

# How to avoid stress induce ulcer ?

By: Farah Abdul Kareem

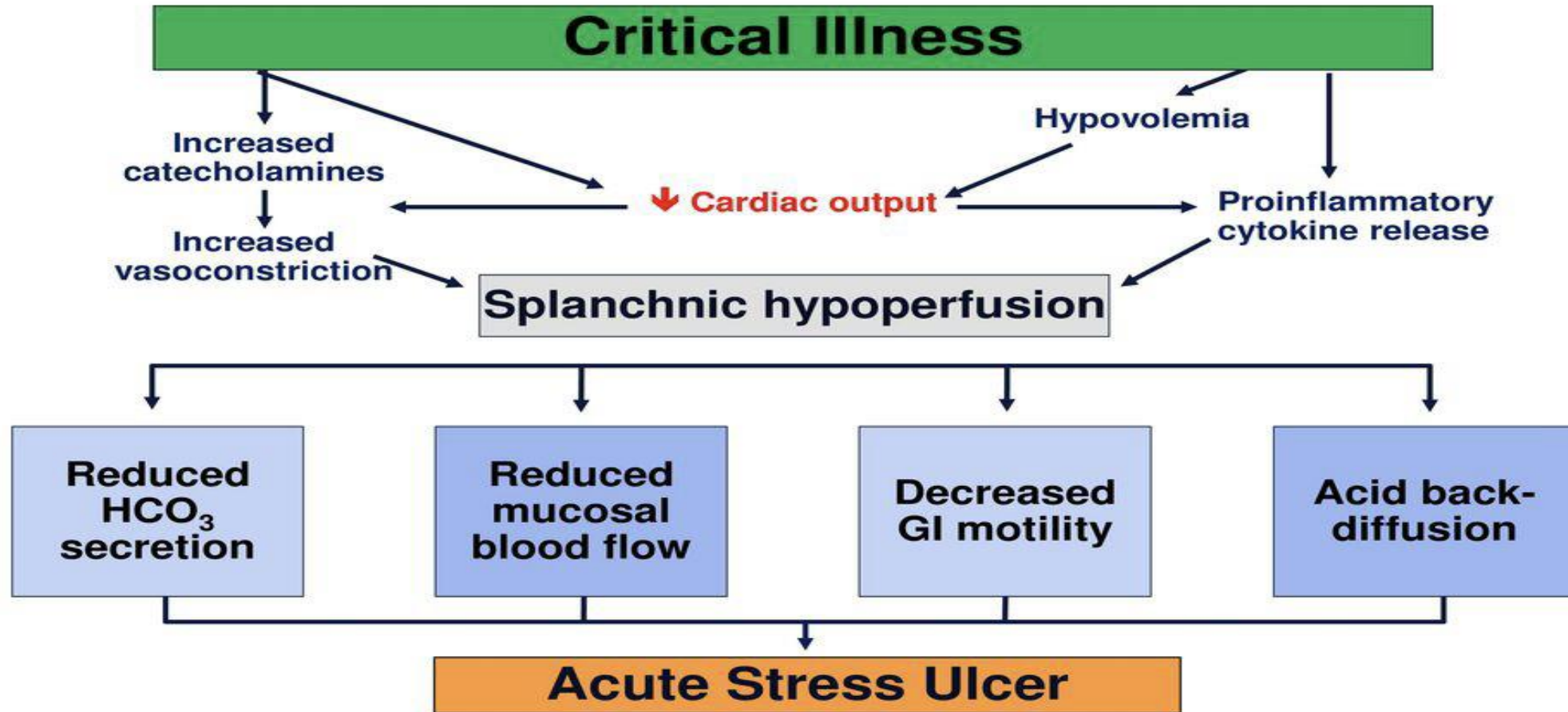


## Characteristics of SRMD

Multiple superficial erosive in upper GIT occur early in the course of critical illness and may progress to deep ulcers.

Stress ulcers are diffuse in nature and **do not respond to endoscopic therapy, they heal over time w/o intervention.**

# Pathophysiology of Stress Ulcers

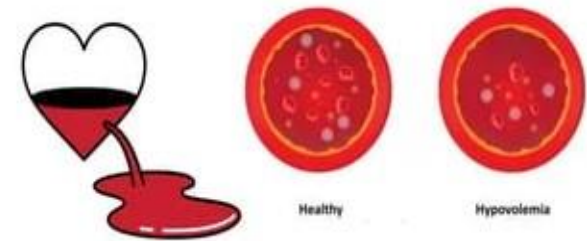


## Pathophysiology of SRMD

1. ↓ **gastric blood flow and mucosal ischemia are the primary causes of stress ulcer-related bleeding.**

**Reduced splanchnic blood flow is caused by mechanisms common to critical illness:**

- Hypovolemia
- Reduced cardiac output
- Proinflammatory mediator release
- Increased catecholamine release
- Visceral vasoconstriction



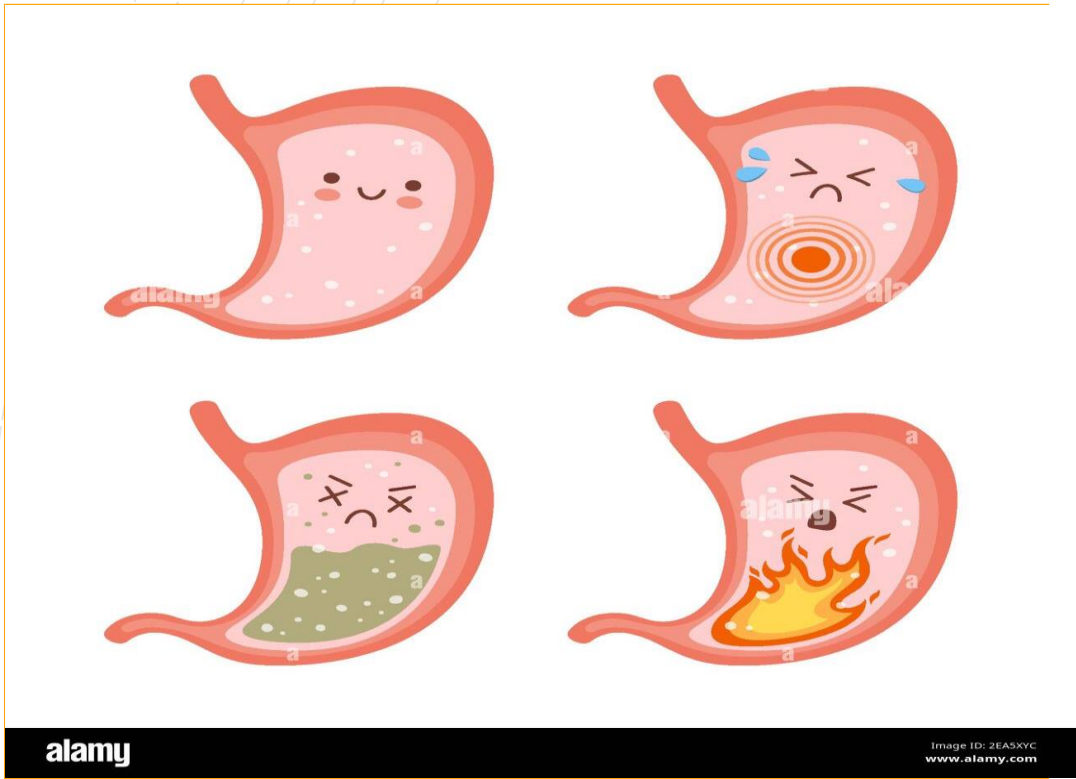
# Pathophysiology of SRMD

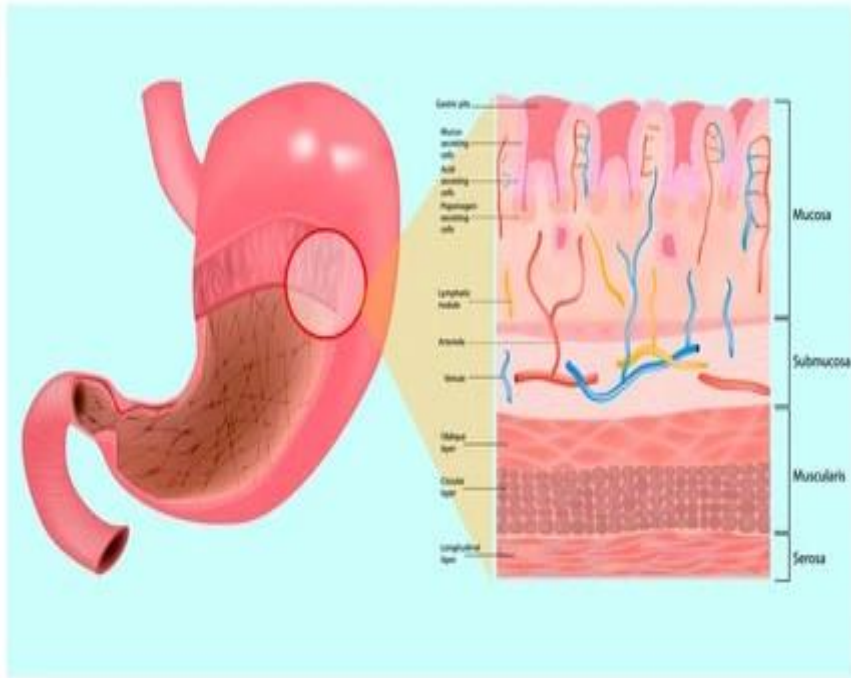
## 2- Additional factors leading to stress ulcer-related bleeding:

- Decreased gastric mucosal bicarbonate production
- Decreased gastric emptying of irritants and acidic contents

Acid back-diffusion

- Reperfusion injury that may occur after the restoration of blood flow after prolonged periods of hypoperfusion

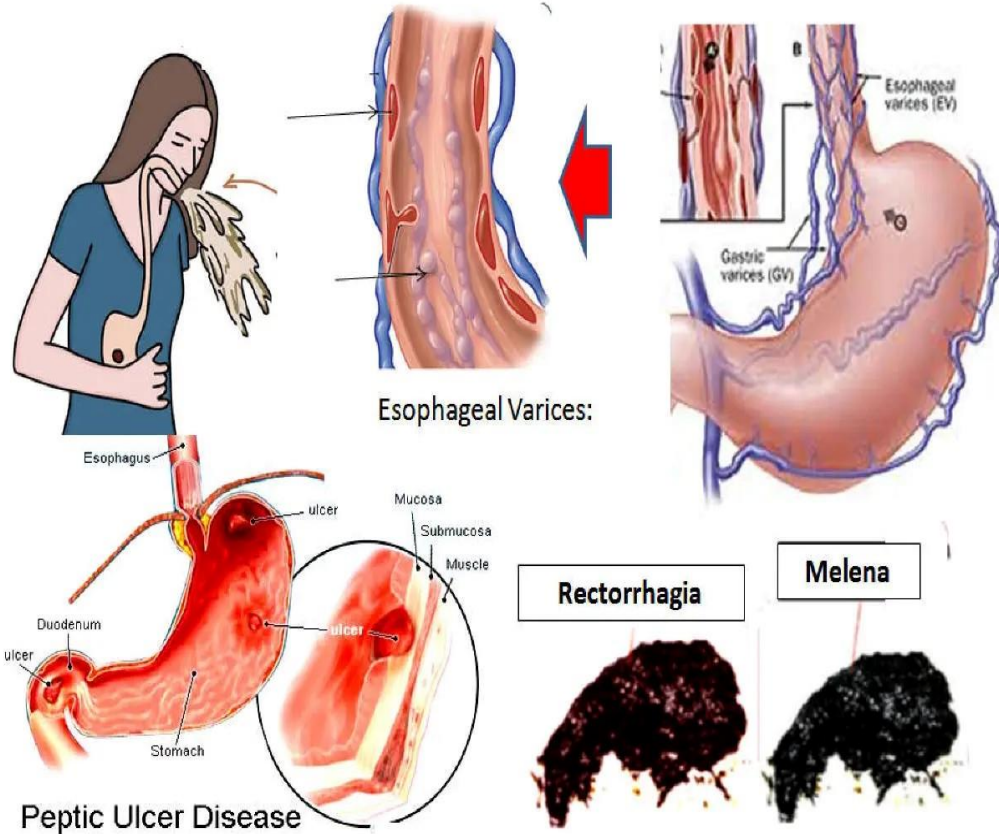




shutterstock.com • 637158385

↓ **nitric oxide** levels which act as a vasodilator, ↑ with in the levels of **endothelin- 1**, which acts as a strong vasoconstrictor that can cause **mucosal damage**.

# Hematemesis and melena



Stress ulcers are typically suspected in patients experiencing:

- Hematemesis
- Melena
- Anemia
- Hypotension or shock.

**Table 2**

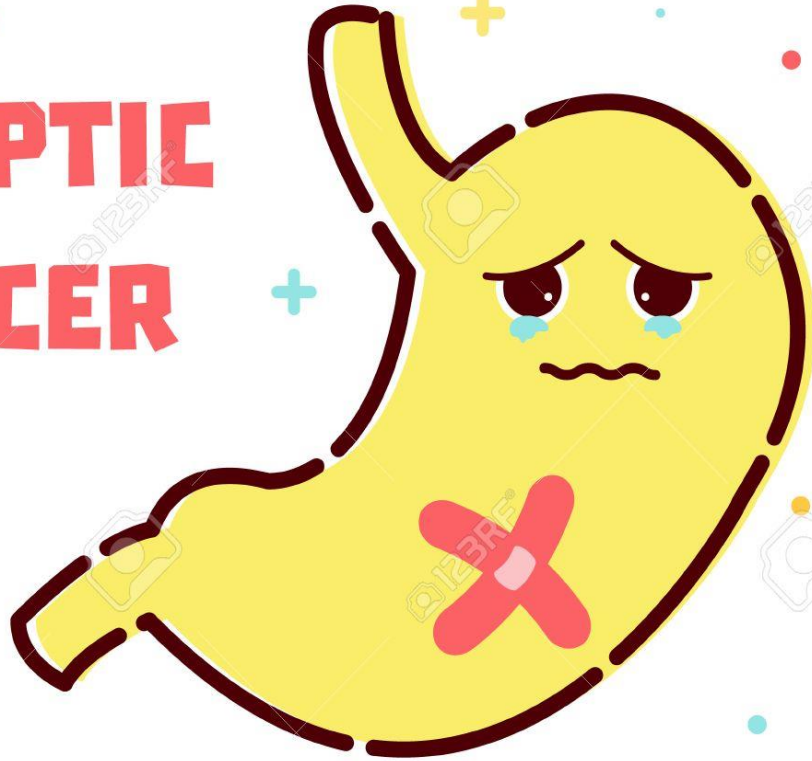
## Stress Ulcer Categories and Definitions

Category	Definition
Stress ulceration with occult GI bleeding	Fecal samples with guaiac-positive test for blood
Stress ulceration with overt GI bleeding	Hematemesis, bloody nasogastric tube aspirate, or melena
Stress ulceration with clinically important GI bleeding	Overt GI bleeding plus 1 or more of the following within 24 hours: Decrease in systolic, mean arterial blood pressure, or diastolic blood pressure of $\geq 20$ mmHg Orthostatic hypotension (systolic blood pressure $> 10$ mmHg) or postural tachycardia (increase in pulse $\geq 20$ beats/minute) Drop in hemoglobin $\geq 2$ g/dL Received transfusion of 2 or more units of packed RBCs Need for vasopressors or invasive interventions (e.g., endoscopy)

GI: gastrointestinal. Source: References 3, 6, 8.



# PEPTIC ULCER



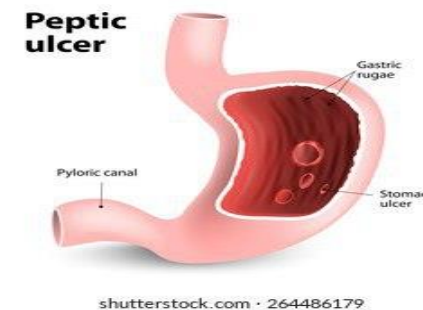
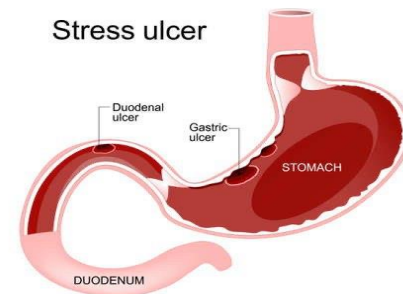
## What is peptic ulcer

Gastrointestinal mucosal injury related to critical illness.

The ulceration may vary from **diffuse superficial injuries** to **deep hemorrhaging ulcerations.**

# Stress Ulcers vs. Peptic Ulcers

<b>Stress ulcer</b>	<b>Peptic ulcer</b>
<p><b>Multiple superficial lesions</b> at the proximal stomach bulb, involve superficial capillaries; result from <b>splanchnic hypoperfusion</b>.</p>	<p><b>Few deep lesions in the duodenum</b>; typically involving a single vessel; result from a <b>break in gastric, duodenal, or esophageal lining</b> from the corrosive action of pepsin.</p>



## Risk factors

### Acute Risk Factors

1-MV (>48 hours) without enteral nutrition

2-Coagulopathy(plat<50,000 mm<sup>3</sup>, INR >1.5, or aPTT > 2 times control)

3- Hypo perfusion (shock, or organ dysfunction)

4-High-dose corticosteroids (>250 mg/day hydrocortisone or equivalent)

5- Significant burn injury (total body surface area 20%)

### Potential Risk Factors

1- Concomitant use (NSAID)

2-Concomitant or recent corticosteroid use

3-History of upper GI haemorrhage, peptic ulcer disease, or gastritis

## Other risk factors

- Spinal cord/head trauma.
- History of GI bleeding within the past year.
- Postoperative transplantation.





Stress ulceration can lead to serious complications including:

- perforation,
- Hemorrhagic shock,
- And death.



stress ulcer management **primarily focuses on preventive measures**, commonly referred to as **stress ulcer prophylaxis (SUP)**.

**These proactive strategies encompass:**

- The regular monitoring of hemoglobin levels,
- and screening for occult blood in both feces and gastric contents.

Moreover, SUP entails the optimization of prophylactic medications designed to curb the excessive production of gastric acid.

## Guideline Recommendations

clinical practice guideline for gastrointestinal bleeding prophylaxis for critically ill patients recommends stress ulcer prophylaxis with a **gastrointestinal bleeding risk of at least 4% based on several well-studied risk factors.**



# Drugs used for SUP

sacralfate	H2RBs	PPIs
<p>Forms physical cytoprotective barrier at the ulcer site which protect gastric mucosa from acid and pepsin</p>	<p>blocking of histamine binding to its G-protein coupled receptor on the gastric parietal cells <math>\Rightarrow</math> <math>\downarrow</math> acid production and gastric secretions.</p>	<p>inhibition of <math>H^+/K^+</math> ATPase enzyme at the secretory surface of the parietal cell <math>\Rightarrow</math> inhibition of <math>H^+</math> ions and thereby <math>\uparrow</math> pH of the gastric contents.</p>
<ul style="list-style-type: none"> <li>-constipation</li> <li>-occlusion of the feeding tube</li> <li>-<math>\downarrow K, PO_4</math></li> <li>-aluminum toxicity (especially in the presence of renal dysfunction)</li> <li>-drug binding warfarin, phenytoin, digoxin, fluoroquinolones, theophylline, quinidine, L-thyroxin</li> </ul>	<ul style="list-style-type: none"> <li>-thrombocytopenia (especially in pediatrics)</li> <li>-confusion (especially in elderly),</li> <li>-interstitial nephritis,</li> <li>-rapid infusion-related hypotension and sinus bradycardia,</li> <li>- pneumonia</li> </ul>	<ul style="list-style-type: none"> <li>-rebound acid hypersecretion after discontinuation</li> <li>-diarrhea</li> <li>-interstitial nephritis</li> <li>-pneumonia</li> <li>-high dose IV omeprazole (hearing and vision disturbances, seizures)</li> <li>-hypophosphatemia</li> <li>-osteoporosis and fractures</li> </ul>



According to a  
new study in  
2024

\*PPI was the most widely used acid-suppressing drugs, 45.8%. Furthermore, the H2 receptor antagonist was 42.6%, the sucralfate group was 7.4%, and the antacid group was 4.2%.



Drug  
interactions for  
ppi


All agents are hepatically metabolized by **CYP isoenzymes**.

Clinically significant PPI interactions with **clopidogrel** through CYP2C19 inhibition such as **omeprazole and esomeprazole**



## Drug interaction for H2brs

### Cimetidine

Causes oxidation of many drugs and  drug levels.  
(**Warfarin, beta-blockers**)

\* All H2 antagonists may inhibit the absorption of drugs that require an acidic GI environment for absorption.

**SMOKING** has been shown to  the effectiveness of H2 blockers.

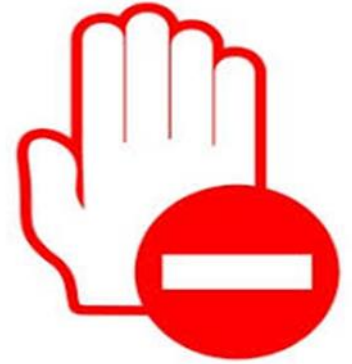


Drug  
interaction for  
H2brs

**Famotidine**

Does interact with ( **ataznavir , digoxin,  
itraconazole and ketokonazole** )

**\* Famotidin more potent effect than cemitoden**



shutterstock.com · 153819233

## Why we not use ranitidine

The drug **ranitidine** can elevate the risk of a person getting cancer.

The active ingredient, ranitidine, contains a contaminant **nitrosodimethylamine** (NDMA), which is a probable human carcinogen.

The above information comes from the Food and Drug Administration (FDA).



Anxiety is when the butterflies in  
your stomach turn into bees.

- Bridgett Devoue | TheMindsJournal

