Pergamon

Anxiety Disorders
17 (2003) 45–58

JOURNAL OF Anxiety Disorders

The role of disgust in faintness elicited by blood and injection stimuli

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Received 26 January 2001; received in revised form 29 May 2001; accepted 10 July 2001

Abstract

To examine the role of disgust in blood-injection fear and faintness, 79 individuals high and low in disgust and distressed either more by blood or injections were exposed to both a blood slide and a needle slide. Self-reported faintness and systolic and diastolic blood pressure were measured. High disgust individuals reported more faintness to both slides, but those who were more distressed by blood reported more faintness to the blood slide and those who were more distressed by needles reported more fear to the needle slide. Both systolic and diastolic blood pressure assessments manifested a diphasic response. The diphasic response pattern was most evident among the high disgust subjects, who were more distressed by blood than injections. Results are discussed in terms of the relevance of disgust in the etiology of fear and faintness in blood-injury phobia.

Keywords: Disgust; Faintness; Fear; Blood-injury phobia

Emotional fainting occurs around blood and with a disturbingly high frequency among those who are fearful of blood and injury (Öst, 1992; Öst, Sterner, & Lindahl, 1984; Page, 1996, 1998; Thyer, Himle, & Curtis, 1985). The type of fainting has been described as vasovagal syncope (Lewis, 1932), a term that describes all fainting of presumed vasovagal origin. Vasovagal syncope can be triggered by events of an emotional (e.g., blood) and nonemotional nature (e.g., micturition, immersion of one's face in water, body tilt, and anticipation of electric shock; Thyer et al., 1985). This tendency to faint is partially inherited and

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0887-6185/02/$ – see front matter
PII: S0887-6185(02)00169-X

the genes responsible are shared with those that underlie blood fears (Page & Martin, 1998). The physiological mechanisms responsible for vasovagal syncope are well described, with the response involving two phases (Graham, 1961; Graham, Kabler, & Lunsford, 1961). In the first phase, there is a domination of sympathetic nervous system activity consistent with elicitation of the fight-or-flight response (Thyer & Curtis, 1985; Thyer et al., 1985). Of particular relevance to fainting is the initial rise in blood pressure. In the second phase, there is a relative increase in parasympathetic nervous system activity that is consistent with a conservation–withdrawal response (Marks, 1988; Vingerhoets, 1984). The occurrence of fainting is particularly related to the drop in blood pressure in the latter part of the diphasic response. If the drop is of sufficient magnitude to affect cerebral blood flow, syncope will occur as a consequence of cerebral anoxia. However, the genetic and physiological studies fail to address an important step in the causal sequence. Namely, what processes bridge the gap between the observation of the blood and injury stimulus and the recruitment of the physiological response?

If people only fainted following blood loss, vasovagal syncope could be explained purely in terms of hemodynamics. However, people can faint without any loss of any blood (Öst et al., 1984). If people always fainted following removal of a fearful stimulus, fainting could be explained as an exaggerated relief reaction (Graham, 1961; Graham et al., 1961). However, fainting is virtually nonexistent in all anxiety and phobic disorders, except blood-injury phobia (Connolly, Hallam, & Marks, 1976). If people inherited or acquired a physiological process that increased the likelihood of fainting, once again the explanation of fainting would be relatively straightforward. However, fainting among phobics appears to be particularly linked to situations and objects involving blood and injury (Marks, 1988). Therefore, there appears to be something about blood and injury that results in such stimuli being processed in a manner that elicits a diphasic vasovagal response.

One plausible hypothesis about the way that blood and injury stimuli can elicit fainting can be generated from the work of Davey and colleagues (e.g., Davey, 1994; Davey et al., 1998; Mattchett & Davey, 1991; Webb & Davey, 1992). They have convincingly demonstrated that phobic reactions do not always involve the emotion of fear (cf. Arrindell, 2000). Sometimes ‘‘phobic’’ reactions involve the emotion of disgust. Fearful phobic reactions tend to be associated with animals that are classifiable as predatory (i.e., capable of inflicting pain and injury). Examples include sharks and lions. ‘‘Phobic’’ reactions that involve disgust tend to be associated with animals that are classifiable as potential contaminants (i.e., capable of causing illness if consumed or touched). Examples include toads, maggots, and blood. It is also possible that the category of disgust-relevant animals can be broken down further into fear-relevant, dry or nonslimy invertebrates, and slimy or wet looking animals (Arrindell, 2000).

While the structure of animal phobias is being clarified (Arrindell, 2000; Taylor, 1998), for the present argument, making the assumption that the processes responsible for the two emotions of fear and disgust are at least partially independent, places stimuli involving blood and injury in a curious position