**Supplemental Text Box 3**

**Tonic Immobility**

Tonic immobility has been known by many different names: the passive defense reflex,1 immobility reflex,2 feigning death/death feint,3 letisimulation, playing possum/*tot stellen*, still reaction, akinesis, terror paralysis, contact defensive immobility, animal hypnosis, and catalepsy.4–6

Clinical features of tonic immobility in animals include the following:

* *Onset* involves a sudden, prolonged stillness (often in bizarre postures), absence of vocalizations, and a lack of responsiveness to external stimuli (including painful stimuli) that lasts seconds to hours, depending on species and circumstances.5,7,8
* *The animal’s body* may be hypertonic (muscle rigidity) and can often be manipulated (waxy flexibility).9–11 In some species—for example, the rabbit—hypertonicity may be followed by hypotonicity.12–15 Tremors in the extremities can occur.16 In hens and rabbits, a decrease in electromyographic activity has been documented.12,17
* The animal often has a *glassy*, *unfocussed gaze* or stare and periods of eye closure.12,18–20
* There is a *loss of the righting reflex*17,21–23 and of mono- and polysynaptic reflexes,24 and a weakening or disappearance of conditioned reflexes.25
* There is a *drop in body temperature*.9,26,27
* *Heart rate changes* show a biphasic response. During the induction of tonic immobility (when the animal is being restrained), heart rate usually increases.28–30 Once tonic immobility is established, heart rate decreases, often markedly (which can include asystole or a potentially fatal cardiac arrhythmia).2,12,16,26–28,30–35
* *Respiratory changes* also show a biphasic response. During induction of tonic immobility, respiratory rates typically accelerate.27,29 Once tonic immobility is established, respiration slows or becomes very shallow.6,12,20,26,27,36
* *EEG activity* also shows a biphasic response.12 During induction and in the early stages of tonic immobility, EEG activity is characteristic of a state of alertness/arousal—an EEG pattern characterized by relatively low voltage, fast activity in the cortex, and theta rhythms in the subcortical regions and sometimes the cortex together with an immobility and unresponsiveness—and there appears to be ongoing processing of the event.2,7,12,17,22,27,37–43 Thus, while animals may appear dead or unresponsive to exteroceptive stimuli, they are still processing information, and new learning does occur. If tonic immobility is maintained for a longer period, the EEG changes to a low-arousal pattern—high-voltage slow EEG activity.12,44 Whereas the high-arousal EEG pattern is associated with a very elevated heart rate or with only a slight decrease in heart and respiratory rates (depending on the species), the low-arousal EEG pattern is associated with further decreases in heart and respiratory rates.12,37 Klemm (1966) highlighted that the early stages of tonic immobility—in which the animal’s body is immobile and unresponsive, and the EEG shows an arousal pattern—reflects a clear case of EEG-behavioral dissociation. How these animal data translate to humans is not known.
* *Defecation* commonly occurs.45–49
* *Termination is abrupt* and is followed by renewed struggle or attempts to escape.8

In humans, tonic immobility has been described in accounts given by survivors of sexual assault, physical assault, attacks by wild animals, shell-shocked soldiers, and plane/car crash victims.[[1]](#footnote-1),9,47,51–55 54,56 57 50,56,58–63 Victims report a subjective experience of immobility, subjective feelings of coldness, numbness, and analgesia, uncontrollable shaking, eye closures, and vivid recall of details.5,9,47,51,52,64,65 They also report fear in combination with dissociative experiences (derealization and depersonalization) and a sense of entrapment/inescapability, futility, or hopelessness.9,47,51,60

Although the animal literature strongly suggests that tonic immobility occurs in a biphasic manner and that dissociations between different markers of arousal may occur—for example, an EEG indicative of alertness/arousal combined with behavioral unresponsiveness and immobility—the lack of studies of tonic immobility in humans and the lack of translational studies between animals and both human and nonhuman primates make it difficult to know whether tonic immobility in humans may similarly follow a biphasic pattern. Theoretically, it is possible that, as a function of individual variations in neurophysiological reactivity, humans may show a number of different phases or patterns of arousal within the tonic immobility response. For example, in the initial stages of tonic immobility, when the individual shifts to tonic immobility from a state of extreme sympathetic arousal, behavioral immobility may be coupled with a high-arousal EEG pattern and an elevated heart rate. Once the parasympathetic component of the tonic immobility response is activated, however, a drop in heart rate will ensue. If the drop in heart rate is not extreme, the high arousal EEG pattern could be maintained. If the heart rate drop is extreme, however, and results in significant bradycardia, the EEG pattern is likely to shift to a low-arousal pattern—high-voltage, slow-wave pattern—characteristic of cerebral ischemia.

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1. Some of these accounts use the term *freezing* to describe the immobility attributed to the affected individuals.50 The descriptions of the immobility, however, suggest a state of tonic immobility, in which the individuals cannot be roused into any action, rather than the attentive immobility of the freeze response, in which individuals are primed to respond and would, in theory, burst into action when the opportunity arises or when prompted to so. [↑](#footnote-ref-1)