

Pain-Tracks: Cumulative Pain from Different Causes of Death in Domestic Chickens (Broilers and Egg-Laying Hens)

Analyses performed by the Welfare Footprint Project team based on the work presented in the books [Quantifying Pain in Laying Hens](#) and [Quantifying Pain in Broiler Chickens](#).

Summary of Estimates

Estimated cumulative time in pain (hours), of three intensities, due to different causes of death in chickens

Death by:	Sepsis	Dehydration	Canibalism	Exsanguination
	Hours	Hours	Hours	Hours
Excruating pain	2.2 [1.5-3]	Not likely to be felt	0.075 [0.05-0.1]	Not likely to be felt
Disabling pain	131.5 [64.1-198.9]	17.8 [14.8-20.8]	0.121 [0.083-0.159]	0.012 [0.001-0.023]
Hurtful pain	398.5 [163.3-633.7]	148.3 [48.4-248.2]	0.09 [0.062-0.118]	0.003 [0-0.006]

Death by Dehydration

The effects of dehydration, which seem to be comparable in birds, humans and other mammals (Box 1), are often studied based on the percentage of body mass loss associated with dehydration, as it largely determines clinical symptoms. Accordingly, the temporal segments in Pain-Track 1 are based on this criterion, described in more detail in Table 1. The duration of each segment is estimated mostly from observations of the association between percentages of body mass loss and water deprivation times in broiler chickens [1], depicted in Figure 1. The data is consistent with evidence from trials conducted in the 1960's, suggesting that survival times may be longer than a week [1,2]. For example, in a trial involving extreme feed and water deprivation in broilers aged 0, 1, 4 and 8 weeks¹ [1], average survival times were, respectively, 5, 7, 16 and 23 days. Therefore, we set the lower and upper limit of survival at 5 and 25 days, respectively. The intervals describing the duration of each temporal segment in Pain-Track 1 are purportedly wide to accommodate heterogeneity in environmental conditions and in the age, size and metabolism of individuals, all of which affect the rate of water and body mass loss. The hypotheses for the intensity of the pain experienced at each interval are described and justified in Table 1.

¹ Such a trial would not be allowed nowadays for ethical considerations.

Pain Track 1. Hypotheses on the temporal evolution of pain associated with fatal cases of dehydration in birds. The column on the right shows the estimated time in pain of each intensity as a result of this experience.

	I. <2% mass loss	II. 2-5% mass loss	III. 5-10% mass loss	IV.10-15% mass loss	V.>15% mass loss	Cumulative Pain in hours (SD)
Excruciating						
Disabling			50%	70%		17.8 (3.0)
Hurtful	10%	70%	50%	30%	50%	148.3 (99.9)
Annoying	90%	30%			50%	117.5 (99.8)
	1-6 h	10-24 h	40-54 h	20-30 h	1-400 h	

Box 1. Pathophysiology of Severe Dehydration

At the initial stages of water deprivation, plasma Na⁺ concentration (osmolality) is kept constant at the expense of blood volume. If deprivation lasts longer, osmolality increases since the decrease in blood volume is blunted to protect the heart against the increased blood viscosity. The reduction in blood volume eventually leads to arterial hypotension. Compensatory mechanisms promote peripheral vasoconstriction, increased cardiac contractility and tachycardia, which in turn increases myocardial oxygen demand which, together with reduced tissue perfusion, may lead to myocardial failure. Finally, the anaerobic metabolism triggered in response to reduced perfusion may produce acidosis and, together with myocardial dysfunction, contribute to multiorgan failure [3].

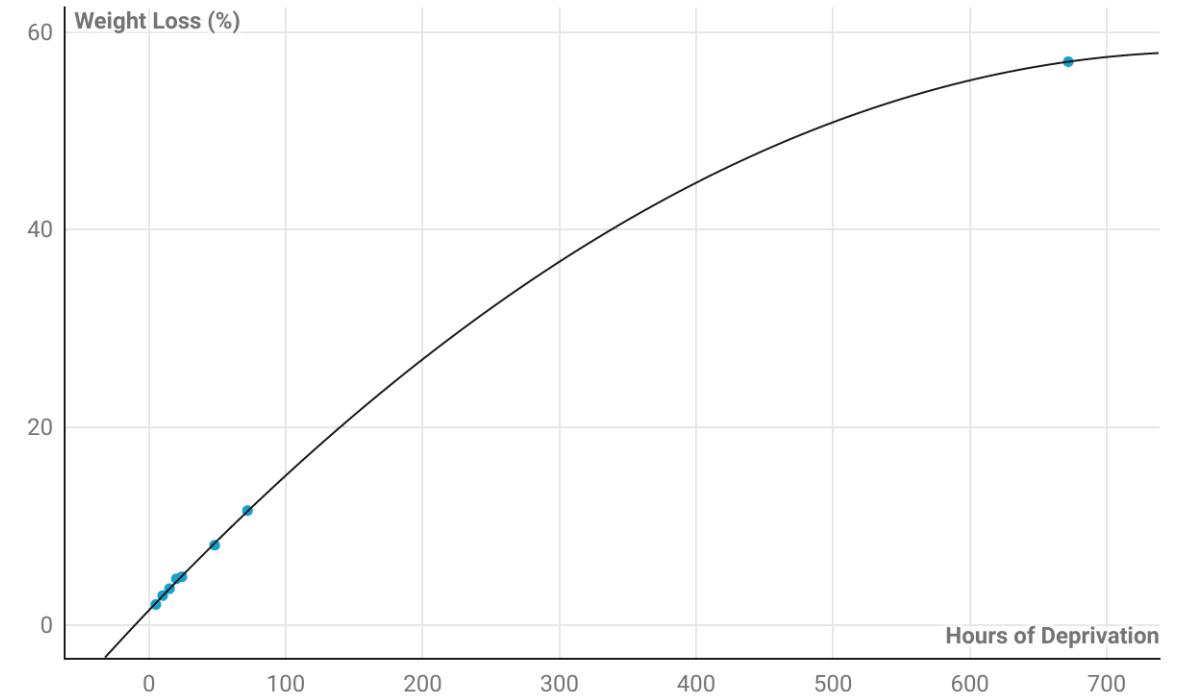


Figure 1. Mass loss (%) as a function of time of feed and water deprivation in 8-week old broilers at 27 °C [1].

Table 1. Justification for hypotheses of pain intensity associated with fatal cases of dehydration, as described in Pain-Track 1. (A) <2% mass loss; (B) 2-5%; (C) 5-10%; (D) 10-15%, (E) >15% mass loss .

(A)	In general, dehydration becomes noticeable only after 1-2% of loss of water volume. With dehydration levels lower than 2%, subjects may experience some thirst and discomfort (an adaptive mechanism to ensure that animals seek and consume water [4]), but the sensation is likely to be mild, not preventing individuals from conducting their normal routines. In the first few hours of water dehydration, we therefore assume that the degree of aversiveness associated with water deprivation is thus most likely to fit the Annoying criteria of pain.
(B)	As the volume of water loss increases, symptoms become more severe, with heart and respiration rates increasing to compensate for the lower plasma volume and blood pressure [5]. From an evolutionary perspective, increasing levels of discomfort and unpleasantness are expected. Water is possibly the most critical of all survival needs. As with food, attending to this drive should take precedence over other motivations, as failure to do so can very impair biological functioning. Thus, it is unlikely that after some hours of water deprivation this is a sensation that can be ignored. In laying hens, a few hours of water deprivation leads to frustration-induced aggression towards a subordinate hen [120]. Thus, we tentatively estimate that the intensity of the unpleasantness at this stage is most likely of a Hurtful intensity.
(C)	With over 5% water loss, human patients can become sleepy, experience nausea, and may feel tingling in their limbs. In birds, the stress of going without water might also trigger a molt. In an experiment involving water deprivation in White Leghorn hens, changes in the electrocardiogram (particularly tachycardia) started 24 hours after the onset of water deprivation [122]. Temperature also dropped and blood cholesterol significantly increased. The amplitudes of the ECG components continued to increase daily as weight loss and dehydration became more intense. Accordingly, we assume that the unpleasantness of the sensation may reach a Disabling level with at 50% probability. Annoying pain is unlikely at this stage.
(D)	If greater than 10% of body weight is lost in water in humans, cardiac output is diminished, and oxygen delivery to the tissues falls [123]. Some authors have suggested that most birds would require veterinary care when reaching 10% weight loss due to dehydration [124]. When this increases to 15%, muscle cramps may be experienced, vision can be compromised and urination is reduced and may become painful. Studies report that 40% of humans experience muscle cramps following dehydration, which are considered to have pain intensity between moderate and severe [125]. We estimate that when dehydration surpasses 10% of body weight lost in water, the likelihood of Disabling pain increases to 70%.
(E)	Although there have been suggestions that a loss of between 15-25% of the body water in mammals and birds [121,126] would be lethal, earlier experiments with chickens report higher figures. For example, a 45% loss of body water prior to death due to water deprivation has been reported in birds from 8 to 18 weeks of age [127]. Likewise, in trials with chickens (0 to 8 weeks of age) deprived of feed and water, a maximum weight loss of 57% prior to death was observed [117]. Evidence of the pathophysiology of extreme dehydration in humans suggest, however, that pain intensity may in fact subside in this last stage. In rats, severe dehydration has been shown to cause a significant decrease in pain perception, probably as a consequence of alterations in the metabolism of dopamine and serotonin [128]. Pain and discomfort can be also moderated by the accumulation of ketones in the body, as the body uses fat instead of carbohydrates to satisfy its energy needs. This process generates a form of self-sedation, the causes of which are still unknown but may be related to the metabolism of central neurotransmitters [129]. At this stage we therefore assume no Disabling pain, and distribute the likelihood of pain among the Annoying and Hurtful.

Death by Exsanguination due to Neck Blood Loss

In poultry, neck-cutting by severing both carotid arteries and external jugular veins leads to rapid blood loss: when severing is effective, high pressure jets of blood should flow for approximately 5-10 seconds [6]. The interruption of the supply of oxygen results in progressive brain dysfunction, eventually leading to loss of consciousness and brain death.

In humans, neck-cutting by a sharp blade is deemed as one of the fastest methods to induce brain death [7,8]: severing of the carotid by slashing results in death in approximately 5-15 seconds [7,9]. In cattle, evidence indicates that it will take 10 seconds up to over 3 minutes for animals to lose consciousness after the cut [10]. In broiler chickens, evidence based on EEG activity and standing posture [11] provide an idea of expected times to loss of consciousness following exsanguination. In one experiment, the severing of both carotid arteries and jugular veins of the neck lead to a quiescent electroencephalogram (EEG) within approximately 15 – 30 seconds [12]. Several factors may, however, modulate the rate of blood loss hence these times. In those cases where not all vessels are severed, bleeding will take longer, extending the time to the onset of unconsciousness [6]: in birds with only one carotid artery and one jugular vein severed, a quiescent EEG may take four times longer, from 1 to 2 minutes, to be achieved [12]. To reflect these heterogeneities, our hypothesis for the onset of unconsciousness encompasses the range of times reported in the literature for poultry, and includes both the complete and incomplete severing of the arteries and veins (5-120 s).

Intensity of experience

It is widely acknowledged that neck incision or puncture is painful [11,13–15] (it has been, in fact, the recognition of this painful nature that led to legislation on the need of stunning before bleeding). Bleeding to death is not necessarily painful by itself. It is the injury resulting in bleeding or, in the case of internal hemorrhages, the expansion of internal cavities, that causes pain [16]. Since the onset of death is relatively fast, cutting and bleeding are grouped into a single phase in which the intensity of pain remains invariable while it lasts, before consciousness is lost [8].

In humans, anecdotal reports by stabbing survivors [17] suggest that cuts with sharp blades are hardly painful in the first seconds after the injury is inflicted. Next, the trauma caused during neck cutting triggers a local inflammatory reaction on the skin, muscles, vessels, and peripheral areas, producing hyperalgesia at the site of incision and secondary hyperalgesia in the area surrounding the cut [18]. Pain caused by incisions, or “incisional pain”, has been widely studied in the context of surgical procedures and is known to increase with time in the absence of analgesia [18]. Neck incision has also been proven to be painful in anesthetized calves during the 30 seconds following incision, based on EEG responses [19,20]. Given the large extent of cuts associated with death by blood loss, we assume that incisional pain before loss of consciousness is predominantly Disabling (70%). Still, some authors argue that clean incisions may minimize pain and distress [21]. We therefore consider a 20% likelihood of Hurtful pain and a 10% likelihood of Annoying pain.

Pain-Track 2. Hypothesis for the intensities and temporal evolution of the pain associated with the incision of vessels and carotids in conscious birds and loss of consciousness by blood loss. The column on the right shows the estimated time in pain of each intensity as a result of this experience.

	Incisional pain	Cumulative Pain (sec)
Excruciating		
Disabling	70%	43.75 (40.25)
Hurtful	20%	12.5 (11.5)
Annoying	10%	6.25 (5.75)
	5 - 120 sec	

Death by Fatal Cannibalistic Attacks

Under commercial conditions, the strong motivation to forage and ground peck is typically not fulfilled properly, giving rise to the emergence of so-called abnormal behaviors, or behaviors that serve no function and are not as prevalent in their wild counterparts. One such behavior is injurious pecking, an umbrella term used for pecking behaviors that are re-directed towards flock mates, in which the target are their feathers (commonly referred to as ‘feather pecking’) and tissues (in this case, the terms ‘tissue pecking’ and ‘cannibalistic pecking’ are used, or ‘vent pecking’ when the targeted area is the vent). Injurious pecking occurs to varying degrees and compromises the welfare of affected birds in multiple ways.

There are different forms of feather and tissue pecking, each with different consequences for the affected bird’s welfare [22]. Here, we estimate the cumulative pain associated with ‘Severe tissue pecking’ (forceful pecks on bald patches of the skin of the victim, leading to bleeding of skin and underlying tissues). Tissue pecking can ultimately result in victim death due to excessive blood loss and serious tissue damage. In the case of vent pecking, which is the most common cause of fatal cannibalism in hens, pecking starts at the cloaca, surrounding skin and tissues, and can progress to evisceration and death. We refer to fatal tissue pecking events as fatal cannibalistic attacks. The hypotheses for the temporal evolution of the pain associated with pecking leading to a fatal cannibalistic attack are exposed in Pain-Track 3.

Pain-Track 3. Proposed temporal evolution of the pain associated with vent pecking leading to a fatal cannibalistic attack. The column on the right shows the estimated time in pain of each intensity as a result of this experience.

	Initial pecks (before immobility)	Pecking attack (immobile phase)	Cumulative Pain (minutes)
Excruciating		30%	4.5 (1.5)
Disabling	60%	40%	7.3 (2.3)
Hurtful	40%	30%	5.4 (1.7)
Annoying			
	0.3 - 4 min	5 - 15 min	

Unfortunately, ethological information on cannibalistic attacks is extremely scant, so we base our assessment on the following descriptions: (1) “*When attacking a victim, the cannibal typically approached from behind with the head slightly lowered. The pecks were vigorous and directed at the wound. The pecked bird usually vocalised and moved away, or was aggressive towards the pecking bird. If the pecked bird did not escape or show aggression, the cannibal might continue to peck and, if the victim moved, she was often followed*” [23]; (2) “*Cannibalism has been observed as starting with the pecking of fresh wounds in the back or excretory channels of a hen by nearby birds. The attacked birds are usually silent and make little attempt to escape. Pecking often continues until part of the intestine is obtained. **Death follows, usually in 10 minutes**, but pecking of the corpse continues*” [24].

These descriptions are different in that, in the first, victims vocalize vigorously and move away, whereas in the second attacked birds make little attempt to escape. Leaving aside differences in personalities (modulated by genetic and/or environmental factors), we assume that targeted birds initially react to the attackers (i), but then surrender if the attack continues. Chickens are known to react to a number of fear- and pain-eliciting stimuli by going into a state of complete motor paralysis, and it is not different with feather and tissue pecking events.

There is some evidence that chickens may experience reduced pain while in this paralysis state [25]. In one study [26], electroencephalogram readings showed a characteristic high-amplitude low-frequency (HALF) activity during this period of immobility similar to that seen in sleep and in catatonic states like tonic immobility. A similar pattern has been reported for general anesthesia in humans, where a progressive increase in HALF activity is observed as anesthesia deepens [27]. These observations would suggest that chickens may not experience pain, or may experience reduced pain, when immobile in the later stages of a cannibalistic attack [25]. In general, the altered electroencephalogram patterns persist for short intervals after the triggering event, alternating with periods of normality [26]. This is likely an adaptive response to ensure the animal has the possibility of regaining awareness of the situation in order to escape from an attacker, but it may also indicate that analgesia may be frequently interrupted during an attack.

Analgesia in situations of tonic immobility has been reported for other species [28,29]. In guinea pigs, tonic immobility has been shown to induce endogenous analgesia involving opioid synapses, which increased their threshold to noxious stimuli such as heat [30]. In fact, analgesia induced by stress and severe pain has been documented in many species (including humans [31]). In addition and presumably as a mechanism to protect the organism from being overwhelmed by pain during dangerous situations, analgesia has also been observed in situations such as uncontrollable footshock, restraint, inescapable swimming in cold water and body pinching [32].

Despite evidence to suggest that chickens have a remarkable ability to suppress intense pain [33], conclusive evidence as to whether tonic immobility has analgesic effects in chickens is still lacking. Additionally, it is not possible to completely rule out the possibility that the immobility observed in victims of cannibalistic attacks emerges as a result of tonic immobility or learned helplessness [25], a phenomenon in which an animal exposed repeatedly to an inescapable stressor gives up reacting, with no attempts of withdraw or protection, even when escape becomes possible. Thus, the possibility that pecking attacks are extremely painful for victims cannot be completely discarded.

The hypotheses of pain intensity described in Pain-Track 3 (immobile phase) consider two possibilities: (1) that pain of lower intensity (hurtful) is experienced while immobile for some time (30%), but interspersed with periods of disabling pain (based on the observation altered electroencephalogram patterns persist for short intervals after the triggering event, alternating with periods of normality), and (2) that immobility is associated with learned helplessness, hence pain is experienced at very high levels of pain intensity (hence a 30% probability of excruciating pain).

Death by Sepsis (as a result of severe infections)

In Pain-Track 4 we propose a tentative scheme for the temporal evolution of the pain associated with the septic form of a bacterial infection in the abdomen (a typical infection in laying hens and other

egg-laying females), partitioned into five time segments. The hypotheses for the intensity and duration of each of the segments proposed in Pain-Track 4 are described and justified in Table 2.

Pain-Track 4. Hypothesis for the temporal evolution of the pain associated with the fatal (septic) form of acute bacterial peritonitis. The column on the right shows the estimated time in pain of each intensity as a result of this experience.

	Inflammation	Sepsis (initial symptoms)	Severe sepsis (organ failure)	Septic shock	Cumulative Pain (hours)
Excruciating			30%		2.25 (0.75)
Disabling	20%	90%	40%	10%	131.5 (67.4)
Hurtful	70%	10%	30%	80%	398.5 (235.2)
Annoying	10%			10%	74.3 (35.1)
	2 - 8 weeks	12 - 24 hrs	5 - 10 hrs	2 - 4 hrs	

Table 2. Justification for the hypotheses of pain intensity and duration associated with fatal cases of sepsis, as described in Pain-Track 4: (A) inflammation; (B) sepsis; (C) severe sepsis; (D) septic shock .

(A) Once infected fluids build up, inflammation and swelling of the abdomen ensues, associated with diffuse abdominal pain [34]. Other symptoms at this phase include fluffed feathers, dyspnoea, lethargy, lack of vocalizations, depression, lameness, anorexia and swollen vent [35], which would suggest that the the pain most likely endured is at least of a hurtful nature, although possibly interspersed with bouts of higher pain intensity (represented by the 20% likelihood of Disabling pain) as based on the severity of these symptoms (alternatively, these percentages may be interpreted as the temporal variation in the levels of pain over the period). Anecdotal reports by backyard hen keepers [36,37] suggest that inflammation may last up to two 8 weeks until development of the initial symptoms of sepsis.

(B) In human medicine, sepsis is defined as the manifestation of a systemic inflammatory response in the presence of infection. Clinical diagnosis of sepsis requires finding a focus of infection that is accompanied by at least two signs of systemic inflammation, which include fever, tachycardia, tachypnoea and high white blood cell counts. However, in poultry, the clinical diagnosis of sepsis is not well-defined [38]. Progression from sepsis to severe sepsis (when sepsis is accompanied by major organ failure) has been well established in humans. Still, direct extrapolation of temporal parameters from humans to poultry is made difficult by the employment of several treatment strategies in humans, which affect the time course of the disease. Therefore, we rely predominantly on models of sepsis in rodents. These models suggest that death due to sepsis, including the development of severe sepsis and septic shock, takes 24 to 48 hours, from the onset of symptoms until death following septic shock. This estimate is also in line with observations that human patients with severe sepsis do not take long to reach septic shock, usually 48 to 72 hours (Dr. Pedro Salinas, MD, *personal communication*), even those who receive some therapeutic support. Without treatment, the time is bound to be shorter. In animal models of sepsis, systemic and local inflammation is often regarded as a major source of pain [39]. In a study that inoculated mice with fecal flow in the abdominal cavity to induce peritonitis and sepsis, the first signs of sepsis included hunched posture, piloerection, lethargy, and diarrhea, with death occurring after 48 hours. In another model where sepsis was induced by an intraperitoneal injection of stool, 75% of mortality occurred within 24 hours [40]. During the first 12 hours, septic mice progressively manifested symptoms that included increasingly labored breathing and minimal response to auditory and tactile stimuli, indicating the disabling nature of the pain. The CDC lists "extreme pain or discomfort" as a potential sign of sepsis, with an estimated 70% of patients developing peripheral neuropathy (when peripheral nerves are damaged), which often results in intense pain [39]. Intravenous morphine is routinely used for pain relief, providing an indication of the level of pain endured. Sepsis also involves changes on endothelial tissues and microcirculation, primary and secondary immune tissues, coagulation, parenchymal tissues and neurological disturbances that directly affect microglial cells and neurons [41]. Furthermore, systemic inflammation has been linked to generalized hyperalgesia [39], hence the high likelihood that pain is experienced at a Disabling level at this phase.

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- (C) The term 'severe sepsis' denotes sepsis that is accompanied by major organ failure, whereas 'septic shock' refers to the added complication of unresponsive cardiovascular collapse [38]. Sepsis-associated inflammation at this stage has been linked to pulmonary, cardiovascular, renal, hematological, hepatic and neurological dysfunction. In broiler chickens, septicemic carcasses are frequently associated with hemorrhages on the heart, liver, kidneys, muscles and serous membranes. The kidneys, liver and spleen are often swollen and with excess blood. Muscles are also compromised (e.g., *"Very few birds with septicemia survive catching and transportation to make it to the processing plant alive. If the bird prior to catching has had a septicemia event, often the muscles have wasted away and have broken down because of changes in muscle metabolism"* [42]). In humans, this stage is marked by severe muscle pain, described as extreme (*"I feel like I might die"; "My sepsis felt like my body was on fire, burning from the inside out"*) [43,44]. Therefore, we assume that pain is Excruciating for 30% of the time during this phase (we consider that longer durations would not be possible without the neurological system shutting down). On the other hand, the pathophysiologic consequences of severe sepsis may reduce pain perception. For instance, sepsis-associated encephalopathy is implicated in brain damage of regions related to nociceptive modulation [45]. Because the resulting decrease in cognitive and nociceptive sensitivity may attenuate pain responses [38], we attribute the same likelihood (30%) to a less intense (Hurtful) category. The remaining proportion is allocated to the intermediate intensity category. The homogeneous spread of probabilities in three categories of pain intensity reflects not only the likely heterogeneity of the phenomenon, but also the current lack of knowledge about its manifestation at the individual level.
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- Individuals who fall into septic shock usually die within hours. In humans, a maximum of one day has been reported [41], although this time is frequently shorter than 12 hours [46]. In both humans and animals, multiple organ failure will be associated with severe refractory hypotension, which leads to rapid death [41]. At this stage, brain inflammation is believed to profoundly alter the local synaptic circuitry, resulting in reduced nociception [45]. During sepsis, especially in septic shock, there is a deterioration of the small nerve fibers responsible for the transmission of pain, reducing the ability to experience pain [47]. A similar phenomenon, referred to as critical illness polyneuropathy, was also described, with up to 70% of human patients in septic shock having their ability to feel pain affected [48]. In general, there seems to be a general reset of the pain pathways when the end is near [49]. Very few patients in septic shock, around 10%, maintain their state of consciousness unaltered until a few minutes before dying. In these cases, the cause of death is related to multiple organ dysfunction without neurological compromise. Accordingly, we consider that pain is more likely felt at the Hurtful level during this phase, with few individuals (10%) able to feel pain at a higher intensity. We also consider the possibility that, due to the impairment reported, some individuals only experience an annoyance (10%).
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