Rapid Response Teams

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Disclosures

Learning objectives

- To understand the purpose rapid response teams
- To provide a diagnostic framework to identify early signs of the decompensating patient and build confidence in managing an acute patient
- To promote effective follow-up and handoffs regarding a potentially unstable patient
- To view a rapid response as an inflection point in the patient’s clinical course

Introduction

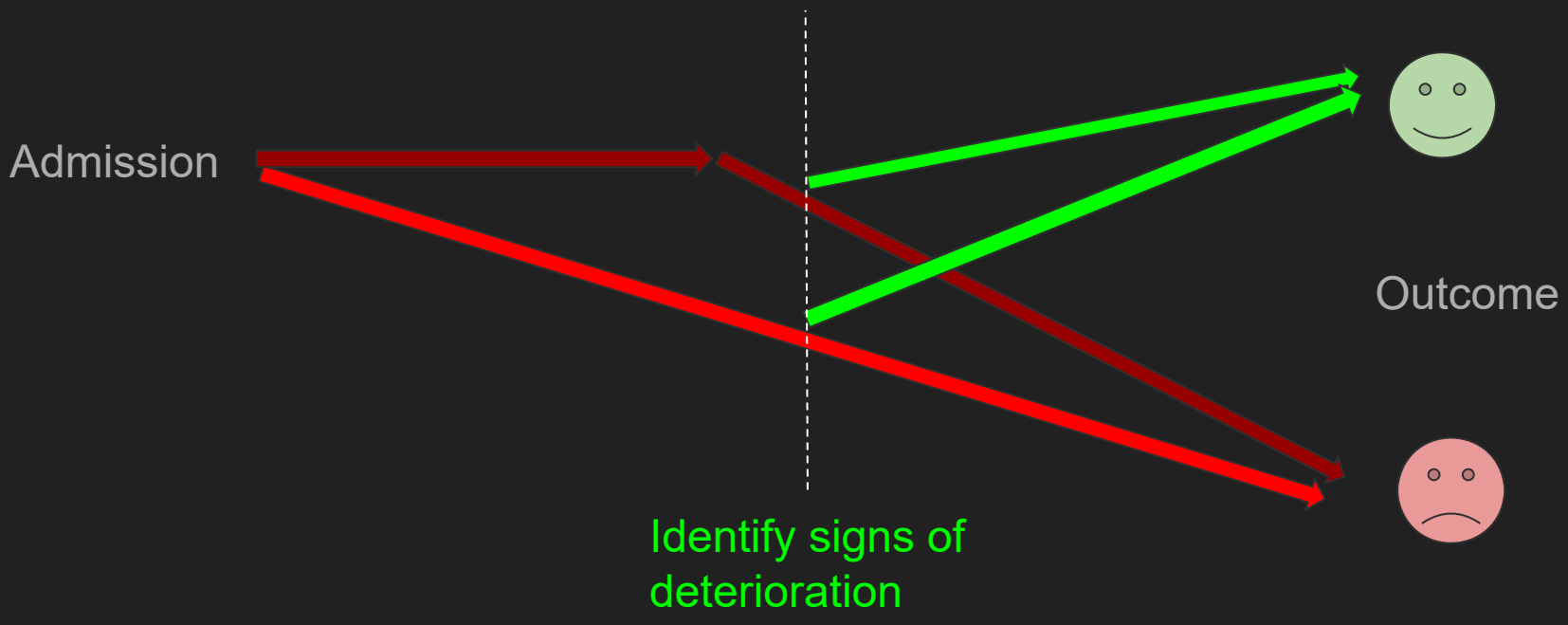
Rapid Response Team (RRT) activation	Clinical deterioration, acute change in mentation, patient appearance, vital signs, or institution-dependent protocol (eg. chest pain)
Code Blue	The patient has experienced cardiopulmonary arrest Activation of ACLS protocols
Code Stroke	The patient has a new or worsening neurological deficit

Introduction

- 2004 - Institute for Healthcare Improvement's 100,000 Lives Campaign
- Initial data mixed results, non-uniform implementation
- 2016 meta-analysis:

  RRTs were associated with significantly lower in-hospital mortality (relative risk, 0.88) and fewer non-ICU cardiac arrests (RR, 0.62). (Solomon RS et al. 2016)
- No universal composition for RRTs
- Commonly included - respiratory therapist, intensive care unit RN, a physician or advanced practice provider
- The Society of Critical Care Medicine recommends that at least one team member have experience in rapidly eliciting goals of care (SCCM, 2024)

General approach



General approach

General approach

Before - During - After

Before

- Signing out “watcher” patients and follow up plans
- Identifying any foreseen issues
- If “A” then “B” type statements

For example,

“Can you keep an eye on my septic patient in room 507? I have spoken with the ICU already. If their blood pressure drops again after additional fluids, then they will be admitted to the ICU”

“I have a patient in room 210 with COPD exacerbation that I weaned to oral steroids today. If their respiratory status worsens I was planning to resume IV steroids and consider CTA to look for PE”

During

- Introduce self and role
- Identify and receive report from bedside RN
- Helpful reports are brief but include pertinent information
    - For example, “This is Mr. Smith, he’s a 70 year old who was admitted two days ago with congestive heart failure and is receiving IV diuretics. About 5 minutes ago his telemetry began reading an irregular heart rate in the 140s. He reports palpitations. He does not have a history of afib. Here is his most recent set of vitals.”
- “Look test” - ABCs
- Immediate action: Does the patient require emergent care now? (eg. airway, cath lab, cardioversion, surgery)
- Proceed to quick history and physical

During - Assessment

- Abbreviated history from the patient
- Helpful points of a quick physical exam:
  - General appearance: diaphoresis, pallor, icterus
  - Neuro: Level of consciousness, pupils, facial expression, gross motor
  - Neck: JVP, stridor
  - Lung fields, work of breathing, accessory muscle usage
  - Cardiac exam, telemetry if applicable
  - Abdominal palpation
  - Periphery: edema, pulses, perfusion of extremities
- Don’t forget about a fingerstick glucose! Also a bladder scan
- Great time for POCUS if you are comfortable and it is readily available

During - Focusing the differential

Most problems that arise such as dyspnea, altered mental status, chest pain have expansive differentials that are not always practical to consider during a rapid response

This can be narrowed down if you keep in mind that this is usually something that developed quickly, can be related to current illness, or related to being hospitalized

Ask yourself - Is this related to current illness? Is this related to something in initial differential that was felt less likely at the time? Is this related to something we did (procedure or medication)? Is this related to something we didn't do (not continuing a home med)?

After

- Treatment: Treatment can be administered in the room, but waiting for more data is also ok if it will be available in a reasonable amount of time
- Dispo: Does the patient need to be moved to a different unit (eg. telemetry, neuro) or does care need to be escalated (ICU)?
    - For patients who are borderline, how much “wiggle room” is there?

“If, then” statements:

“Let’s try NPPV for two hours and recheck a blood gas, if it is the same or worse, then I will transfer them to the ICU”

After - Escalation of care

- If patient is oriented, confirm code status and discuss potential for escalation, to confirm it is within their goals
- Involve family when possible or when code status is unclear. Notify someone that the patient is being admitted to the ICU if they require a higher level of care
- Even if a patient has been accepted to the ICU, you can still initiate treatment
- For patients who are borderline, I recommend giving the intensivist a heads up

After - Communication

- Communicate plan with the bedside RN, other members of the rapid response team, and the patient
- Write a brief note in the chart that states the event, treatment administered, and plan of action, pending diagnostics
- Written or verbal sign out to the provider assuming care of the patient which includes any pending diagnostics

My 3 rules for RRTs

Rule 1: Take a breath

Walking into a rapid can be stressful, particularly when you have never met the patient before and do not know ahead of time why the rapid was called

Take a brief pause before you enter the room - stress is contagious

Rule 2: “Looks bad” rule

If a patient looks very sick, but nothing immediately is jumping out in the differential, or you would like to add some objectivity to your assessment:

CBC, CMP, Mag, lactic acid, ABG, EKG

But in particular, I look at the h/h, lactic acid, EKG, and blood gas

Rule 3: “Mr. Miyagi” rule

“Walk on road, hm? Walk right side, safe. Walk left side, safe. Walk middle, sooner or later, get squish, just like grape.”

-*The Karate Kid*, 1984

-Always follow up and react to pending labs and imaging
-Never just get one troponin
-Be sure you have reasonably ruled out emergent conditions

Unofficial rule #4

Poor prognostic signs:

“The patient was supposed to be discharged tomorrow”

“Pending GOC conversation in the morning”

“The patient is a farmer”

Case examples

Case 1 Hypoxia

You are paged regarding Mr. Burton, a 78 year old male for hypoxia and increased work of breathing.

The nurse tells you that he normally wears 2L oxygen at home and now is saturating 86% on 6L nasal cannula. He is visibly distressed. Full set of vitals currently being obtained.

As you are walking to the room you tell the RN to place a non-rebreather mask and activate a rapid response.

You take a quick glance at the chart.

He was admitted two days ago for multiple falls at home related to orthostatic hypotension and UTI, currently awaiting rehab placement. He has a history of dementia, HFpEF, COPD, T2DM. His home antihypertensives were held on admission and he was placed on IV fluids

Case 1

Differential?

-Pulmonary edema

-Pulmonary embolism

-Exacerbation of COPD

-Aspiration

-ACS

-Drug reaction

Case 1

You arrive to the room and assess the patient. He tells you he feels winded but it helps when he is sitting up. He reports no chest pain, palpitations, dizziness, nausea or vomiting. He ate dinner about 30 minutes ago and about two hours ago received his second IV dose of ceftriaxone.

Vitals: BP 130/80, HR 95, RR 26, O2 Sat 93% on non-rebreather mask, temp 98F

On exam:
He is alert and oriented, speaking in short sentences. Non-diaphoretic. No cyanosis.
He is sitting upright in bed without accessory muscle usage.
Heart rate is regular without murmur noted. Lungs are well-aerated but demonstrate diffuse end-expiratory wheezes, as well as rales bilaterally
Abdomen is soft and nondistended
There is 2+ bilateral lower extremity edema. Extremities are warm.
There are no rashes or lesions visible on skin

Case 1

You ask the RT to start the patient on CPAP. You order an albuterol nebulizer and a dose of IV furosemide. IV fluids are discontinued.

Your workup is notable for hypoxemia on the arterial blood gas with a normal pH and pCO_2. An EKG is unchanged from his baseline. CXR shows increased vascular markings and softening of the costophrenic angles bilaterally.

The patient's effort of breathing is much better with NPPV and he maintains good urine output following the diuretic.

Case 1

Follow up -

Response to diuretics

Consider a short interval follow up EKG

Is work of breathing and saturation improved on NPPV?

Consider when to repeat a blood gas if needed

Case 2 Altered mental status

You arrive at the room of Ms. Draper, a 68 year old woman who was admitted late afternoon for acute cholecystitis, awaiting surgical evaluation. The bedside nurse reports that over the last few minutes, the patient has become very difficult to arouse.

On chart review you see that she has CKD3, T2DM on insulin, and HTN. Her creatinine on admission was elevated compared to baseline.

She is currently NPO. The bedside RN tells you that the patient received a dose of IV pip-tazo, and a dose of IV morphine in the ED prior to arriving on the floor. She did not receive any insulin yet during hospital stay.

Case 2

BP 110/58, HR 66, RR 10, O2 Sat 92% on non-rebreather, Temp 98F

You notice that the patient is responsive only to noxious stimuli. She has symmetric miosis. Respirations are slow and shallow but she has breath sounds in all lung fields. Her abdomen is soft without rigidity or guarding.

POC glucose 109, ABG pH 7.30, pCO2 70, PO2 68

You administer 0.4 mg IV narcan and the patient wakes up and vomits. She is uncomfortable but now responds to verbal stimuli and respirations improve

What is your next step?

Case 2

Follow up -

Duration of Narcan - 60-90 minutes which is shorter than most opiates

Have another dose available with as-needed parameters, and if it needs to be given consider a naloxone infusion

Case 3 Chest pain

A rapid response is called to the room of Mr. Holden, a 60 year old male who was admitted the night prior for new-onset heart failure. He has a PMH of COPD, uncontrolled T2DM, HTN, HLD, and tobacco use. He was started on IV furosemide at the time of admission. Echocardiogram has not yet been performed.

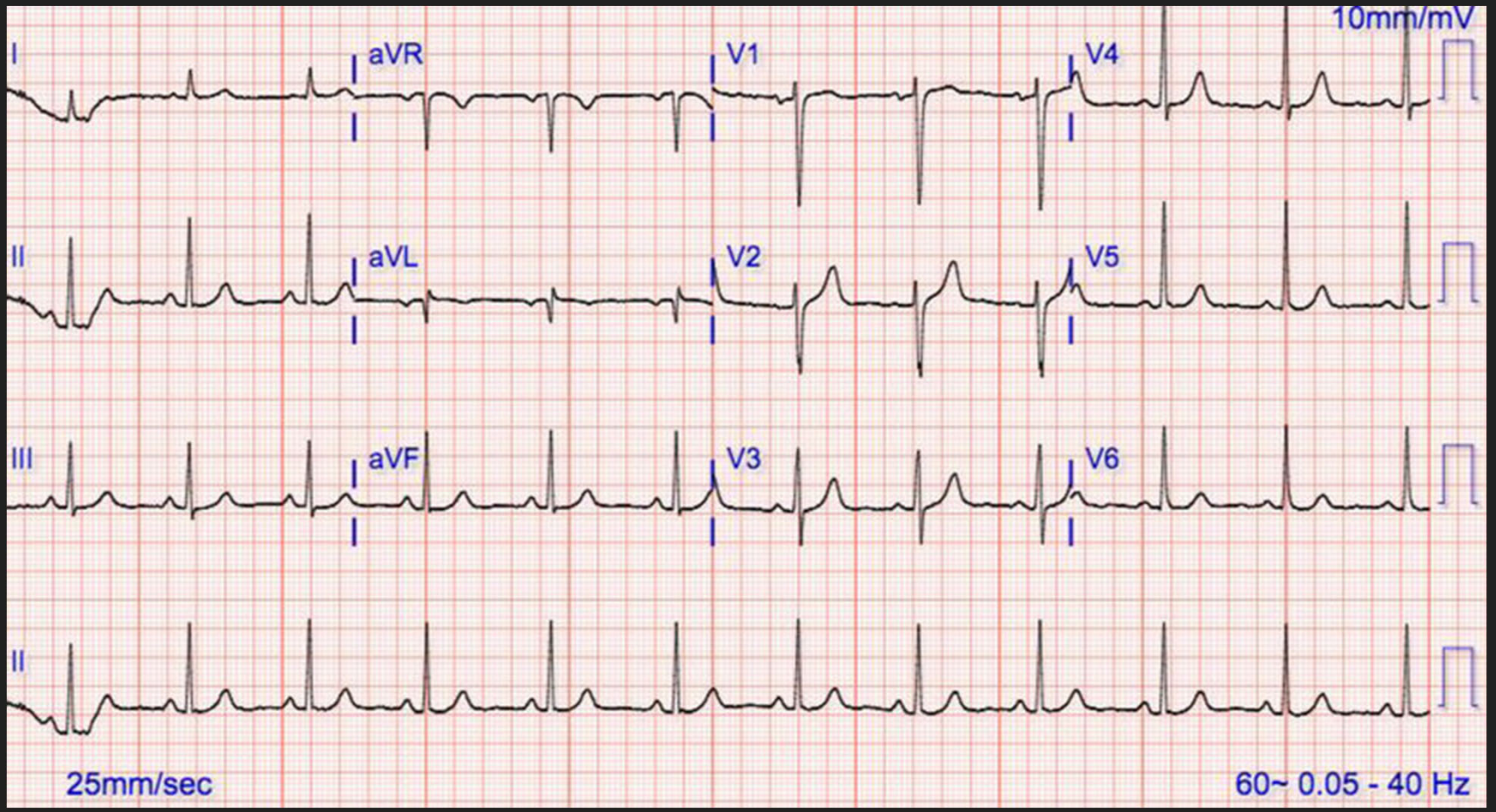
He reports worsening dyspnea at rest and substernal chest pressure that radiates to his left arm. He rates this 9/10. He does not have dizziness, vomiting, or palpitations.

Case 3

BP 165/90, HR 80, RR 22, O2 Sat 94% on 4L nasal cannula, Temp 98F

He appears uncomfortable, diaphoretic, and dyspneic in conversation. His JVP is 12 cm, his heart rhythm is regular and you hear an S3 sound. He has crackles at the bilateral lung bases. Radial pulses are symmetric. There is 2+ pitting edema in the lower extremities bilaterally.

You review his EKG and administer a dose of SL nitroglycerin, 325 ASA, and 2 mg IV morphine while you await a full set of labs including troponin, and a CXR



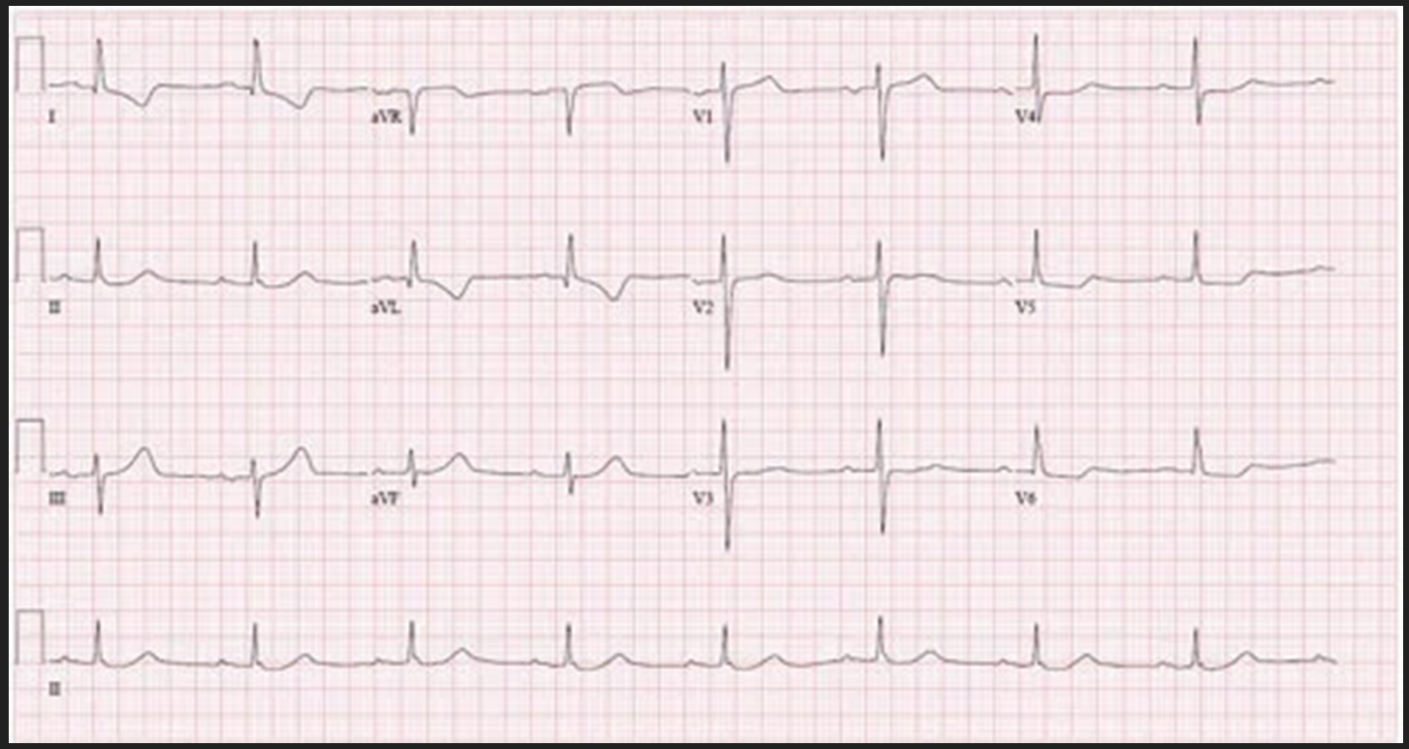
Reference photo 1

Case 3

The chest X ray shows small bilateral pleural effusions and increased interstitial markings, similar to prior. Otherwise, no new findings.

He feels slightly better with the prior interventions and now rates his pain a 4/10. He still appears dyspneic.

Now what?



Reference Photo 2

Case 3

You start a beta blocker, high intensity statin, and a heparin drip while you await a return page from cardiology to expedite his evaluation

A bedside echocardiogram shows EF 35% with motion defects in the lateral left ventricle

The patient is taken for urgent left heart catheterization which reveals a 90% occlusion in the LCx, determined to be the culprit lesion

Case 4 Hypotension

You hear a rapid response called to the room of Mr. Kamal. He is a 78 year old man who was admitted two days ago after suffering a CVA with hemiplegia.

BP 70/30, HR 118, RR 18, O2 Sat 92% on 2L O2, Temp 99.5F

He is ill-appearing and mottled. Wakes with minor stimulus and can follow commands. He is dysarthric. He has shaking rigors. The heart rhythm is regular without murmur. Lung fields aerated throughout with rhonchi heard in the mid right chest. Abdomen is soft, nondistended, nontender. Distal pulses are weak but symmetric. His NIHSS is similar to prior. Fingerstick glucose is 130.

Case 4 - Differential?

Sepsis

Blood loss

Medication effect

PE with tamponade

Cardiogenic

Central/neurogenic

Case 4

ABCs - start giving some fluids

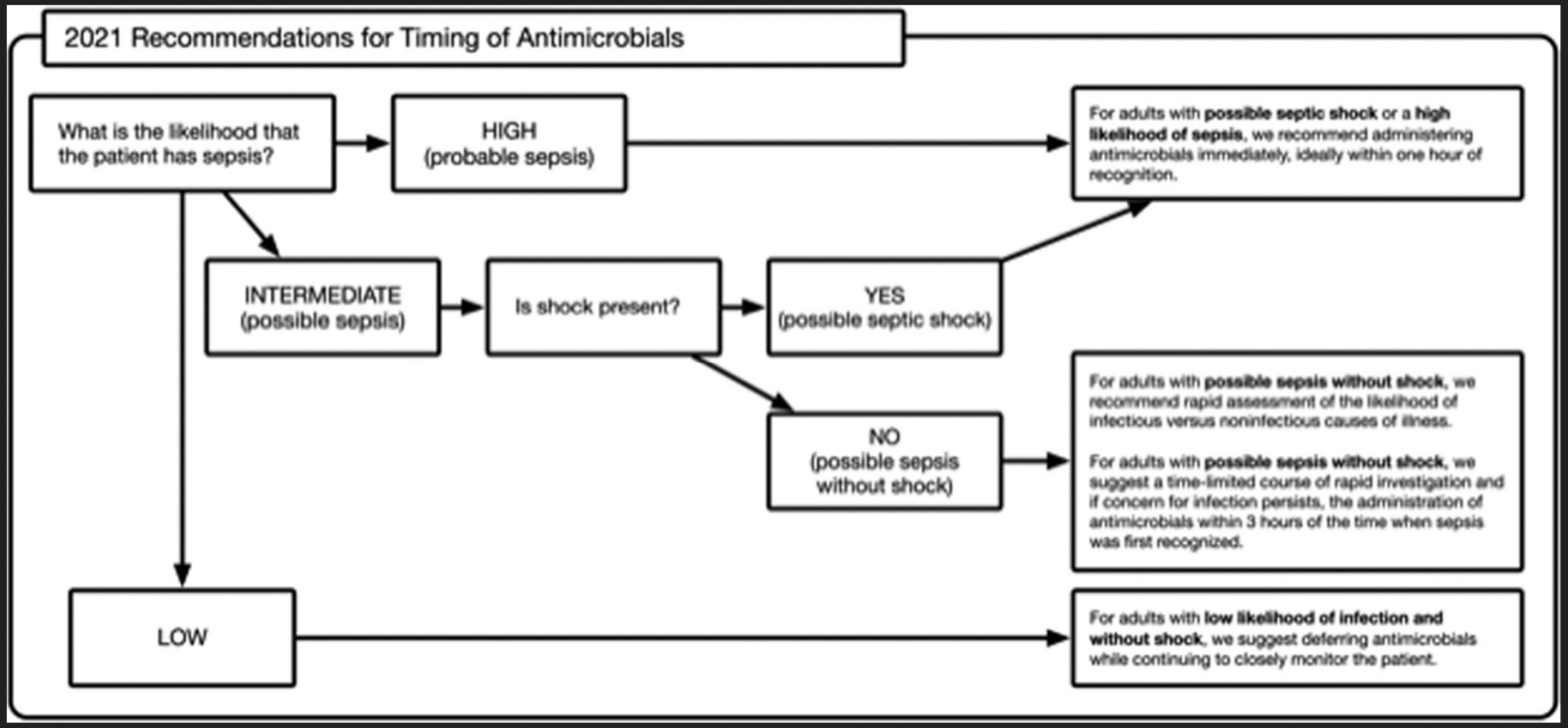
Consider taking a rectal temperature

Look at ins and outs, bladder scan

CBC, CMP, lactic acid, UA, viral swab, EKG, CXR

Blood cultures

What treatment do you initiate at this time?



Critical Care Medicine49(11):1974-1982, November 2021.

Main risk factors for multi-drug resistant pathogens.

MRSA	1. Previous infection/colonization by MRSA in the last 12 months 2. Hemodialysis or peritoneal dialysis 3. Presence of central venous catheters or intravascular devices 4. Administration of multiple antibiotics in the last 30 days (in particular with cephalosporins or fluoroquinolones) 5. Immunodepression 6. Immunosuppressor treatments 7. Rheumatoid arthritis 8. Drug addiction 9. Patients coming from long-term care facilities or who have undergone hospital stay in the last 12 months 10. Close contact with patients colonized by MRSA
ESBL	1. Previous infection/colonization with ESBL in the last 12 months 2. Prolonged hospitalization (>10 days, in particular in ICU/hospice/long-term care facilities) 3. Presence of permanent urinary catheter 4. Administration of multiple antibiotics in the last 30 days (particularly with cephalosporins or fluoroquinolones) 5. Patients with percutaneous endoscopic gastrostomy

J. Infect. Public Health. 2021;14:1750–1766

Pseudomonas aeruginosa	1. Previous infection/colonization with P. aeruginosa in the last 12 months 2. Administration of multiple antibiotics in the last 30 days (particularly with cephalosporins or fluoroquinolones) 3. Pulmonary anatomic abnormalities with recurrent infections (e.g., bronchiectasis) 4. Elderly patients (>80 years) 5. Scarce glycemic control in diabetic subjects 6. Presence of permanent urinary catheter 7. Prolonged steroid use (>6 weeks) 8. Neutropenic fever 9. Cystic fibrosis
***Candida* spp.**	1. Immunodepression 2. Presence of central venous catheters or intravascular devices 3. Patients in total parenteral nutrition 4. Prolonged hospitalization (>10 days, particularly in an ICU) 5. Recent surgery (particularly abdominal surgery) 6. Prolonged wide-range antibiotic administration 7. Previous necrotizing pancreatitis 8. Recent fungal infection/colonization

J. Infect. Public Health. 2021;14:1750–1766

Case 4

1L of LR has infused so far and the blood pressure is now 80/50

Broad spectrum antibiotics have been initiated

Your workup is remarkable for WBC 18k, hemoglobin 10.8, lactate 3.2

UA with few WBCs, negative for nitrites and leukocyte esterase

CXR shows opacity in RML

TABLE 1. - Selected New and/or Revised Recommendations in the 2021 Surviving Sepsis Campaign International Guidelines for the Management of Sepsis and Septic Shock

2016 Recommendation	2021 Recommendation	Rationale for Change
We recommend that in the resuscitation from sepsis-induced hypoperfusion, at least 30 mL/kg of IV crystalloid fluid be given within the first 3 hours.	For patients with sepsis-induced hypoperfusion or septic shock we suggest that at least 30 mL/kg of IV crystalloid fluid should be given within the first 3 hours of resuscitation.	This panel downgraded this recommendation from a strong recommendation to a weak recommendation based on the low quality of the evidence. There are no prospective intervention studies comparing different volumes for initial resuscitation in sepsis or septic shock. However, a retrospective analysis of adults presenting to an emergency department with sepsis or septic shock showed that failure to receive 30mL/kg of crystalloid fluid therapy within 3 hours of sepsis onset was associated with higher in-hospital mortality ([10]). Furthermore, the average volume of fluid received pre-randomization the PROCESS ([11]), PROMISE ([12]), and ARISE ([13]) trials was in the range of 30 mL/kg, suggesting this fluid volume has been adopted in routine clinical practice ([14]).
We suggest using either balanced crystalloids or saline for fluid resuscitation of patients with sepsis or septic shock.	For adults with sepsis or septic shock, we suggest using balanced crystalloid instead of normal saline for resuscitation.	There are many, increasingly recognized potential adverse effects of normal saline including hyperchloremic metabolic acidosis. A network meta-analysis showed in an indirect comparison that balanced fluids were associated with decreased mortality compared with saline ([15]). In the 2018 SMART single-center cluster-randomized RCT comparing saline to balance fluid, the pre-specified subgroup of patients admitted with sepsis experienced lower 30-day mortality when randomized to balanced fluids versus saline (OR, 0.90; 95% CI, 0.67, 0.94) ([16]).
Not addressed	For adults with septic shock, we suggest starting vasopressors peripherally to restore mean arterial pressure rather than delaying initiation until a central venous access is secured.	Prompt initiation of vasopressors is an integral component of septic shock management. Vasopressors have been traditionally administered via central venous access due to concerns of extravasation and local tissue injury and ischemia. However, placement of central venous access requires specialized expertise and is time consuming, potentially leading to delays in administration. A recent systematic review showed that peripheral administration of vasopressors is generally safe, particularly if infused distally to the antecubital fossa and for short periods of time (< 6 hr) ([17], [18]). Peripheral administration of vasopressors is associated with shorter time to administration and faster time to achieving a MAP > 65 mm Hg ([19]).
Not addressed	For adults with sepsis or septic shock we suggest against using IV vitamin C.	A 2017 single center before and after study reported reduced mortality with administration of high-dose Vitamin C, hydrocortisone, and thiamine among patients with sepsis and sepsis shock ([20]). However, an updated meta-analysis by the guideline panel found no association between vitamin C and reduced mortality.
We suggest against using IV hydrocortisone to treat patients with septic shock if adequate fluid resuscitation and vasopressor therapy can restore hemodynamic stability. If this is not achievable, we suggest IV hydrocortisone at a dose of 200 mg/day.	For adults with septic shock and an ongoing requirement for vasopressor therapy we suggest using IV corticosteroids.	Since the 2016 guideline, three large RCTs have been published ([21–23]). An updated meta-analysis found systemic corticosteroid to accelerate resolution of shock (MD, 1.52 days; 95% CI, 1.71 to 1.32) and increase vasopressor-free days (MD, 1.5 days; 95% CI, 0.8 to 3.11 days) ([24]). However, corticosteroid use increased neuromuscular weakness (RR, 1.21; 95% CI, 1.01 to 1.45), without a clear effect on short- or long-term mortality ([24]). The overall quality of evidence was moderate. The panel judged the desirable effects (shock resolution, vasopressor-free days) to outweigh the undesirable effects. This observation, combined with consideration of the resources required, cost of the intervention, and feasibility supported a weak recommendation in favor of using low-dose corticosteroid therapy in septic shock.
Not addressed	For adult survivors of sepsis or septic shock, we recommend assessment and follow-up for physical, cognitive, and emotional problems after hospital discharge.	Given the prevalence of new and worsening physical, cognitive, and emotional problems experienced by sepsis survivors, we recommend assessment and follow-up of these problems after discharge.

Case 4

Follow up -

Response to fluids? Need for escalation of care?

Source identified?

Reflex lactic acid, re-assmentment of adequate perfusion

Case 5 Tachycardia

You are called to the room of Ms. Nagata, an 86 year old female who was admitted a few hours ago for sepsis secondary to urinary tract infection.

You are given a set of vitals over the phone as you head to the room.

BP 110/70, HR 161, RR 14, O2 sat 96% on room air, Temp 99F

Case 5

Differential:

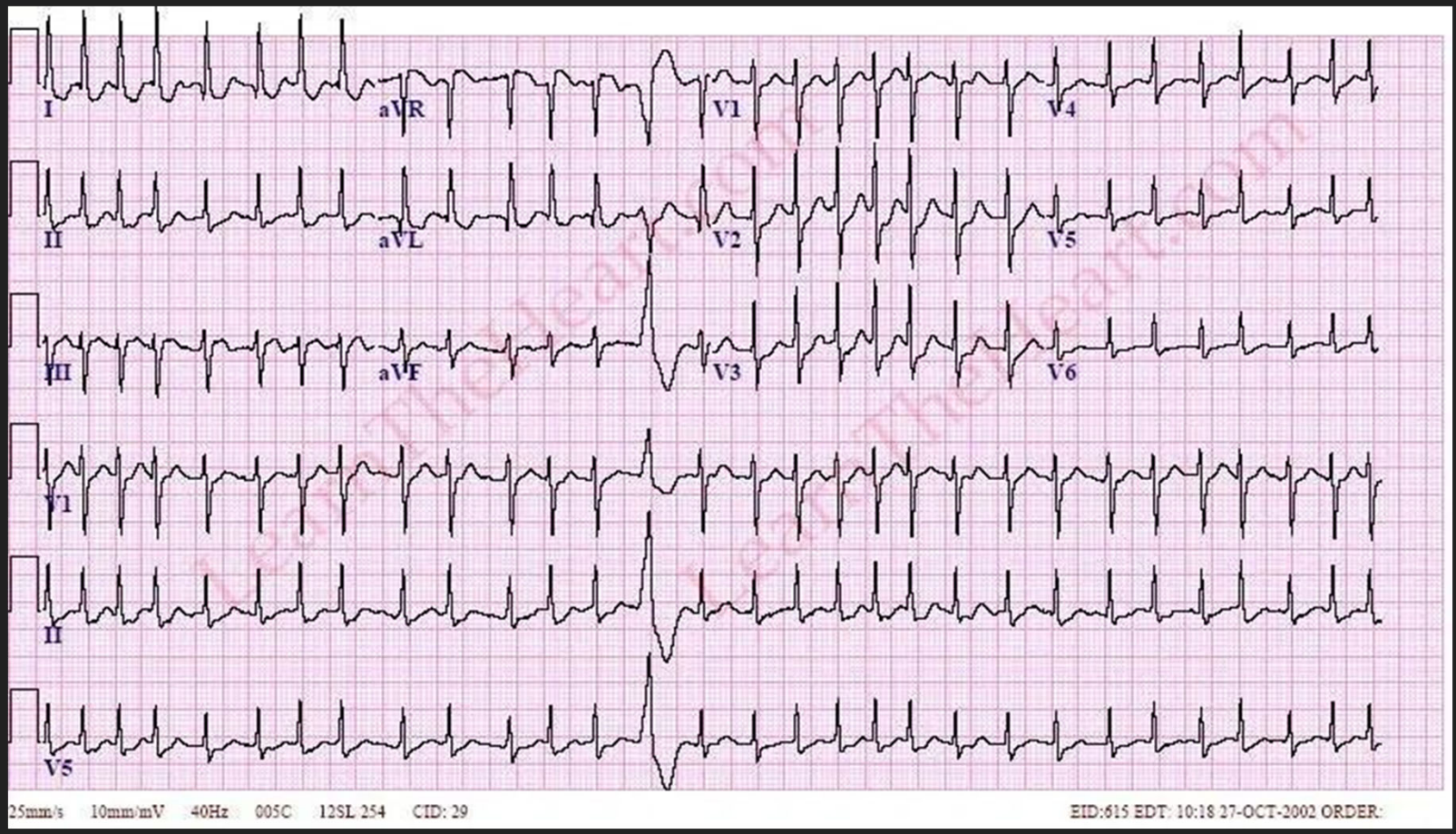
Wide ddx for tachycardia - is it sinus origin or arrhythmia?

Hypovolemia, sepsis, fever, pain, hypoxia, PE, withdrawal syndrome, anxiety

Case 5

You arrive to the room and assess the patient. She is nondiaphoretic but slightly dyspneic in conversation. She does not report any pain but states she feels a fluttering sensation in her chest and feels short of breath.

You review the EKG at bedside



Reference Photo 3

Case 5

Treatment options for acute afib RVR:

AV nodal blockers - metoprolol, esmolol, diltiazem

Antiarrhythmics - amiodarone, digoxin

Hemodynamic instability or active ischemia - cardioversion

Initial Options for Acute Rate Control

	Major Pharmacologic Considerations	Notable Contraindications	Typical Initial Dose
Diltiazem (IV)	• Rapid onset and rapidly titratable • Easy to transition to oral • Generally considered most effective option for achieving immediate rate control	• ADHF	• 5-20mg (may repeat x 1 after 15 min) • Followed by 5mg/hr infusion (titratable up to 15mg/hr)
Diltiazem (Oral)	• Easy to directly transition to outpatient regimen	• ADHF	• 30mg q6h (immediate release) • Once effective dose established, can convert to extended release
Metoprolol (IV)	• Easy to transition to oral metoprolol • Beneficial in HFrEF (long-term)	• ADHF • COPD / Acute bronchospasm	• 2.5-5mg (may repeat q10-20 min to max total dose 15mg) • If HR successfully lowered, then can convert to oral metoprolol
Esmolol (IV)	• Rapid onset and rapidly titratable • No oral form, so transitioning to outpatient regimen is not as clear as with other options	• ADHF • COPD / Acute bronchospasm	• Loading dose of 500mcg/kg over 1 min • Followed by 50mcg/kg/min • Can titrate up to max of 200mcg/kg/min
Amiodarone (IV)	• Best acute option for patients with ADHF • Risks conversion to sinus rhythm • IV bolus can cause hypotension • Many toxicities with long-term oral use	• Known left atrial thrombus • Inadequate anticoagulation (if a-fib > 48 hours)	150mg over 10 min, followed by 1.0mg/min x 6 hrs followed by 0.5mg/min
Digoxin (IV or PO)	• Slowest onset of action • Rate control is most prominent when patient at rest • 3rd line agent • Does not cause hypotension	• Renal failure	• Requires initial load given in 3 divided doses over 12 hours • Consider consultation with pharmacy

Eric Strong: Acute Atrial Fibrillation, 2020

Case 5

Follow up -

Did you control the heart rate with your treatment?

Underlying cause? Hypo or hypervolemia, electrolytes

Anticoagulation? CHA2DS2VASc and HASBLED

EKG upon conversion to sinus rhythm, or when HR improved if ischemic changes on initial EKG

Summary

Rapid responses are called when there is a change in clinical status of a patient

Effective handling of rapid responses relies on timely assessment and triage of the patient, as well as communication with other team members involved in the patient's care

Always make sure you have altered, or at least stabilized, the trajectory of a decompensating patient

Don't be afraid to reach out to your specialists, or intensivists if you are concerned

We are on all the same team

Questions?

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Reference Photo 1: Repeat-12-lead-EKG-showing-normal-sinus-rhythm.png (850×460) (researchgate.net)

Reference Photo 2: circumflex-occlusion-figure1.jpg (500×264) (emra.org)

Reference Photo 3: Management of Atrial Fibrillation (AF) with Rapid Ventricular Response (RVR) - Manual of Medicine