Background As consciousness decreases in the dying process, patients lose their ability to swallow and clear oral secretions. Air moves over the secretions, which have pooled in the oropharynx and bronchi, resulting in turbulence and noisy ventilation with each breath. This is often described as ‘gurgling or ‘rattling noises.’ While there is no evidence that patients find this ‘death rattle’ disturbing, evidence from bereaved surveys suggests the noises can be disturbing to the patient’s visitors and caregivers who may fear that the patient is choking to death. Similar sounds may occur in patients who are not imminently dying, such as in those with brain injuries or in disorders like Amyotrophic Lateral Sclerosis in which increased production or decreased clearance of secretions occurs. Two sub-types of the death rattle have been proposed, although the significance regarding treatment has not been established: Type 1 = predominantly salivary secretions and Type 2 = predominantly bronchial secretions. Death rattle is a good predictor of near death; one study indicated the median time from onset of death rattle to death was 16 hours.

Non-Pharmacological Treatments
- Position the patient on their side or in a semi-prone position to facilitate postural drainage
- A minute or two of Trendelenburg positioning can be used to move fluids up into the oropharynx for easier removal; aspiration risk is increased, however.
- Gentle oropharyngeal suctioning is used although this can be ineffective when fluids are beyond the reach of the catheter. Frequent suctioning is disturbing to both the patient and the visitors.
- Reduction of fluid intake.
- Communication with family and caregivers aimed to address associated fears and interpretations.

Pharmacological Treatments While multiple studies have questioned the utility of pharmacologic treatments for death rattle, muscarinic receptor blockers (anti-cholinergic drugs) are the most commonly used class of medication for this symptom. Such agents include scopolamine, hyoscyamine, glycopyrrolate, and atropine. All of these agents can cause varying degrees of blurred vision, sedation, confusion, delirium, restlessness, hallucinations, palpitations, constipation, and urinary retention. The primary difference in these drugs is whether they are tertiary amines which cross the blood-brain barrier (scopolamine, atropine, hyoscyamine) or quaternary amines, which do not (glycopyrrolate). Drugs which cross the blood-brain barrier are apt to cause CNS toxicity (sedation, delirium).

<table>
<thead>
<tr>
<th>Drug</th>
<th>Trade Name</th>
<th>Route</th>
<th>Starting Dose</th>
<th>Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>scopolamine (hyoscine) hydrobromide</td>
<td>Transderm Scop</td>
<td>Patch</td>
<td>One 1.5 mg patch</td>
<td>~12 h (24 h to steady state)</td>
</tr>
<tr>
<td>hyoscyamine</td>
<td>Levsin</td>
<td>PO, SL</td>
<td>0.125 mg</td>
<td>30 min</td>
</tr>
<tr>
<td>glycopyrrolate</td>
<td>Robinul</td>
<td>PO</td>
<td>1 mg</td>
<td>30 min</td>
</tr>
<tr>
<td>glycopyrrolate</td>
<td>Robinul</td>
<td>SubQ, IV</td>
<td>0.2 mg</td>
<td>1 min</td>
</tr>
<tr>
<td>atropine sulfate</td>
<td>Atropine</td>
<td>SubQ, IV</td>
<td>0.1 mg</td>
<td>1 min</td>
</tr>
<tr>
<td>atropine sulfate</td>
<td>multiple</td>
<td>Sublingual</td>
<td>1gtt (1% ophth. soln)</td>
<td>30 min</td>
</tr>
</tbody>
</table>

Pharmacological pearls
- Glycopyrrolate has five times the anti-secretory potency compared to atropine but is poorly and erratically absorbed orally. The clinical significance of this is unclear.
- The scopolamine patch releases ~1 mg over 72 hours. It takes 24 hours to reach steady state and for acute symptoms other drugs should be used. The patch should be placed on hairless skin just behind the ear, is changed every 72 hours, and more than one patch can be used at a time.
• Hyoscycamine is available in short-acting, sustained-released, orally dispersible tablet, and oral solution formulations.

References

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