Chicken or Egg?

James-Lange Theory of Emotion

The autonomic nervous system is known to be linked integrally to emotions.

In his classic description of the adrenal response, Walter Cannon suggested that the autonomic nervous system served rather like a support system. An emotion-provoking stimulus triggers the appropriate emotion and thence the autonomic nervous system (ANS) into action such as fight or flight.

In contrast, William James (1890) proposed an alternative view in which the ANS response is directly triggered by the stimulus and the emotional experience in fact lags behind, depending on a ‘reading’ of the autonomic reaction. Rather simplistic representation of this theory might be that someone is ‘fearful because he is running from a bear’.

Cannon attempted to contradict James’ view by pointing out various flaws in the argument, such as the fact that paralysed people with loss of sensation still experience the full range of emotions.

The fact that, at the time (1927), Cannon was the leading physiologist in the world, whereas James was merely a gifted writer and psychologist, meant that Cannon’s view prevailed.
However, some of Cannon's objections have since been rendered invalid by our increasing understanding of the ANS.

For instance, patients who are paralysed and have lost 'somatic' sensation, still retain an active ANS (in fact, often, it becomes hyperactive). These patients report a lack of emotional intensity, such as feeling 'as if' they were angry.

So in fact, we are now aware that the ANS, and thus a number of physical sensations, has an intimate link with our emotions; in fact, it is a two-way street.

Note that we can become 'conditioned' in our responses to pain and other stimuli. Previously neutral stimuli paired with aversive events can evoke fear and other negative emotions. In essence, we 'learn' our response to pain over a period of time, and it can become ingrained.

General Adaptation Syndrome

Hans Selye looked beyond the body's immediate response to stress and observed that: (a) Long term exposure to stressful situations can deplete the organism's ability to maintain the stress response, and (b) The pattern of these deleterious effects is independent of the source of stress.

In 1956, he outlined a three-stage progression of responses to stress termed the General Adaptation Syndrome: Alarm, Resistance and Exhaustion.

Stage of Alarm. When a stressor is first encountered, the initial series of responses depends upon the autonomic nervous system, the immune system and other defences to cope with the emotional, behavioural and physiological aspects of the stressor.

Stage of Resistance. Involves maintenance of this reaction to the stressor, which includes
reparative processes such as fever regulation, tissue repair, control of inflammation, etc.

**Stage of Exhaustion**. The defences fail, metabolic reserves are depleted, physiological functions undergo a general decline, and serious illness (or even death) ensues.

Note that this general response is independent of the initial trigger event, being more closely related to the interpretation of the environment than to the physical intensity of the aversive stimuli.

The degree to which an event is stressful to an individual may vary, but one of the main factors is thought to be the element of control over the situation that the people perceive themselves to have.

For instance, in natural and technological disasters (earthquakes, train crashes), the individual may have no control over the situation, which can compound the effect of the stressful life event.

A further significant component is the presence of conflicting consequences (e.g. in animal studies, reward or punishment).

In animal experiments, exposure to shock (even if unpredictable and uncontrollable) will not cause physical illness such as stomach ulcers unless the frequency of occurrence is fairly high: an occasional brief shock does not cause this problem.

However, acute trauma such as surgery can lead to the 'shock syndrome' a diffuse outpouring of the entire autonomic nervous system.

I suspect this is even more likely if post-operative pain control is sub optimal.
In animals, a lack of coping response for acute, profound stressors can cause sudden death through hyperactivity of the parasympathetic nervous system (part of the ANS).

Relatively mild stressors, if not controllable by the individual, can lead to suppression of the immune system, which in turn can increase the vulnerability to diseases, trigger allergies, or lead to autoimmune disorders.

Note that acute episodes of stress lower the level of neurotransmitters (catecholamines such as noradrenaline: see below) in the reward system.

When an animal is acutely or chronically stressed, the hippocampal (part of the brain) level of the growth factor BDNF dramatically decreases; antidepressants (but not other psychotropic medications) have the opposite effect.

Neuroimaging studies have found that stress-related disorders are associated with reduced hippocampal volume, which may be related to elevation in plasma cortisol levels and the duration or severity of the stress or depressive episode.

Reduced hippocampal volume in patients suffering from major depression has been seen in various studies.

Conclusion:

“Feedback from the autonomic nervous system plays an important role in determining whether or not an emotion will be experienced; environmental cues interact with this feedback to determine the nature of the emotional response.”

(Source: Drugs, Brains and Behavior by C. Robin Timmons & Leonard W. Hamilton).