–injury related fainting, be it disgust associated or not.

Steptoe and Wardle (1988) found that individuals with high blood–injection–injury anxiety and a history of fainting do not differ from control subjects in RSA, whilst they do differ in heart rate and blood pressure, showing a biphasic response in the individuals with high anxiety and a history of fainting. Gerlach et al. (2006) found evidence for more sympathetic activation in blood–injection–injury anxious subjects than in controls during venipuncture, but neither during nor after venipuncture, they found evidence for increased parasympathetic activation. Sarlo, Buodo, Munafò, Stegagno, and Palomba (2008) also did not find any evidence for parasympathetic activation or a biphasic response in different hemodynamic variables. They found an imbalance in sympathetic functioning in blood phobics instead. They assume that vasodilation is supported by the activation of the β_2 -adrenoreceptor in the skeletal muscle vasculature. A large rise in circulating epinephrine levels has been found in patients who fainted spontaneous or during the head-up tilt test (Klingenheben, Kalusche, Li, Schopperl, & Hohnloser, 1996; Sra et al., 1994; Vingerhoets, 1984), which supports the assumption that a deregulation of the sympathetic branch of the nervous system facilitates fainting. Moreover, Donadio et al. (2007) demonstrated that arousal elicits inhibition of muscle nerve sympathetic activity in blood–injection–injury phobics with a fainting history. In a study from Van Overheld, de Jong, and Madelon (2009) disgusting stimuli only lead to parasympathetic responses in the digestive system of highly blood–injection–injury anxious subjects, but no parasympathetic-induced changes in the cardiac system were found.

To investigate if individuals with high blood–injection–injury-fear and a history of fainting in blood–injection–injury-relevant situations show an excessive activation of the parasympathetic nervous system when they are confronted with disgusting stimuli with and without blood, we analyzed heart rate, blood pressure, skin conductance levels, respiration rate and respiratory sinus arrhythmia. If we could find a decrease in heart rate and blood pressure as well as an increase in respiratory sinus arrhythmia compared to baseline when blood–injection–injury fearful subjects are confronted with disgusting stimuli, this would support Page's (1994) theory. The reaction should then only be seen in blood–injection–injury-anxious subjects with fainting history, but not in controls. In contrast, if we find an increase in heart rate, blood pressure and skin conductance levels, this would show that the confrontation with the disgusting pictures leads to sympathetic activation.

As Page (1994) also holds that disgust is predominant in blood–injection-fearful subjects and is crucial for fainting, we also investigate whether disgusting stimuli with or without blood elicit more feelings of disgust, faintness or anxiety in the blood–injection–injury anxious individuals with a history of fainting than they do in a control group. Our design allows us to distinguish between effects of stimuli with blood and the effects of disgust, something that has not been previously studied using peripheral physiology measures. Van Overheld et al. (2009) cannot rule out that blood-related material might lead to relevant parasympathetic activation, but they could not find any evidence for cardiac parasympathetic activation when they confronted high and low blood–injection–injury anxious with disgusting stimuli. If only blood associated stimuli rather than all disgusting stimuli should bring along faintness and parasympathetic activation, this would support the notion of fainting being a coagulation-supporting safety mechanism without disgust association. Our design also allows us to investigate the relation of

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