Vasovagal Reactions During Interventional Pain Procedures

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Fact: The overall incidence of vasovagal reactions (VVR) ranges from 1-8% during interventional pain procedures, though certain patient populations may be at greater risk. Younger age, male sex, and a history of a VVR are associated with an increased likelihood of VVR. In select patients, moderate sedation may be considered for prevention of a repeat vasovagal reaction.

Psychological distress or a noxious stimulus can trigger a vasovagal reaction that causes bradycardia, hypotension, and a reduction in vascular tone [1,2]. Vasovagal reactions (VVRs) typically present as dizziness, a sense of warmth or flushing, diaphoresis, nausea, blurred vision, or loss of vision. Other less common symptoms may include tinnitus, chest discomfort, weakness, yawning, or anxiety [3,4]. An extreme VVR is vasovagal syncope, in which the patient loses consciousness, usually within two minutes of onset of symptoms [5] and may even convulse.

VVRs can occur as an adverse event during interventional procedures for spine pain, with incidence rates ranging between 1.1% - 8% [6-8]. A study of more than 8,000 patients found that male sex, age (18-35), and pre-procedural pain score (<5/10) were associated with increased risk of VVR [6]. Another study reported greater likelihood of a VVR associated with cervical compared to lumbar spine injections (8% vs. 1%, respectively) [7]. A patient with a history of VVR during an interventional spine procedure is more than 7-times more likely (23% vs 3%) to experience a VVR during a subsequent procedure [9].

Preprocedural Considerations

Monitoring
Patient monitoring (pulse oximetry, non-invasive blood pressure monitoring, pulse) pre-, intra-, and post-procedure can help identify early bradycardia or hypotension. If a patient experiences a VVR during an interventional pain procedure, this information can assist the physician in determining the extent, duration, and management of the response. It is critical to differentiate VVR from other conditions such as cardiac syncope, allergic reaction, and high spinal block.

Moderate Sedation in Patients with Documented History of VVR
A 2015 study reviewed 134 interventional procedures performed for patients with a history of previous VVR [9]. Of these, 90 procedures were performed without moderate sedation and 21/90 (23.3% [95% Confidence Interval (CI) 15.2-32.1%]) were complicated by a repeat VVR. Conversely, none of 44 repeat injections performed with moderate sedation resulted in a repeat VVR [0% (95% CI 0-9.6%)] (χ2 = 12.17, P < 0.00048). In fact, in this entire cohort, there were no VVRs in any patients, regardless of previous history, when moderate sedation was used. Therefore, in patients with a history of VVR, the use of moderate sedation may play a role in the prevention of repeat VVR.

Psychological Distress
Psychological distress can trigger VVR [1,2]. Although the mechanism of the psychological trigger is unclear, strategies should be implemented to reduce anxiety, including informing patients about what they can expect during the procedure and designating a care team member to attend to the patient during the procedure.
Management Considerations

**Patient-Performed Physical Counter-maneuvers**
In both non-randomized and randomized trials, isometric muscle contractions have been shown to increase cardiac output and mean arterial blood pressure (MAP) while decreasing syncope occurrence [10,11]. The most effective maneuver combines leg crossing and buttocks clenching, but improvement in MAP can also occur with arm contractions. This effect seems to be mediated largely by the sympathetic nervous system increasing vascular resistance during the maneuvers, as well as mechanical compression of the venous vascular beds in the legs and abdomen. Unfortunately, such maneuvers may not be practical for use during the performance of an interventional pain procedure in which the patient is most often prone, and significant movement while a needle is in place is not advisable. However, these maneuvers could be useful once the needle is removed.

**Trendelenburg Position vs. Passive Leg Raise**
Trendelenburg position, in which the patient is placed at an angle with the feet above the head may be used to treat early symptoms of VVR. However, this maneuver does not appear to have a substantial effect on the vascular system; fifteen degrees of tilt in healthy normovolemic patients resulted in only 1.8% central displacement of blood volume [12]. Another study suggested that there was no clinically significant change in cardiac output, cardiac index, MAP, systemic vascular resistance, and oxygenation with 10-30 degrees of Trendelenburg in critically ill patients [13].

In post-operative patients, Trendelenburg position has shown to increase MAP an average of 10.7mm Hg +/- 3.5mm Hg, but this may not be associated with an improvement in blood flow or oxygenation [14]. Others postulate that the fluid shifts that occur with Trendelenburg positioning may ultimately result in decreased cardiac output [15,16].

In the case of hypovolemia, a meta-analysis of 21 studies reported that Trendelenburg position increases cardiac output by 9%, or 0.35 L/min, and passive leg raise increases cardiac output by 6%, or 0.19 L/min [17] at one minute following the positional intervention. However, between 2-10 minutes post-positional intervention, only passive leg raise demonstrated sustained cardiac output benefits.

**Ammonia Inhalants**
Ammonia inhalants have traditionally been used as a means for arousing a patient that has lost consciousness. There is no literature pertinent to the use of such agents in the context of a VVR due to a medical intervention. Given that ammonia inhalants are a noxious stimulus, caution is warranted, especially in patients with respiratory comorbidities [18].

**Intravenous (IV) Fluids**
In cases where bradycardia leads to hypotension, if IV access is available, consideration can be given to administering a bolus of IV fluids as a means of increasing MAP [19].

**Ice Packs**
In many instances, patients respond to cooling measures to counteract the parasympathetic state of VVR. Physiologically, a hypothermic state will trigger the sympathetic system to increase cardiac contractility and stroke volume to restore cardiac output. Ice has traditionally been considered as a means for triggering this phenomenon. Cold stimulation to the lateral neck has been shown to increase heart rate variability [20].

**Summary and Recommendations**
- VVR can occur during interventional pain procedures. Differentiating this response from other conditions such as cardiac syncope, allergic reaction, or high spinal block is critical to its management.
- Younger age, male sex, and history of VVR are associated with an increased likelihood of VVR.
- Patient monitoring (pulse oximetry, non-invasive blood pressure monitoring, pulse) pre-, intra-, and post-procedure can help identify early bradycardia or hypotension.
- Many treatments historically used during VVR are supported by limited evidence. The magnitude of their effectiveness is unknown in the context of interventional pain procedures.
- The use of moderate sedation in select patients, particularly those with a history of VVR, demonstrates effectiveness in the prevention of VVR.
References