The Safe and Effective Use of Pharmacological Agents Used for Sedation During Radiological Procedures

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ABSTRACT: **The use of pharmacological agents to decrease the pain and anxiety associated with the performance of invasive and noninvasive radiological procedures is well documented. The use of such agents is also increasing. This article is intended both as a review for the nonanesthesia professional administering these agents and a primer for those who wish to administer these agents. The physiological mechanisms by which anxiety occurs, and is modulated, are discussed, as are those surrounding pain. A discussion of the continuum of consciousness and sedation is discussed. Individual agents are reviewed, along with their strengths, weakness, warning, and contraindications. Finally, the safety and monitoring of the sedated patient is reviewed. (J Radiol Nurs 2014;33:132-144.)**

KEYWORDS: **Conscious sedation; Pharmacological agents; Anxiety; Pain; Safety.**

INTRODUCTION

The ability to obtain a good quality radiological image is an important component of a patient's workup and diagnosis. In addition, safely performing an invasive procedure in the radiology department can be an important component of patient's treatment plan as well. Both obtaining images and performing procedures can be a challenging task in the radiology nurse's patient populations. The patient can experience pain, fear, anxiety, or a combination of all the three. The use of pharmacological adjuncts, such as anxiolytic or pain medications, to make the patient's experience of these tests or procedures less stressful is an accepted and continuously growing part of the radiological nursing profession.

PHYSIOLOGY OF PAIN, FEAR, AND ANXIETY

The physiology of how people feel, experience, perceive, and respond to pain, fear, and anxiety is a very complex topic. Fear and anxiety are similar emotions and are quite often used interchangeably. Fear can be described as a response to an actual threat or danger, whereas anxiety can be described as a response to a perceived threat and tends to evolve over time from one's past experiences. Whether the threat is real or perceived, the physiological response is similar, in that, it involves psychological and physical responses that can lead to an alarm reaction, a significant part of which is increased sympathetic autonomic activity (Fear Anxiety, 2013). The threat, as well as the reaction, can even be conscious or subconscious in nature (Huang, Wun, & Stern, 2011).

Whether the threat that a patient experiences is real fear or perceived anxiety, the amygdala is the area of the brain that is stimulated (Huang et al., 2011). The amygdala is a part of the limbic system, deep within the brain, and is responsible for managing a person's emotions, such as the response to fear (What causes

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anxiety?, 2011). The amygdala receives information by way of nerve signals from a variety of locations in the brain such as the thalamus, sensory cortices, prefrontal cortex, and hippocampus (Figure 1). Each of these areas has a specific purpose for sending signals to the amygdala. The thalamus receives sensory information from the peripheral and cranial nerves, which will then direct the information to the prefrontal cortex and the amygdala (Holt, Ongur, Wright, Dickerson, & Rauch, 2008). Signals that are sent to the prefrontal cortex are evaluated there and then a rational judgment can be made about the information. Signals sent from the thalamus directly to the amygdala produce a much faster basic response (What causes anxiety?, 2011). Once the amygdala is activated, outgoing neurophysiological signals are sent to many locations in the brain, including the motor cortex, basal ganglia, hypothalamus, and brainstem. These regions will then begin the physical reactions to these potentially threatening stimuli with which all of us are familiar. Emotional responses, such as fear, will activate the sympathetic nervous system, also known as the "flight or fight" response (Holt et al.). This activation of the sympathetic nervous system is because of the stimulation of the hypothalamus and will lead to effects such as increased blood pressure, blood glucose, muscle strength, and mental activity (Hall, 2011). This emotional response pathway is quick and often occurs in the subconscious (Holt et al.).

The afferent signals that the amygdala receives from the sensory cortices, prefrontal cortex, and hippocampus are more developed, conscious, and thought out responses to sensory input. The hippocampus is responsible for memories. The signals transferred between the hippocampus and the amygdala help form memories from previous experiences of fear and provide the emotional response that is brought about from remembering a previous experience (Holt et al., 2008). An example would be a child beginning to cry simply walking into the hospital for a test because of a memory of an unpleasant experience at the hospital.

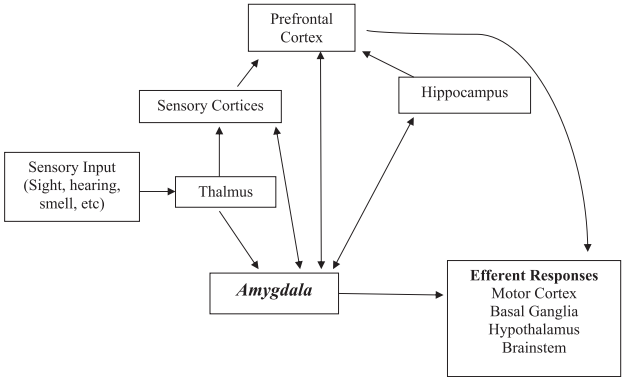

Figure 1. Amygdala afferent and efferent signals. The figure shows the graphical representation of the how the amygdala, part of the limbic system, sends and receives signals from a variety of areas to process and respond to fear and anxiety (Holt et al., 2008; What causes anxiety?, 2011).

There are many neurotransmitters that are involved in the transmission of fear and anxiety. Some of the more well understood neurotransmitters are γ-aminobutyric acid (GABA), glutamate, dopamine, norepinephrine, and serotonin (What causes anxiety?, 2011). GABA functions as an inhibitory neurotransmitter and prevents or decreases the transmission of nerve signals. GABA works on the $GABA_A$ receptor site on a variety of neurons in the brain. Glutamate, as precursor of GABA, is the major excitatory neurotransmitter in the central nervous system (CNS; Hall, 2011). Receptors that glutamate binds with include the *N*-methyl-D-aspartate (NMDA) and α-amino acid-3-hydroxyl-5methyl-4isozazole propionic acid (AMPA) receptors (Nagelhout & Plaus, 2010).

Each $GABA_A$ receptor has multiple binding sites, and as the neurotransmitter GABA binds to the site, the receptor opens and allows the anion, chloride, to enter into the cell. As chloride ion levels increase inside the cell, the ability of the nerve to send a signal is diminished as the negative ion pushes the membrane potential farther from threshold, making it harder for the nerve to "fire" (Nagelhout & Plaus, 2010). Many of the medications that are used as anxiolytics, such as benzodiazepine and barbiturates, work by binding to $GABA_A$ receptor sites and are considered GABA agonists or agents which make the GABA receptor work to allow more chloride ion entry into the cell. Although binding to different areas of the receptor, the result is similar, in that, once a benzodiazepine or barbiturate binds, the effect of GABA is enhanced. Propofol, etomidate, and chloral hydrate work by binding to the $GABA_A$ receptor as well (Brunton, Lazo, & Parker, 2006).

NMDA and AMPA receptors are excitatory when activated by glutamate or another agonist and work together to transmit signals. NMDA and AMPA receptors are nonselective cation channels and when activated allow cations, such as sodium and calcium, to enter the cell leading to signal transmission (Brunton et al., 2006). As these positive ions enter the cell, it moves closer to threshold, making it easier for the cell to fire and send an impulse.

Ketamine is an NMDA receptor antagonist: In other words, it prevents glutamate or other agonists from working. Once bound to NMDA receptors in the brain, ketamine blocks signals to the thalamus and cortex. Sensory stimuli are able to reach the cortical regions of the brain but cannot be interpreted, leading to diminished responses and altered sensorium (Nagelhout & Plaus, 2010).