External pacing vs drugs in bradycardia

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Kusumoto FM, et al.
2018 Bradycardia Clinical Practice Guidelines

2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay

Definition

- The National Institutes of Health defines bradycardia as a heart rate <60 bpm in adults other than well trained athletes.

- A sinus rate <50 bpm and/or a sinus pause >3 seconds as potential components of the definitions of SND.

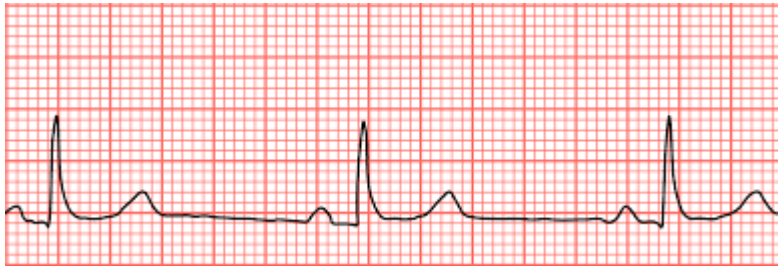
Clinical Manifestations of Bradycardia

- Fatigue
- Syncope
- Presyncope
- Transient dizziness
- Heart failure symptoms
- Confusional states

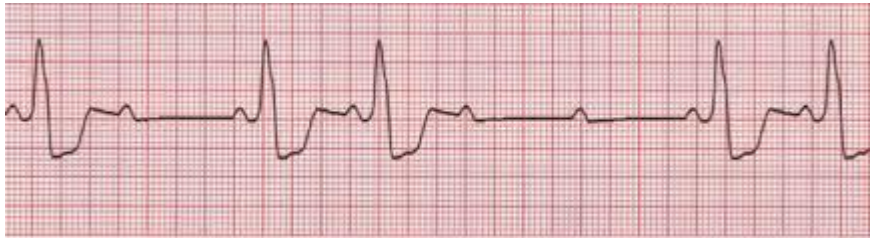
Inadequate Perfusion

- Hypotension
- Altered mental status
- Signs of shock
- Ongoing ischemic chest pain
- Evidence of acute pulmonary edema

- Bradycardia can be broadly classified into 2 general categories:
- SND



- Atrioventricular block



Recommendation for History and Physical Examination in Patients With Documented or Suspected Bradycardia or Conduction Disorders		
COR	**LOE**	**Recommendation**
I	C-EO	In patients with suspected bradycardia or conduction disorders a comprehensive history and physical examination should be performed.

Recommendation for Acute Management of Reversible Causes for Bradycardia Attributable to SND		
COR	**LOE**	**Recommendation**
I	C-EO	In symptomatic patients presenting with SND, evaluation and treatment of reversible causes is recommended.

Acute Medical Therapy: Atropine

- Atropine is a parasympatholytic drug that blocks the muscarinic acetylcholine receptor.
- In the sinus node, it facilitates sinoatrial conduction and increases sinus node automaticity.

IIa	C-LD	1. In patients with SND associated with symptoms or hemodynamic compromise, atropine is reasonable to increase sinus rate (S4.1.2.1-1–S4.1.2.1-4).
III: Harm	C-LD	3. In patients who have undergone heart transplant without evidence for autonomic reinnervation, atropine should not be used to treat sinus bradycardia (S4.1.2.1-12, S4.1.2.1-13).

Atropine- AV Block

- It is more likely to be useful for AV Block at the atrioventricular nodal level and for bradycardia attributable to excess vagal tone.
- It is unlikely to improve AV Block at the His bundle or His-Purkinje level.

IIa	C-LD	1. For patients with second-degree or third-degree atrioventricular block believed to be at the atrioventricular nodal level associated with symptoms or hemodynamic compromise, atropine is reasonable to improve atrioventricular conduction, increase ventricular rate, and improve symptoms (S6.3.2-1–S6.3.2-3).

Atropine- AV Block

- Because of its short duration of action, it is generally used as a bridge to longer-lasting therapy, such as infusion of a beta-adrenergic drug or temporary pacing.

Atropine	0.5-1 mg IV (may be repeated every 3-5 min to a maximum dose of 3 mg) (S5.3.2.4-20–S5.3.2.4-24)

Chronotropic Agents

- For patients who remain symptomatic following atropine
- For whom temporary cardiac pacing is either not readily available
- not successful in alleviating symptoms

IIb	C-LD	**2. In patients with SND associated with symptoms or hemodynamic compromise who are at low likelihood of coronary ischemia, isoproterenol, dopamine, dobutamine, or epinephrine may be considered to increase heart rate and improve symptoms (S4.1.2.1-5–S4.1.2.1-11).**

Chronotrophic Agents- AV Block

- Isoproterenol, dopamine, dobutamine, and epinephrine exert direct effects to enhance atrioventricular nodal and, to a lesser degree, His-Purkinje conduction.

IIb	B-NR	2. For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise and who have low likelihood for coronary ischemia, beta-adrenergic agonists, such as isoproterenol, dopamine, dobutamine, or epinephrine, may be considered to improve atrioventricular conduction, increase ventricular rate, and improve symptoms (S6.3.2-3–S6.3.2-7).

Isoproterenol

- Isoproterenol is a nonselective beta agonist with both chronotropic and inotropic effects on cardiac myocytes, enhancing sinus and atrioventricular nodal function without exerting a vasopressor effect.

Isoproterenol	20-60 mcg IV bolus followed doses of 10-20 mcg, or infusion of 1-20 mcg/min based on heart rate response (S5.3.2.4-26–S5.3.2.4-32)	Monitor for potential development of ischemic chest pain

Dopamine

- Dopamine is a catecholamine with mixed alpha-adrenergic, beta-adrenergic, and dopaminergic effects that depend on dosage, distribution, and metabolism.
- Higher doses may be required for a chronotropic response but must be used judiciously because of the association with profound vasoconstriction and proarrhythmias.

Dopamine	5 to 20 mcg/kg/min IV, starting at 5 mcg/kg/min and increasing by 5 mcg/kg/min every 2 min (S5.3.2.4-25)	Dosages of >20 mcg/kg/min may result in vasoconstriction or arrhythmias

Dopamine

- Clinical efficacy of dopamine was shown to be equivalent to transcutaneous pacing in 1 small randomized trial of patients with unstable bradycardia unresponsive to atropine in the prehospital setting.

CLINICAL PAPER

A randomized controlled feasibility trial comparing safety and effectiveness of prehospital pacing versus conventional treatment: 'PrePACE'☆,☆☆

Laurie J. Morrison[a,b,c,d,e,f,h,*], Jennifer Long[a], Marian Vermeulen[f,h], Brian Schwartz[b,c,e,i,j], Bruce Sawadsky[b,c,j,k], Jamie Frank[a,l], Bruce Cameron[a,l], Robert Burgess[l], Jennifer Shield[l], Paul Bagley[l], Vivien Mausz[l], James E. Brewer[m], Paul Dorian[d,e,g]

Epinephrine

- Epinephrine is a catecholamine with strong alpha-adrenergic and beta-adrenergic stimulatory effects, including increasing chronotropy, inotropy, blood pressure, and myocardial oxygen consumption.

Epinephrine	2-10 mcg/min IV or 0.1-0.5 mcg/kg/min IV titrated to desired effect (S5.3.2.4-17, S5.3.2.4-31, S5.3.2.4-33)

Beta-blocker and Calcium channel blocker Toxicity

- Cardiovascular effects of beta-blocker and calcium channel blocker toxicity are systemic and can be fatal because of profound negative chronotropic and inotropic effects, as well as vasodilation.
- Pharmacotherapy is supportive and directed toward improving hemodynamic stability.

Calcium

- Because of improvements in heart rate and blood pressure, coupled with low risk of adverse effects, it is often recommended as a first-line therapy.
- It is demonstrated reduced mortality and hemodynamic improvement.

IIa	C-LD	1. In patients with bradycardia associated with symptoms or hemodynamic compromise because of calcium channel blocker overdose, intravenous calcium is reasonable to increase heart rate and improve symptoms (S5.3.2.2-1–S5.3.2.2-3).

Glucagon

- Glucagon is a vasoactive polypeptide, which counteracts the effects of beta blockers by activation of hepatic adenyl cyclase that promotes glycogenesis.

IIa	C-LD	2. In patients with bradycardia associated with symptoms or hemodynamic compromise because of beta-blocker or calcium channel blocker overdose, glucagon is reasonable to increase heart rate and improve symptoms (S5.3.2.2-4, S5.3.2.2-5).

High-dose Insulin

- High-dose insulin therapy is associated with improved heart rate, hemodynamic parameters, and mortality in beta-blocker and calcium channel blocker overdose.
- Side effects include hypoglycemia and hypokalemia, which are usually mild.

IIa	C-LD	3. In patients with bradycardia associated with symptoms or hemodynamic compromise because of beta-blocker or calcium channel blocker overdose, high-dose insulin therapy is reasonable to increase heart rate and improve symptoms (S5.3.2.2-6, S5.3.2.2-7).

Digoxin-specific antibody (Fab)

- It is a monovalent immunoglobulin that rapidly binds to intravascular digoxin.
- Clinical response rates to digoxin Fab are as high as 80% to 90%, particularly in the acute setting

IIa	C-LD	1. In patients with bradycardia associated with symptoms or hemodynamic compromise in the setting of digoxin toxicity, digoxin Fab antibody fragment is reasonable to increase heart rate and improve symptoms (S5.3.2.3-1–S5.3.2.3-8).

Methylxanthines

- The methylxanthines -theophylline and aminophylline- exert positive chronotropic effects on the heart, likely mediated by inhibition of the suppressive effects of adenosine on the sinoatrial node.

IIa	C-LD	1. In post-heart transplant patients, aminophylline or theophylline is reasonable to increase heart rate if clinically indicated (S5.3.2.4-1–S5.3.2.4-4).
IIa	C-LD	2. In patients with SND associated with symptoms or hemodynamic compromise in the setting of acute spinal cord injury, aminophylline or theophylline is reasonable to increase heart rate and improve symptoms (S5.3.2.4-5–S5.3.2.4-7).

Aminophylline- AV Block

- Aminophylline is a nonselective adenosine receptor antagonist and phosphodiesterase inhibitor.
- Experimental evidence suggests a role of increased adenosine production in development of AV Block in acute inferior MI.

IIb	C-LD	3. For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise in the setting of acute inferior MI, intravenous aminophylline may be considered to improve atrioventricular conduction, increase ventricular rate, and improve symptoms (S6.3.2-8–S6.3.2-11).

Second- or third-degree atrioventricular block associated with acute inferior MI		
Aminophylline	250-mg IV bolus	

Transcutaneous Pacing

- It was reported in 1952 and became commercially available in the early 1980s.
- It is now universally available in combination with external defibrillators.

Transcutaneous Pacing

- No improvement in survival to hospital discharge when used in the prehospital phase of bradyasystolic cardiac arrest
- Its use appears to be greater when applied to patients with a perfusing rhythm or early in the course of cardiac arrest.

Transcutaneous Pacing

- Analgesic and/or anxiolytic agents should be considered in conscious patients.
- Early preparation has shown to be effective in the perioperative setting for rapid treatment of bradycardia.

Transcutaneous Pacing Limitations

- Assessment of myocardial capture by ECG alone may be difficult and should be confirmed by assessment of pulse or intra-arterial pressure.
- Because prolonged use of transcutaneous pacing may be unreliable and poorly tolerated, it should generally serve as a short-term bridge to temporary or permanent transvenous pacing or resolution of bradycardia.

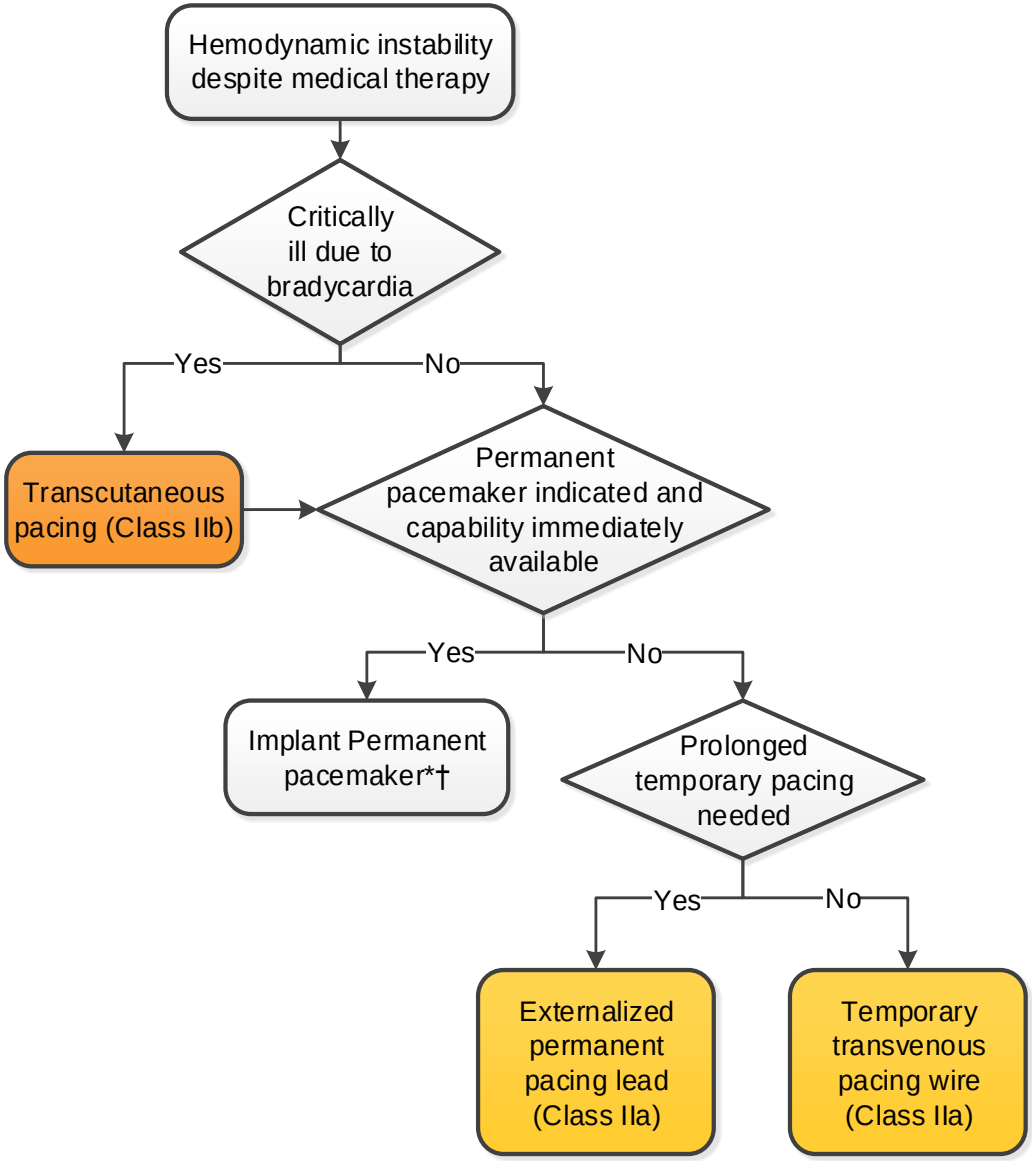
Temporary Pacing

Recommendations for Temporary Pacing for Bradycardia Attributable to SND		
COR	**LOE**	**Recommendation**
IIa	**C-LD**	**In patients with persistent hemodynamically unstable SND refractory to medical therapy, temporary transvenous pacing is reasonable to increase heart rate and improve symptoms until a PPM is placed or the bradycardia resolves.**
IIb	**C-LD**	**In patients with SND with severe symptoms or hemodynamic compromise, temporary transcutaneous pacing may be considered to increase heart rate and improve symptoms until a temporary transvenous or PPM is placed or the bradycardia resolves.**
III: Harm	**C-LD**	**In patients with SND with minimal and/or infrequent symptoms without hemodynamic compromise, temporary transcutaneous or transvenous pacing should not be performed.**

Temporary Pacing for Atrioventricular Block

Recommendations for Temporary Pacing for Bradycardia Attributable to Atrioventricular Block		
COR	**LOE**	**Recommendation**
IIa	**B-NR**	**For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise that is refractory to medical therapy, temporary transvenous pacing is reasonable to increase heart rate and improve symptoms.**
IIa	**B-NR**	**For patients who require prolonged temporary transvenous pacing, it is reasonable to choose an externalized permanent active fixation lead over a standard passive fixation temporary pacing lead.**
IIb	**B-R**	**For patients with second-degree or third-degree atrioventricular block and hemodynamic compromise refractory to antibradycardic medical therapy, temporary transcutaneous pacing may be considered until a temporary transvenous or PPM is placed or the bradyarrhythmia resolves.**

Acute Pacing Algorithm



The efficacy of transcutaneous cardiac pacing in ED

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ABSTRACT

Introduction: Transcutaneous cardiac pacing (TCP) is a rapid, time-saving, and noninvasive ventricular stimulation that is tolerated by conscious patients despite the painful intervention for treatment of symptomatic bradycardias. The goal of this study was to determine the efficacy of TCP in unstable bradycardia patients in emergency department (ED).

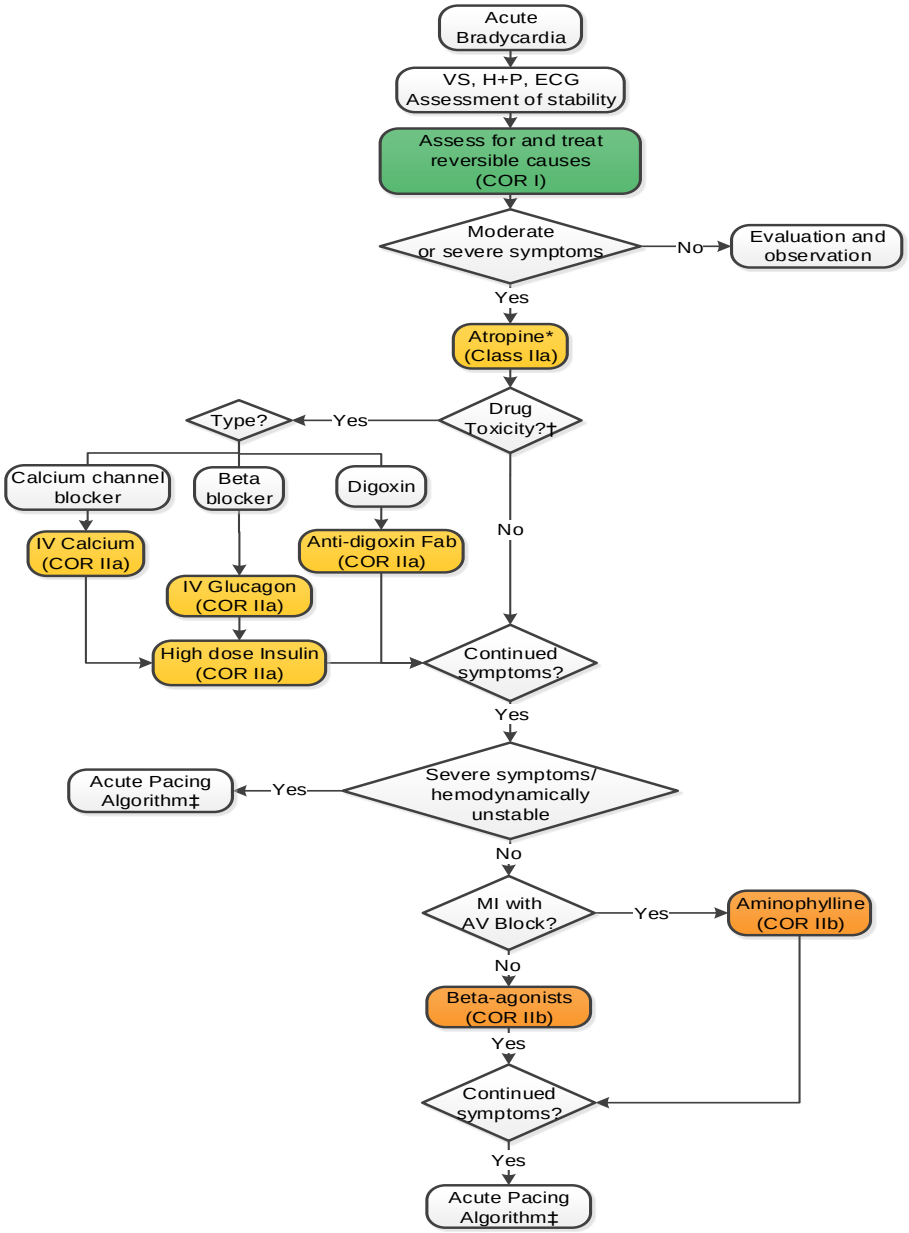
Methods: This single-central, observational clinical study was conducted on patients older than 18 years who presented with acute unstable bradycardia to the tertiary care university ED. Primary outcome measure was to determine the efficacy of TCP in unstable bradycardia patients in the emergency settings. Efficacy of TCP was to determine changes of clinically significant vital signs and electrocardiography.

Results: Of 349 patients who visited the ED presenting with bradycardia, 89 patients who met the criteria were included in the study. There was a statistically significant difference between before and after the first administration TCP in mean systolic (71.2 [64.8-77.6] and 105.3 [97.6-112.9 mm Hg]) and diastolic blood pressure (42.9 [38.8-47.0] and 61.0 [56.4-65.5] mm Hg) and median heart rate (40 [39-42] and 74 [71-78] beats/min, $P < .0001$).

Conclusion: Transcutaneous cardiac pacing is a clinically effective treatment modality in patients with atropine-resistant unstable bradycardia.

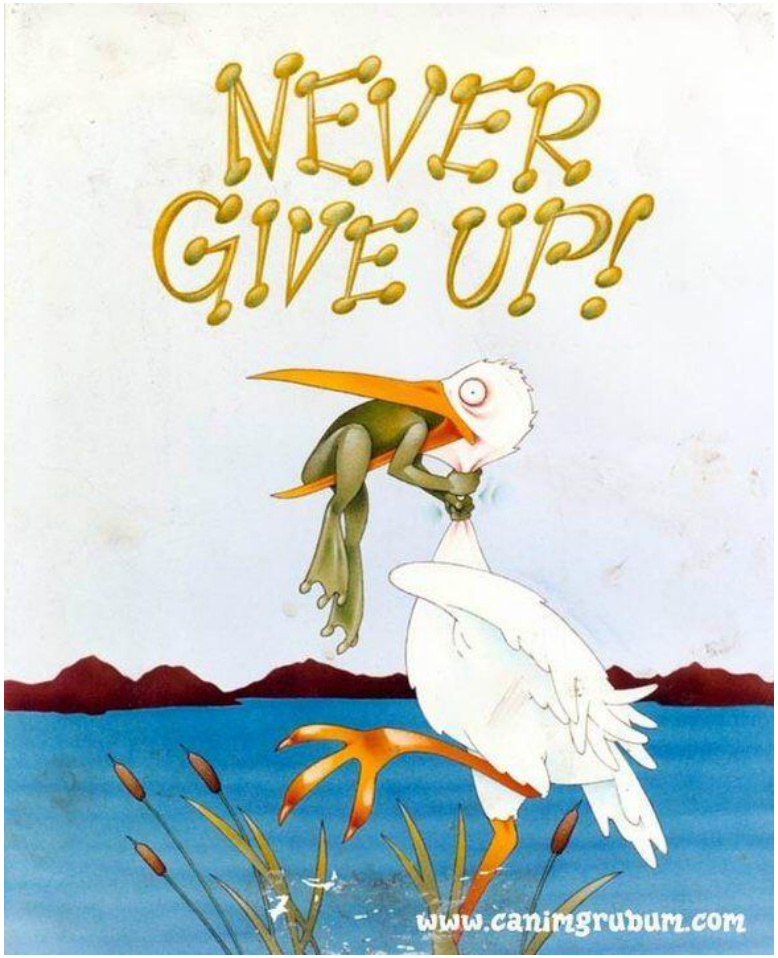
- 349 patients with bradycardia
- 32% of patients responded to atropine
- No differences between mortality rates in TCP and non-TCP groups
- Significant increase in blood pressure in TCP group
- Mortality rates were lower in dopamin+TCP than only TCP group

Acute Bradycardia Algorithm



Take Home Messagges

- Drugs and external pacing are combined consecutive treatment options.
- TCP is more useful in patients with perfusing rhythm than cardiac arrest.
- If there is no iv access, use TCP
- Progress quickly through these actions as the patient could be in pre-cardiac arrest and need multiple interventions done in rapid succession.



Thank You

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