

RECOGNIZE THE CRISIS *weakness that has reached the muscles of breathing or swallowing*

DEFINITION

Worsening myasthenic weakness causing **respiratory failure requiring ventilatory support**, or **severe bulbar weakness** threatening the airway. It is the presentation of roughly 15–20% of myasthenic patients at some point in their disease.

WARNING SIGNS

Bulbar failure — nasal speech, dysphagia, weak cough, pooling secretions, jaw weakness. Plus **orthopnea**, accessory muscle use, paradoxical abdominal movement, tachypnea, and a **falling single-breath count**. Anxiety and restlessness often precede the collapse.

FIND THE TRIGGER

Infection is the commonest · surgery · pregnancy or the postpartum period · **tapering immunosuppression** · **starting a precipitating drug** · and, importantly, **starting high-dose corticosteroids**, which can transiently worsen weakness before helping.

MONITOR AND SECURE THE AIRWAY *the single most important step*

- 1 **Admit to ICU or a high-dependency area** for anyone with bulbar involvement or a declining respiratory trend. **Early intubation and mechanical ventilation are perhaps the most important steps** in managing myasthenic crisis.
- 2 **Emergency intubation should be avoided.** Plan it. A crash intubation in a patient with bulbar weakness, retained secretions and no respiratory reserve is far more hazardous than a controlled one.
- 3 **Track FVC and negative inspiratory force serially**, alongside bulbar function and secretion clearance. As in Guillain-Barré, **oxygen saturation is a late sign** — hypercapnia precedes desaturation, so a normal SpO₂ is falsely reassuring.
- 4 **Non-invasive ventilation has a defined role** and may avert intubation in **selected patients without significant bulbar weakness or CO₂ retention**. It is **not appropriate where bulbar failure threatens aspiration**.
- 5 **Consider holding acetylcholinesterase inhibitors** while ventilated. They increase secretions and add little once the patient is supported, and stopping them removes any diagnostic confusion with cholinergic excess.

TREAT THE CRISIS *rapid immunotherapy plus the underlying regimen*

Therapy	Detail
Plasma exchange	Often preferred in crisis , with a faster onset of action — typically within days. Requires vascular access and causes hemodynamic shifts, so it is less suitable in septic or unstable patients.
IVIG	Comparable overall efficacy with an easier logistic profile and no need for central access. Preferred where there is sepsis, poor access, or hemodynamic instability. Watch for volume load, renal impairment, and thrombosis.
Corticosteroids	Started or increased for longer-term control — but expect a transient worsening in the first 1–2 weeks , which is why they are usually begun after or alongside plasma exchange or IVIG, and under close observation.
Treat the trigger	Culture and treat infection , correct electrolytes, and review every recently started drug. The crisis rarely resolves while the precipitant is active.

DRUGS THAT PRECIPITATE CRISIS *check this list before prescribing anything*

ANTIBIOTICS

Aminoglycosides · **fluoroquinolones** · **macrolides** · telithromycin. These are the commonest inadvertent precipitants, often given for the very infection that triggered the deterioration — so choose the antibiotic deliberately.

OTHER AGENTS

Magnesium (including obstetric infusions and laxatives) · **beta-blockers** · **calcium channel blockers** · **neuromuscular blocking agents**, to which these patients are exquisitely sensitive · iodinated contrast · procainamide · penicillamine · statins · and **immune checkpoint inhibitors**, which can cause a myasthenic syndrome with overlapping myositis and myocarditis.

CHOLINERGIC CRISIS *the classical mimic — rare now, but you must be able to name it*

HOW IT DIFFERS

Caused by **excess acetylcholinesterase inhibitor** rather than too little. Weakness looks identical, but there are **muscarinic features**: **miosis**, salivation and lacrimation, bronchorrhea, sweating, abdominal cramps and diarrhea, bradycardia, and fasciculations.

WHY IT IS UNCOMMON NOW

Modern dosing rarely reaches cholinergic toxicity. In practice, **the safe move in a ventilated patient is to stop the anticholinesterase**, which removes the ambiguity entirely and reduces secretions. **Edrophonium testing is largely historical** and is not needed to resolve this at the bedside.

WEANING AND AFTERWARDS *extubation failure is common — plan for it*

Stage	Detail
When to wean	Only once the immunotherapy has taken effect , the precipitant is treated, and secretions are manageable. Do not wean on the basis of oxygenation — it is muscle strength and bulbar function that determine success.
Restart anticholinesterase	Reintroduce before extubation , at a reduced dose, to support strength during the trial.
Extubation failure	Common , and often driven by secretion clearance rather than gas exchange. Anticipate it, and consider early tracheostomy where ventilation is prolonged, rather than repeated failed attempts.
Before discharge	CT chest for thymoma if not already done · optimize long-term immunosuppression · give the patient a list of drugs to avoid and a medical alert card · arrange neurology follow-up · vaccinate appropriately before escalating immunosuppression, and counsel on infection as the commonest trigger of the next crisis.

PEARLS & PITFALLS

- Intubate electively.** Waiting until the patient fails is the decision that turns a manageable crisis into an arrest.
 - Normal oxygen saturation is meaningless here.** Follow the FVC, the bulbar function, and the CO₂.
 - Check the drug chart first** — a quinolone or magnesium given for something else is a common and reversible precipitant.
- High-dose steroids transiently worsen weakness.** Start them under observation, usually after or with plasma exchange or IVIG.
 - NIV can work, but not with bulbar weakness** — the aspiration risk outweighs the benefit.
 - Screen for thymoma** with CT chest in every newly diagnosed patient, and consider thymectomy in appropriate generalized disease.