University of CINCINNATI Emergency Medicine

# ANNALS OF B POD

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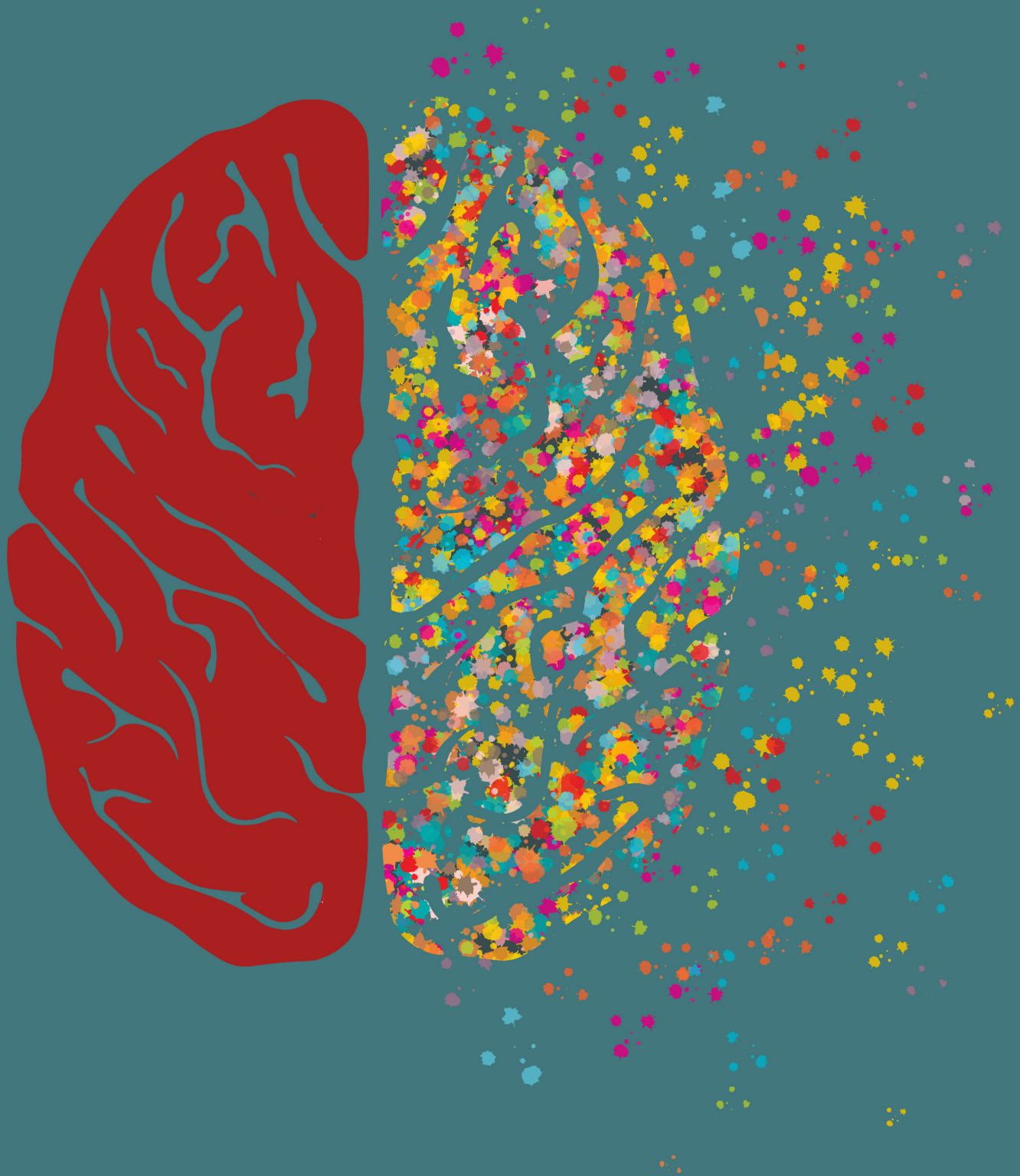

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*Since 2008*

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# SUBCLAVIAN STEAL SYNDROME

Payton Leech
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## History of Present Illness

The patient is a female in her 40s who presents to the emergency department (ED) with a chief complaint of an intermittent headache and vertigo for the past week. She describes it as a pressure-like pain over the frontal portion of her head that is sometimes relieved with naproxen. She has associated left-sided neck pain that is worse when turning her head to the right, sometimes associated with intermittent numbness and tingling in her left arm. Review of systems identifies intermittent nausea and "a burning" substernal chest pain that started with the other symptoms as well. She specifically denies photophobia, phonophobia, extremity weakness, and vomiting.

## Past Medical History

GERD
Hidradenitis suppurativa
Obesity

## Past Surgical History

Tubal Ligation

## Medications

Cyclobenzaprine
Duloxetine
Ibuprofen
Ranitidine

## Allergies

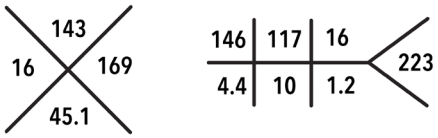
No known drug allergies

## Physical Exam

T 98.3 F
HR 87
BP 98/50
RR 18
SpO2 100% on RA

The patient is awake and alert, sitting in bed in no acute distress. Cardiopulmonary examination is unremarkable. Pupils are equal and reactive to light. Her neurologic exam is notable for intact cranial nerve function, extraocular movements, and visual fields to full confrontation. She has full strength in her upper and lower extremities with sensation intact to light touch in all nerve distributions. Gait is narrow and stable. Blood pressures taken in both upper extremities were noted to be the following: left upper extremity 70/40, right upper extremity 120/80. Left radial and brachial pulses were difficult to palpate, however they were detectable with doppler signal.

## Diagnostic Tests




Troponin - <0.04 / BNP - 16

EKG - Normal sinus rhythm,

CT head, CTA head and neck: left subclavian artery thrombosis with reconstitution of blood flow distally

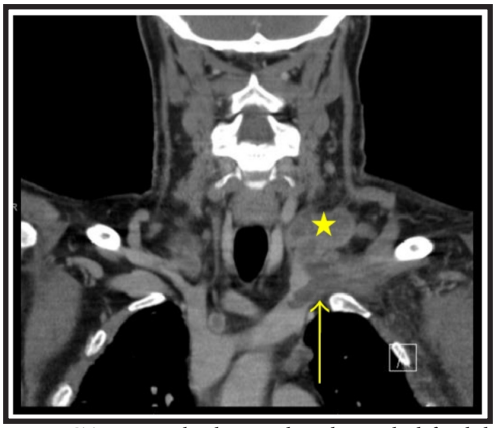


*Image 1: CT angiography showing thrombus in the left subclavian vein*

## Hospital Course

The patient was started on a heparin drip and admitted to vascular surgery with a neurology consult for her left subclavian artery thrombosis. Her headache and dizziness improved during the course of her admission. She had a negative MRI of her brain and neurology attributed her symptoms to Subclavian Steal Syndrome. Her hypercoagulability workup was negative, and she was discharged on rivaroxaban with vascular surgery follow up for consideration of subclavian bypass versus conservative management. At her outpatient visit, the patient had palpable pulses in bilateral upper extremities and complete resolution of her left upper extremity claudication and vertebrobasilar insufficiency symptoms. Thus, surgery was deferred, and she elected to proceed with medical management.

## Discussion

### Pathophysiology and Epidemiology

Subclavian Steal Syndrome (SSS) emerges as a compensatory phe

**Subclavian Steal Syndrome**
*Continued on page 15*

# ELECTROCUTION INJURY

Hamza Ijaz, MD
*University of Cincinnati R2*

## History of Present Illness

The patient is a middle-aged male who presents in cardiac arrest after a suspected electrocution injury. The patient was holding an aluminum ladder when it struck a powerline, and he became unresponsive. Bystander cardiopulmonary resuscitation (CPR) was initiated and 911 was called. Upon Emergency Medical Services (EMS) arrival, the patient was pulseless, apneic, and unresponsive. Advanced Cardiac Life Support (ACLS) protocol was initiated by ground EMS providers, and the patient was determined to be in ventricular fibrillation (VF). EMS delivered five defibrillations and established an intraosseous line in the right humerus to administer four rounds of code-dose epinephrine and two doses of amiodarone per ACLS protocol. An attempt at intubation was unsuccessful due to oropharyngeal secretions obstructing visualization, and resuscitation was continued with a supraglottic device in place. Despite these interventions, the patient was in refractory VF on Air Care arrival.

## Past Medical History

Unknown

## Past Surgical History

Unknown

## Medications

Unknown

## Allergies

Unknown

## Physical exam

T
N/A

HR
Pulseless

BP
N/A

RR
N/A

SpO2
100% via supraglottic device

The patient is an unresponsive male in cardiac arrest who was undergoing CPR via a mechanical device. The patient has a supraglottic device in place without signs of tracheal deviation. Breath sounds are clear and equal bilaterally. The head exhibits no signs of trauma. The pelvis is stable. No lesions are present on the chest or abdomen. There is a white and black circular eschar on the left lower extremity.

## Diagnostics

Glucose 331 mg/dL

## Hospital Course

At the first pulse check upon the Air Care crew's arrival, the patient remained in VF, receiving a sixth shock. As the patient was in refractory VF, dual-sequential defibrillation at 200 Joules was attempted leading to asystole at the next pulse check. CPR was resumed and epinephrine 1 mg and calcium chloride 1 ampule were administered. The patient remained in asystole at the next pulse check, and end-tidal CO2 capnography was noted to be down-trending. At this time, further resuscitative efforts were determined to be futile and the patient was pronounced dead with family present at the patient's side.

## Discussion

### Background

Approximately 6,500 injuries and 1,000 deaths occur annually in the United States (US) as the result of electrocution.[1] Of these deaths, less than 30% (50 - 300) are caused by lightning strikes.[2] The extent of injury is dependent on the type, duration, and voltage.[3] Injuries sustained from a higher voltage mechanism or longer duration of exposure tend to be more severe.

### Charge

Charge is the fundamental unit of electricity, measured by the unit coulomb (C). The coulomb is equal to the charge carried by $6.24 \times 10^{18}$ electrons. It should be noted that one C of charge does not specifically refer to electrons as many particles can carry a charge, with many particles having a greater capacity than an electron. For example, in a copper wire electrons are used to carry current; however, in the human body sodium, chloride, and potassium may be used to carry a charge as the body does not have free electrons.

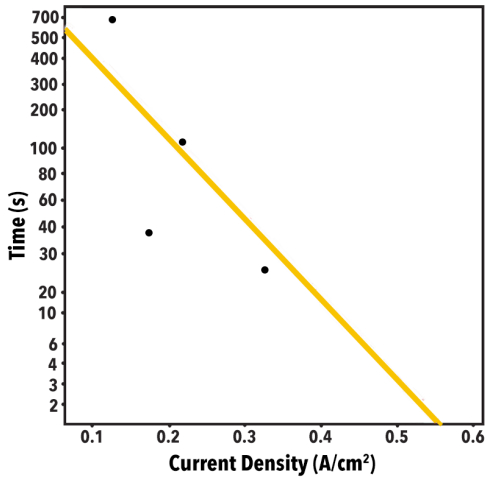



*Figure 1: The time to epidermal necrosis varies inversely with the exponent of the current density.*

### Current, Voltage and Resistance

Electrical current is the flow of charge and is measured in ampere (A). One A is equivalent to one C ($6.24 \times 10^{18}$ electron if referring to a copper wire) of charge per one second (1 C/second). Electrical current density, or the strength of current per unit area, is directly associated with tissue injury as demonstrated by Sances et al. Figure 1 shows the relationship between increased current density and decreased time to tissue necrosis. Paradoxically, de-

creased current density was associated with increased temperature achieved presumable secondary to longer exposure time.[4]

There are two types of current: direct current (DC) and alternating current (AC). DC flows unidirectional while AC flows cyclically. AC is typically used for household and industrial electrical power with a frequency of 50 or 60 Hz.[1] The cyclical nature of AC can lead to tetanic contractions resulting in unintentional grasping of the current and increased exposure to the electricity.[3] As a result, AC current can cause more severe injuries with a lower voltage and duration of exposure.

Voltage is the electrical potential difference between two points, or the pressure that forces a current along a path, measured by volts (V). A volt was originally defined by the volts generated by a standard battery but is now based on a solid-state circuit and defined as the difference in electric potential between two points when the electric current of one A dissipates one watt (W) of power. This is equivalent to a potential difference that generates one joule (J) of energy per C of charge passing through it. The amount of electrical current delivered can be characterized as either high-voltage (>1000 V) or low-voltage (<1000 V). Household outlets in the US carry 120 V AC whereas powerlines carry approximately 7000 V AC.

$$V = \frac{\text{potential energy}}{\text{charge}} = \frac{J}{C} = \frac{W}{C}$$

Resistance is the impedance to flow and is measured in ohms (Ω). One Ω of resistance is equivalent to the resistance present in a circuit when one V of pressure is required to generate one A of current. Under dry conditions the resistance offered by the human body is 100,000 Ω; however, moisture or skin breakdown can decrease the human body's resistance to 1kΩ (1000 Ω). Difference tissues within the body have difference tissue resistivity (resistance of a 1 $cm^3$ cube of the tissue, measured as Ωcm). For example, the resistivity of blood is 150 Ωcm, fat is 2,200 Ωcm, and bone is 10,000 Ωcm.[5]

### Power and Energy

Power, measured in watts (W), is equal to flow rate (A) times pressure (V). Energy is the product of power (W) and time (seconds).

### Mechanisms of Injury

Electrocution injuries vary in location and extent, including direct tissue damage, systemic consequences, and mechanical injury secondary to a fall, tetany, or other trauma.[1] One of the most severe injury patterns caused by electrocution is an electrical arc injury. Electricity at very high voltages arcs from one conductor to another, causing flash burns (>20,000°C), electrical damage, and physical damage from the blast force.[3] High voltage injury also results in musculoskeletal and soft tissue death, putting patients at increased risk for developing rhabdomyolysis and compartment syndrome. Soft tissue burns can range from small and painless burns to extensive electrical burns requiring admission and management from a burn specialist.

Tetanic contraction leading to musculoskeletal injury is another common injury pattern associated with high-voltage electrocution. When AC current flows through the victim's muscles, it results in direct electrical stimulation with the potential to cause fractures and joint dislocations.[6] Atypical injury patterns, such as posterior shoulder dislocations, can occur.

Lethal ventricular arrythmias are the electrical injury of greatest concern. Cardiac arrhythmias are a common cardiovascular manifestation, with the most common fatal arrhythmias being VF and ventricular tachycardia (VT). VF occurs through three different mechanism in electrical injury: (1) shock on T, (2) rapid cardiac capture, and (3) long term cardiac capture.[7-11]

Shock on T is caused by a single shock that occurs during the T wave, a period of ventricular relaxation where the cardiac myocytes return to their resting states. In the middle of the T wave some of the cardiac myocytes have returned to their resting state while some remain active. This is a vulnerable period of the cardiac electrical cycle as an electrical shock during this period can lead to unpredictable electrical activity within the heart and ultimately cause VF. According to the IEC (International Electrotechnical Commission) there is a 50% probability of VF with a 5,000 μC unidirectional impulse into the T-wave.[12]

Direct rapid cardiac capture is the result of strong repetitive currents which capture the cardiac myocytes at a high rate. Rapid cardiac capture (~450 BPM) leads to VF through the process of wavebreak. Wavebreak occurs when there is electrophysiologic heterogeneity of the cardiac myocytes, allowing for propagation of electrical activity in some regions while encountering refractory tissue in other regions. Direct rapid cardiac induction of VF occurs typically within in 0.1–5 s with the current required decreasing rapidly after the VF threshold has been surpassed.[9-11]

Long-term fast cardiac capture describes a phenomenon where lower (~40% of direct rapid cardiac capture) current densities lead to VF after a longer duration (~90 seconds). These rates remain significantly higher than that required of pacing (> 220) in swine models. The prolonged fast cardiac capture leads to ischemia of the cardiac myocytes, lowering the VF threshold to ~40% of the threshold of direct rapid cardiac capture.[11]

Given the low resistance of nerve tissue, the neurologic system is vulnerable to high-voltage electrical injuries.[13] Patients are at risk for seizures, spinal cord injury, peripheral paresthesias and neuropathies following electrical injury. These symptoms can be delayed, manifesting days to months after the electrocution injury.[1]

### Scene & Prehospital Care

First responders must focus on maintaining their distance from sources of electricity, keeping scene safety a top priority when treating patients suffering from electrical injuries. Providers must also remain aware of potential conductors through which electricity can travel, such as water, metal, and even dry wood at high voltages. Similar to caring for other patients in the pre-hospital setting, providers must weigh the benefit of on-scene resuscitation versus early transport when caring for patients suffering electrical injury. Definitive care for patients in cardiac arrest after electrical injury includes high-quality CPR and defibrillation, with a potential role for dual-sequential defibrillation and esmolol given the refractory VF.

**Electrocution Injury**
*Continued on page 13*

# TCA Overdose

Carl Goff, MD
*University of Cincinnati R2*

## History of Present Illness

A 57-year-old man arrives to the emergency department (ED) via emergency medical services (EMS) with altered mental status and concern for intentional overdose of amitriptyline, a tricyclic antidepressant (TCA). EMS providers state the patient was found outside of an abandoned building. He was initially alert in the company of a bystander who reported the patient made suicidal statements and took an estimated 10 – 20 100 mg tablets of amitriptyline which he is prescribed for depression. The patient is obtunded on arrival to the ED and is unable to provider further history.

### Past Medical History

Hypertension
Osteoarthritis
Degenerative Disc Disease
Chronic pain
Depression
Schizophrenia
Hypogonadism / Erectile Dysfunction

### Allergies

Imitrex
Morphine
Vicodin

### Social History

Retired / disabled roofer
Most close family members deceased
Smokes cigars daily
Occasional alcohol use abuse

### Medications

Alprazolam
Amitriptyline
Buspirone
Olanzapine
Gabapentin
Omeprazole
Sildenafil

### Past Surgical History

Noncontributory

### Family History

Imitrex
Morphine

## Physical Exam

T 36.5
HR 114
BP 124/78
RR 18
SpO2 97% on RA

The patient is obtunded. His airway is patent, but he has poor airway reflexes. He is tachypneic with shallow respirations. The patient is tachycardic, with strong central and peripheral pulses. Pupils are mid-range, equal, round and reactive to light. He is noted to move all extremities symmetrically with strength grossly intact and no obvious focal neurologic deficits. There are no external signs of trauma. He has moist mucous membranes. His skin is warm and dry. The neck is supple without meningismus. The abdomen is soft and non-distended. Extremities are without edema or signs of trauma signs of trauma.

## Notable Diagnostics

Potassium: 3.2
UDS: + Cocaine, TCA
Imaging: Negative CT head and CXR

Initial EKG:

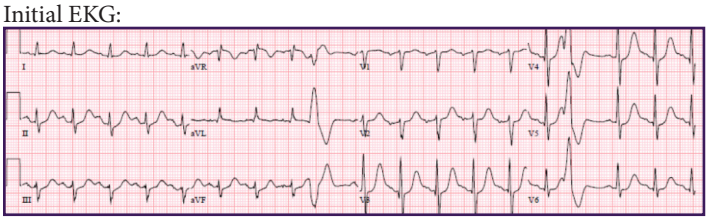


*Figure 2: ECG upon arrival demonstrating a: prolonged QRS and QT intervals and a terminal R wave in lead*

## Emergency Department Course

Upon presentation to the ED the patient is hypertensive and tachycardic with a decreased level of consciousness requiring intubation for airway protection. His ECG demonstrates prolonged QRS and QT intervals and a terminal R wave in lead aVR suggestive of severe sodium channel blockade. The patient is administered 1 ampule of 8.4% (50 mEq) sodium bicarbonate followed by a bicarbonate infusion at 100 mL/hr. Repeat ECG demonstrates improvement in the previously demonstrated derangements. Hypokalemia is noted and is corrected with IV potassium chloride. Head CT is unremarkable. Ultimately, the patient is admitted to the medical intensive care unit (MICU).

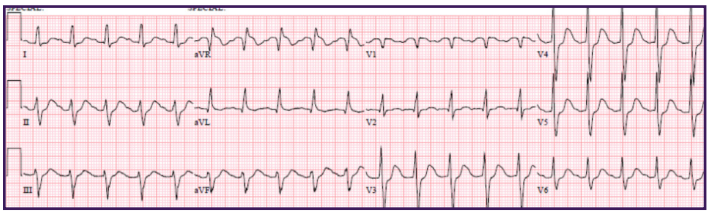


*Figure 3: EKG after bolus administration of sodium bicarbonate*

## Hospital Course

The patient was admitted to the MICU where he developed severe refractory shock with a blood pressure nadir of 47/28 mmHg. He was aggressively resuscitated with IV fluids, but ultimately required norepinephrine, epinephrine, vasopressin, phenylephrine and methylene blue for blood pressure support. He was continued on a sodium bicarbonate infusion and required multiple doses of calcium chloride. Stress-dose steroids were administered. His hemodynamics stabilized with the above measures and the patient was weaned from vasopressors over several days. Ultimately the patient was extubated and transferred out of the ICU.

The patient's subsequent clinical course was complicated by several factors. He was evaluated by psychiatry for major depression and suicidality. His psychiatric medications were discontinued with a plan to re-evaluate his regimen upon recovery from his acute illness. He suffered from multisystem organ dysfunction attributed to refractory shock and prolonged use of high dose vasopressors. Complications included nonischemic cardiomyopathy, acute kidney injury secondary to acute tubular necrosis, shock liver, peripheral vascular compromise and pancytopenia. All these abnormalities resolved spontaneously over the course of his admission. He was ultimately discharged from the hospital to an inpatient rehabilitation facility after a 23-day admission.

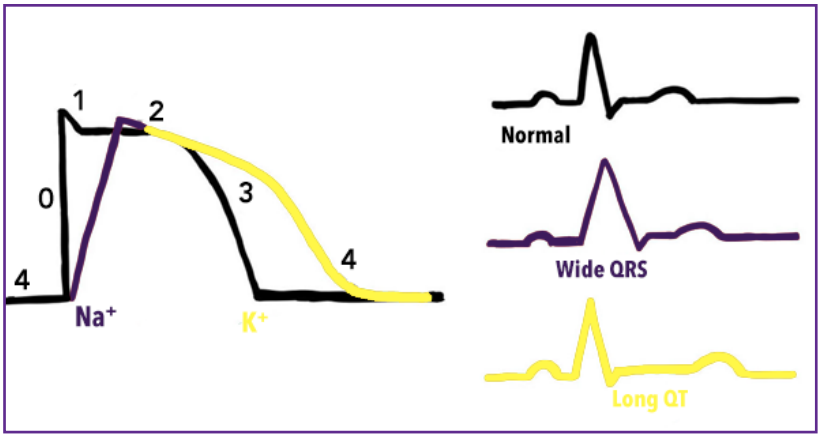



*Figure 4: The cardiac myocyte action potential and the effects of sodium and potassium*

## Discussion

### Epidemiology and Clinical Applications of TCAs

Tricyclic antidepressant (TCA) medications emerged in the latter half of the 20th century as potent antidepressants. Unfortunately, they were
noted to have significant risk of harm when taken in overdose and fell out of favor with the development of safer alternatives including selective serotonin reuptake inhibitors (SSRIs). In the United States, from 1983 – 2003, the rate of overdose with antidepressant medications increased from 0.61 to 3.26 per 10,000 people, however the fatality rate dropped from 73 to 32 per 10,000 people. This discordance is proposed to reflect a reduction in prescribing of TCAs in favor of alternative medications.[1]

Despite their risks, TCAs remain within the armamentarium for the treatment of major depressive disorder (MDD) as they are inexpensive and effective. In the United States, MDD afflicts 7% of adults, 13% of adolescents[2] and an estimated 1-2% of these patients currently take TCAs as monotherapy or in combination with SSRIs.[3,4] Additionally, TCAs have utility in psychiatric conditions beyond MDD including obsessive-compulsive disorder, panic disorder, post-traumatic stress disorder, bulimia and insomnia.[5] TCAs have gained acceptance as a modality for pain syndromes including migraine, neuropathy, chronic lower back pain, fibromyalgia and chronic abdominopelvic pain.[6] This highlights the fact that a significant number of patients continue to be prescribed TCAs.

### Pharmacology

TCAs are rapidly absorbed from the gastrointestinal tract. They are 90-95% protein bound to albumin at therapeutic plasma concentrations and have a large volume of distribution. Onset of therapeutic effects occur at 2-8 hours with therapeutic plasma levels at 50 – 300 ng/mL-1. Behavioral and cognitive abnormalities including agitation, confusion and memory impairment begin to occur at plasma levels over 450 ng/mL-1. Life threatening toxicity occurs at serum levels greater than 1000 ng/mL-1.[4]

TCA medications are metabolized in the liver via the CYP450 system, producing several active metabolites (e.g. amitriptyline is metabolized to nortriptyline). TCA medications have a long elimination half-life of greater than 24 hours (amitriptyline: 31 – 46 hours) and are excreted in the urine.[4] The therapeutic effect of TCA medications in depression is derived from their ability to inhibit reuptake of norepinephrine and serotonin at the pre-synaptic membrane. This increases the concentration of these neurotransmitters available for post synaptic receptor binding, resulting in the downstream effects which manifest clinically as a reduction in depressive symptoms.[4] However, TCAs have many other sites of action including histamine (H1) antagonism, alpha adrenergic (α1) antagonism, GABAA antagonism, muscarinic receptor antagonism, voltage gated sodium channel (Nav) blockade and cardiac rectifier potassium channel (IKr) blockade.[7-11] This leads to variable and profound effects in overdose, but commonly includes depressed mental status, hyperthermia, seizure, tachycardia, QT prolongation, QRS prolongation, tachyarrhythmias, VT/VF and shock. A summary of both receptor site and side effects of TCA overdose can be found in Table 1.

In overdose, the anticholinergic and histaminergic properties of TCA medications may result in slowing of gastric emptying leading to prolonged absorption via enterohepatic recirculation.[7] The amount of free (biologically active) drug in vivo is dependent on serum pH. This is because protein-binding of TCAs exists in a pH-dependent equilibrium, where the amount of biologically active drug is inversely proportional to the fraction of protein-bound drug. As the pharmacologic action of TCAs in overdose tend to potentiate both respiratory and metabolic acidosis, the amount of free drug increases exponentially.[7]

Patients with TCA overdose characteristically deteriorate in impressive fashion, as the physiologic derangements are synergistic. Depressed mental status results in ventilatory compromise and respiratory acidosis. Metabolic acidosis develops due to lactate production from a mixed cardiogenic and vasoplegic shock. Patients are at risk for seizures, which can further worsen the lactic acidosis. As the acidosis progresses, a greater fraction of unbound metabolically active drug enters the systemic circulation and perpetuates toxic effects.[12]

It has been suggested that ingestions of TCAs less than 20 mg/kg

| Site of Action | Clinical manifestation of toxicity |
|---|---|
| 5-HTT | Sedation, serotonin syndrome: tremor, myoclonus, hyperthermia, arrhythmia |
| NET | Tachyarrhythmia, seizure |
| $M_1$ | Sedation, agitation, coma, seizure, respiratory depression, hyperthermia, tachyarrhythmia, anhidrosis, ileus, urinary retention, dry mouth, blurred vision |
| $H_1$ | Sedation |
| $\alpha_1$ | Hypotension |
| $GABA_A$ | Seizures |
| $Na_V$/HERG | Cardiac conduction abnormalities: prolonged QRS and QT, arrhythmias |

*Table 1: Site of action and clinical manifestations of TCA toxicity. 5-HTT: serotonin reuptake receptor; NET: norepinephrine reuptake receptor; M1: muscarinic 1 receptor; H1: Histamine 1 receptor; α1: alpha 1 adrenergic receptor; GABAa: GABA a receptor; Nav / HERG: voltage gated sodium channel / potassium rectifier channel*

are unlikely to be clinically significant. However, variability in indi-

**TCA Overdose**
*Continued on page 14*

# Jaw Dislocation

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## History of Present Illness

The patient is a female in her 20s with a past medical history of asthma and multiple facial surgeries who presents to the emergency department with jaw pain. The patient reports that she previously sustained a mandibular fracture after blunt facial trauma from a line-drive softball three years ago and has had multiple temporomandibular joint (TMJ) dislocations and oromaxillofacial surgeries since this event. She is undergoing continuing evaluation with oral maxillofacial surgery (OMFS) for further management of her recurrent dislocations. Her current episode of discomfort started yesterday evening. She endorses persistent right jaw pain, and feels that her jaw is dislocated on this side. Patient denies difficulty managing her secretions, and endorses no shortness of breath, chest pain, lightheadedness, or dizziness. There was no precipitating trauma yesterday to cause her current discomfort.

## Past Medical History

Asthma
Recurrent TMJ dislocation

## Past Surgical History

Multiple mandibular surgeries with OMFS after mandibular fracture

## Social History

Denies tobacco, alcohol, or drug use

## Physical Exam

T 36.6 C°
HR 78
BP 128/84
RR 18
SpO2 99% on RA

The patient is a well-nourished female on no acute distress. There are no outward signs of trauma on examination of the head. On head, eyes, ears, nose, and throat exam, the patient's mandible is deviated to the right with respect to the maxilla. There is malocclusion of her dentition. Her mucous membranes are moist, and she has no respiratory distress, stridor, or drooling on examination. Patient has difficulty opening her mouth, limiting examination of the oropharynx. The neck is supple and the trachea is midline. Clear and equal breath sounds are noted bilaterally. The remainder of the physical exam is unremarkable

## Hospital Course

The patient's physical exam findings of right mandibular deviation and reported history of multiple jaw dislocations after mandibular fracture are concerning for recurrent left TMJ dislocation. She is in no respiratory distress and are vitally stable without signs of airway compromise. Non-contrast maxillofacial CT is ordered to further assess her dislocation for accompanying fracture, and OMFS is consulted given her complex operative history.

Maxillofacial CT revealed no acute mandibular fracture. This study additionally demonstrated findings of repetitive dislocation of the left TMJ with remodeling of the condylar head and articular eminence, along with malocclusion and rightward displacement of the mandible with respect to the maxilla. O M F S consult recommended transfer to their outpatient clinic for further management and reduction of the jaw. Patient was discharged from the emergency department to the OMFS clinic for further treatment and had successful reduction of her dislocation.

## Discussion

Temporomandibular joint dislocation is an important entity for emergency physicians to recognize, as it causes significant discomfort to patients and is potentially reducible in the emergency department with certain techniques. The mandible most commonly dislocates in the anterior direction, as the superior border of the glenoid fossa and the posterior border of the external acoustic meatus prevent dislocation without accompanying fracture of these bones.1 Spontaneous anterior dislocations tend to occur with movements that hyperextend the mouth widely, such as with yawning or chewing (see table 1). Medical conditions that cause ligamentous laxity, including Marfan Syndrome or Ehlers-Danlos Syndrome, may predispose the patient to jaw dislocation. Dystonic movements from medications or underlying medical conditions like Huntington's disease or epilepsy may also cause jaw dislocation to occur in certain patients.[2] Posterior, lateral, or superior joint dislocations often result from fracture of the bordering bones of the joint space or the mandible itself.[3]

| Causes of Jaw Dislocations | |
|---|---|
| Iatrogenic | Intubation, dental procedures, endoscopy |
| Traumatic | High energy trauma causing fractures of the boundaries of the joint space such as the glenoid fossa |
| Systemic Disease | Ligamentous laxity – Ehlers-Danlos, Marfan syndrome<br>Dystonia – Huntington's disease, Parkinson's disease, epilepsy, muscular dystrophies, tetanus infection |
| Medications | Antipsychotics, phenothiazines |
| Spontaneous | Yawning, taking large bites, laughing, singing, oral sex, vomiting |

*Table 2: Causes of jaw dislocation*

Anatomically, anterior TMJ dislocation occurs when the head of the mandible slides out of the mandibular fossa and locks in front of the articular eminence. The resulting stretch causes spasm of the masseter, pterygoid, and temporalis muscles, which locks the jaw out of place in an open position. Patients with bilateral anterior jaw dislocation typically present to the ED holding their mouth open. In unilateral jaw dislocation, the chin deviates away from the area of dislocation.[3] The patient's speech may be difficult to understand, and they may demonstrate drooling on physical examination. Palpation of the preauricular space is typically painful, and the empty

fossae may be palpable on examination along with the anteriorly displaced coronoid process of the mandible. These patients are usually uncomfortable but nontoxic-appearing and afebrile with normal vital signs.

Prior to reduction of the dislocation, imaging should be obtained to evaluate for accompanying fracture. Jaw radiographs may be appropriate, especially when there is no history of trauma, or in pediatric patients to minimize exposure to radiation.[4] Maxillofacial CTs should be completed if there is a history of traumatic injury to evaluate for any accompanying injuries or fractures.[5]

Several techniques for reduction in the emergency department have been described in the literature. Adequate analgesia and anxiolysis can facilitate both reduction and patient cooperation with certain reduction maneuvers. One of the most commonly discussed techniques for TMJ reduction is the syringe method, which was developed in 2014.[6] In the population studied, 97% of patients had successful reduction, the majority of which occurred within one minute.[6] To accomplish this method, a syringe is placed in the patient's mouth near the posterior molars on the affected side. The patient is then instructed to bite down gently and roll the syringe back and forth between the jaws, encouraging the jaw to slide back into place. The syringe size is chosen based on the largest size that can be accommodated by the patient's mouth while engaging the posterior molars (most commonly a 5 or 10cc syringe is used). This method is a preferred starting point for many as it requires minimal analgesia and does not require the provider to place their hands in the patient's mouth for reduction.

*Syringe Technique*

*Conventional Intraoral Technique*

*Wrist Pivot Technique*

*Extraoral Technique*

Other methods require provider hand placement in the oral cavity to guide the mandible into place. With these techniques, care should be taken to prevent injury to the provider, as the patient's jaw muscles can contract and cause bite injuries and subsequent infection. Tongue blades taped to the anterior and posterior surfaces of the clinician's thumbs or alternatively, finger splints can be used to protect the fingers for the intraoral method. Suction should be at the ready for these techniques. Intraoral manual reduction is conventionally completed from the front of the patient3. With this technique, an assistant should stabilize the patient's head from behind while the provider places their thumbs either on the occlusal surfaces of the mandibular molars or on the gum surface of the mandible just posterior to the molars. The fingers grasp the chin near the angle of the mandible, and the provider exerts a posterior and inferior force on the mandible to encourage the mandibular head to slide back under the articular eminence. Unconventionally, the provider can stand behind a seated patient and place the patient's head against his or her abdomen while placing the thumbs in a similar position with fingers grasped around the chin.[7] Again, downward pressure is applied similarly to encourage reduction of the TMJ. In both cases, the provider may need to facilitate muscle fatigue by exerting continuous pressure or gently rocking the jaw back and forth to relax the muscles. If unsuccessful, auriculotemporal nerve block or local infiltration of anesthetic into the empty joint space may further assist with pain control and promote reduction.[8]

The wrist-pivot method is another option for reduction.[9,10] With this technique, the provider's thumbs are placed on the mental eminence of the mandible with the rest of the fingers placed on the occlusal surfaces of the mandibular molars. The provider's fingers provide inferior-directed pressure while the wrists ulnar deviate and flex to slide the condyles under the articular eminence back into the glenoid fossa.

A technique in addition to the syringe method that avoids intraoral placement of the provider's hands involves palpation of the anteriorly dislocated coronoid process.[11] On one side of the patient, the fingers grasp the angle of the mandible with the thumb over the malar eminence and direct the jaw anteriorly. This causes further dislocation of the ipsilateral joint, but facilitates reduction of the contralateral TMJ. On this contralateral side, the thumb provides continuous downward pressure on the coronoid process while the fingers grip the mastoid process as counterpressure to encourage reduction of the joint. This method may be more uncomfortable for patients given the sustained pressure on bony surfaces and further dislocation of the ipsilateral joint as described above, but was found to be successful in the reduction of dislocation in seven patients for whom traditional intraoral techniques had failed.[11]

A recent randomized clinical trial in 2016 compared the success of the conventional intraoral, wrist pivot, and extraoral methods.[12]Their relative success rates were found to be 86.7%, 96.7%, and 66.7% respectively using randomly allocated groups.[12] The syringe

**Jaw Dislocation**
*Continued from page 12*

# TYLENOL OVERDOSE

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## History of Present Illness

The patient was a twenty-one-year-old female with history of depression and sleeve gastrectomy who presented to the emergency department after ingesting fifty 500 mg acetaminophen tablets (25 g) nine hours prior to presentation in a suicide attempt. She reported multiple episodes of non-bloody emesis that began one hour after the ingestion, without visible pill fragments in the emesis. The patient additionally reported left upper quadrant pain and fatigue. She denied other co-ingestions.

## Past Medical History

Chronic headaches, depressive episode 6 years ago with auditory hallucinations and suicidal ideation

## Past Surgical History

Laparoscopic sleeve gastrectomy

## Medications

None

## Allergies

None

## Social History

None

## Physical exam

The patient was oriented to person, place, and time. Patient's abdomen was distended with tenderness to palpation in the left upper quadrant without rebound tenderness. The patient's skin was dry without rashes, erythema, diaphoresis, or pallor. The patient's remaining physical exam was unremarkable.

## Vital signs

T: 98.7 F | HR: 74 | BP: 124/84 | RR: 18 | SpO2: 100% on RA

## Diagnostics

AST 91 / ALT 97 / Alk Phos 79 / T. Bili 0.7 / D. Bili 0.2 / Lipase 13
Ammonia 49
PT 17 / INR 1.3
VBG: pH 7.46 / PCO2 27 / PO2 34 / HCO3 19 / BE -3.2
Lactate 0.7
Serum Pregnancy Test Negative
Acetaminophen (Drawn approximately 10 hours after ingestion) 125 ug/mL
Salicylate < 3 µg/mL
HCV & HBV Ab negative

## Hospital Course

N-acetylcysteine (NAC) therapy was started given that the patient's serum acetaminophen concentration was 125 ug/mL, 10 hours post- ingestion, above the treatment threshold on the Rumack-Matthew nomogram. She was evaluated in the ED by the psychiatry consult team and was deemed appropriate for outpatient psychiatric follow up, and therefore, an involuntary psychiatric hold was not signed. The patient was then admitted to the Hospital Medicine service for ongoing NAC administration and trending of labs. The patient suffered intractable vomiting that was refractory to antiemetic therapy and developed right upper quadrant pain on hospital day (HD) 1. On HD2, her liver enzymes peaked with AST 3908, ALT 4235, and INR 2.0, at which time the patient was transferred to the MICU. There, the patient received vitamin K supplementation for her elevated INR in conjunction with continuous NAC infusion per the antidotal protocol until HD3, when her liver enzymes began to downtrend. Given improvement in her liver function assays and stable vital signs, the patient was subsequently transferred back to the medicine floor. Due to concerns for impending liver failure, she was evaluated by the transplant social worker, and consents were signed to begin pre-operative assessments for a liver transplant. On HD4, however, the patient refused further therapy and workup and signed out of the hospital against medical advice. Ultimately, the multidisciplinary liver transplant selection committee felt that the patient was a poor transplant candidate given her psychiatric disease and social impediments, subsequently removing her from consideration. The patient was thereafter lost to follow-up.

## Discussion

### Pathophysiology

N-acetyl-para-aminophenol (acetaminophen, APAP) is widely used in the United States for its analgesic and antipyretic properties, and is widely available over the counter in an oral formulation. The mechanism of action of acetaminophen remains unclear, but is postulated to inhibit COX pathways in the central nervous system inhibiting synthesis of prostaglandins.[1] The maximum recommended APAP dose in adults is 4 g/day and 50-75 mg/kg/day in children. In therapeutic doses, APAP is metabolized in the liver, where it is converted to inactive glucuronide and sulfate conjugates (90-95%), which are excreted in the urine.[2] The remaining fraction of APAP (5-10%) is metabolized via the cytochrome P-450 CYP2E1 pathway to a reactive metabolite called N-acetyl-p-benzoquinone imine (NAPQI). In supratherapeutic doses of APAP, the typical metabolism mechanism is overwhelmed and more and more APAP is shunted into the NAPQI pathway. NAPQI is highly reactive due to its free radical properties and causes oxidative damage to hepatocytes. In therapeutic doses of APAP, NAPQI is metabolized to an inactive metabolite (APAP-GSH) by binding the sulfhydryl group of glutathione present in hepatocytes. However, at toxic doses of APAP, the liver's glutathione reserves are rapidly depleted, causing NAPQI to accumulate in the perivenous hepatic cells where it induces DNA fragmentation, mitochondrial dysfunction, and hepatocyte necrosis. Hepatic damage begins when about 70% of glutathione is depleted. In a healthy, 70 kg patient with normal liver function, this typically does not occur until exposure exceeds 16 g of APAP.[3] However, for patients with existing liver dysfunction, malnutrition, concurrent use of P450 inducing agents (e.g., rifampin, phenobarbital, phenytoin, or carbamazepine) or other hepatotoxic medications (INH, TMP-SMX, zidovudine), the

toxic threshold may be significantly reduced.

## Testing

In 1975, Drs. Barry H. Rumack and Matthew created a nomogram to outline treatment thresholds for serum APAP levels measured between four and twenty-four hours after ingestion, as this is when serum levels typically reach their peak.[4] At the request of the FDA in 1981, a second line was added to include a 25% margin of error to account for variability in serum APAP measurements or uncertainty about timing of ingestion.[5] This second line has now been adopted as the standard for treatment threshold on the nomogram, which begins at serum APAP concentrations of greater than 150 ug/mL at 4 hours post-ingestion. Concentrations below this line indicate low risk for hepatotoxicity. Some variations of the nomogram contain a "massive intoxication" threshold, which resembles a treatment line that begins at 300 ug/mL at 4 hours and decreases at the same slope of the standard line to 24 hours. This designation carries different treatment goals, which will be discussed in the next section. Serum acetaminophen levels obtained prior to the four-hour threshold are typically viewed as unreliable as the drug may still be in transit within the stomach and the assay may not reflect a peak measurement. Importantly, clinicians must pay attention to the units of their institution's serum APAP assay and verify that they correlate with the units on the Rumack-Matthew Nomogram as to avoid over- or under-estimating the treatment threshold.

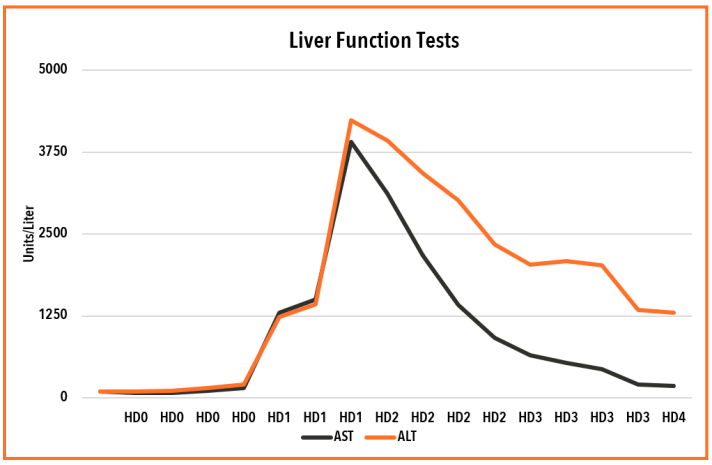


*Figure 5: Liver Function Tests During Hospital Stay*

Despite its widespread use, the Rumack-Matthew Nomogram does present some limitations. The nomogram cannot be used for multiple APAP ingestions and is only validated for one acute ingestion presenting within twenty-four hours. As previously mentioned, concurrent use of medications that induce P450 metabolism, or use of hepatotoxic medications may allow clinically significant toxicity to occur at levels below the treatment threshold. Lastly, altered pharmacokinetics including extended-release APAP formulations or alterations to gastric emptying (stemming from concomitant opioid use or gastroparesis, for example) should preclude the use of the nomogram due to unpredictable variations in the rate of APAP absorption.[6] There are data to suggest that sleeve gastrectomy and other bariatric surgeries may increase gastrointestinal motility and thus potentially increase APAP bioavailability. The clinical relevance of this finding in the setting of toxic ingestion is still uncertain.[7-9] It has been demonstrated that extended release formulations of APAP exhibit similar rates of elimination as the immediate release form after four-hours. However, there are case reports to suggest that delayed absorption and peak concentrations after four hours can also occur.[10-12] IF there is concern for extended release formulation serial testing should be considered.[13]

## Treatment

Treatment of acute APAP overdoses should be prompted by the serum APAP level and never by estimated ingestion quantity, as estimations have been demonstrated to correlate poorly with serum levels.[14-15] For patients with known single ingestion and reliable ingestion time less than 24-hours, the Rumack-Matthew Nomogram should be used to determine the need for treatment. For patients with unknown ingestion time, history of alcoholism or malnutrition, use of synergistic drugs (P450 inducers or hepatotoxins), or delayed gastric emptying, some treatment approaches advocate for empiric NAC treatment - given the unpredictable alterations to APAP absorption and metabolism - while trending of serum APAP concentrations and ALT. Treatment with NAC beyond 24-hours is controversial, but some argue that there is a role for NAC in patients with ALT > 50 with delayed presentation.

NAC serves as the primary treatment for acute APAP overdose, and multiple dosage protocols exist, including a 72-hour oral protocol and the 20-hour IV protocol. The oral regimen includes a loading dose of 140 mg/kg, and is followed by 70 mg/kg every four hours for a total of 17 doses. This can be difficult to endure for patients, as it may cause nausea, has a foul odor and is often not well tolerated by patients, and therefore the IV regimen is more commonly employed than the PO regimen. The 20-hour IV regimen includes an initial loading dose of 150 mg/kg over one hour, followed by an infusion of 50 mg/kg over four hours, and finally a dose of 100 mg/kg over 16 hours. Treatment should be continued at 6.25 mg/kg per hour IV until the serum APAP concentration is undetectable, the ALT is clearly decreasing or within the normal range, and the INR < 2.

For patients with massive intoxication (four-hour level above 300 ug/mL or suspected ingestions of greater than 500 mg/kg), there is data to support increased dosing of NAC. These dosing regimens likely will not change ED management, as the first two doses are unchanged, corresponding to the first five hours of treatment. The third dose is increased to 12.5 mg/kg/hr to 25 mg/kg/hr depending on the four-hour APAP concentration.[16]

Some patients may experience an anaphylactoid reaction with administration of NAC that can manifest as flushing, urticaria, angioedema, and potentially respiratory distress and hypotension. These reactions occur within the first six hours of administration, most often within the first two hours.[17] NAC should only be held in the setting of respiratory distress, hypotension or angioedema, and antihistamines and steroids can be considered for symptomatic treatment.

Activated charcoal should be considered as adjunctive therapy for patients that present within 1-3 hours of ingestion and are able to protect their airway. Activated charcoal provides the greatest benefit to those presenting after massive APAP ingestions18. Dialysis is able to remove APAP and NAPQI from circulation. The Extra

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## Jaw Dislocation

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method was not evaluated in this study. The extraoral method was found to be most uncomfortable for both patient and provider and was the most technically difficult. The wrist pivot method was deemed the easiest. Risk of trauma and infection were still noted with intraoral placement of the thumb or fingers with the conventional and wrist pivot methods.

Once reduction is achieved, the patient should be reassessed for pain or malocclusion. Patients should undergo repeat x-rays to assess for successful jaw positioning and to reassess for iatrogenic fractures. Patients should be instructed to avoid extreme mouth opening, such as yawning, for several weeks to prevent recurrence of dislocation. Non-steroidal anti-inflammatory drugs are generally recommended for pain control, and patients are encouraged to eat a soft diet. Cool or warm compresses applied to the temporomandibular joint may assist further with pain control.[3] OMFS follow up should be scheduled within the week for further recommendations and management, as these patients are prone to recurrence of jaw dislocation.

### Summary

An understanding of the anatomy and common mechanisms of jaw dislocations can assist the emergency provider in diagnosing and treating this debilitating condition in the emergency department. Possessing an arsenal of reduction techniques can help the provider achieve TMJ reduction when initial attempts fail. The syringe technique is typically a reasonable starting point, as it avoids intraoral placement of the provider's hands and is generally well-tolerated by patients. The wrist pivot method may be chosen next, as it is intuitive for providers and has been rated less painful by patients. Precautions should be taken to prevent bite injury with this technique. The conventional intraoral or extraoral counterpressure methods may be attempted if the aforementioned methods fail. Becoming familiar and comfortable with these techniques will help the emergency physician more successfully address this complaint when encountered in the emergency department.

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## Tylenol Overdose

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corporeal Treatment in Poisoning (EXTRIP) suggests that dialysis is recommended if the patient has altered mental status, metabolic acidosis, elevated lactate, and serum APAP concentration of greater than 900 mg/dL, with or without NAC administration.[18] For patients on hemodialysis, the dose of NAC needs to be increased; there are no prospectively validated algorithms and alternative dosing should be administered under the direction of local poison control experts. Fomepizole, a cytochrome P450 inhibitor, can be considered a potential adjective therapy given its ability to shunt APAP metabolism away from NAPQI and towards more benign metabolites; however, larger studies are required to validate its efficacy.[19]

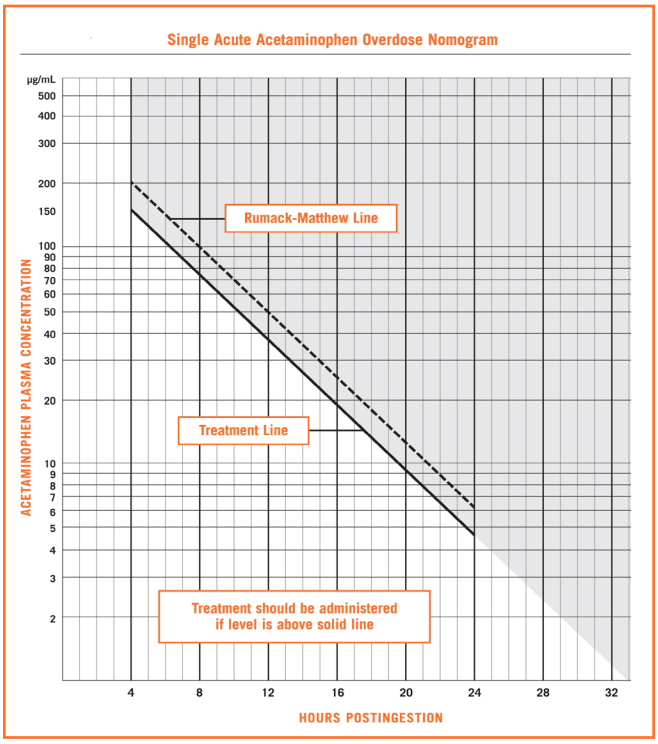


*Figure 6: Rumack-Matthew Nomogram*

### Prognosis

APAP toxicity is the leading cause for fulminant hepatic failure in the United States, accounting for approximately 39% of all cases.[20]Acute APAP overdose carries high morbidity and mortality if it goes undiagnosed or is not treated in a timely fashion. In a 1995 review of 295 cases of liver transplant for acute liver failure, APAP toxicity was the leading cause for liver transplantation, representing 20% of that cohort.[21] Fortunately, 65% of patients with acute APAP toxicity survive the event without need for transplantation if appropriate treatment is provided early.[22]

### Summary

Acute APAP overdose is a disease with high morbidity and mortality. The Rumack-Matthew Nomogram should be utilized to guide treatment decisions, though providers should be aware of its limitations, particularly given the potential for inaccurate timing of ingestion, multiple ingestions, existing liver dysfunction, GI dysmotility, or concurrent use of hepatotoxic or P450 inducing medications. NAC antidotal therapy is safe and effective and should be employed early in patients with elevated serum levels or evidence of organ dysfunction. Providers should familiarize themselves with their institutional treatment protocols. Patients may experience anaphylactoid reaction to NAC; as this is not a true allergic response, patients may be treated symptomatically with antihistamines and continuation of NAC therapy if the patient is stable. Adjunctive therapies, including hemodialysis, activated charcoal, and

fomepizole, may be employed at the discretion of toxicologists or poison control experts in the context of massive ingestions.

Given the low resistance of nerve tissue, the neurologic system is vulnerable to high-voltage electrical injuries.[13] Patients are at risk for seizures, spinal cord injury, peripheral paresthesias and neuropathies following electrical injury. These symptoms can be delayed, manifesting days to months after the electrocution injury.[1]

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## Electrocution Injury

*Continued from page 5*

### Emergency Department Management

Emergency care priorities remain standardized with an assessment of the patient's airway, breathing, and circulation. In patients who progress to cardiac and/or respiratory arrest, standard ACLS protocol should be followed, with a potential role for dual-sequential defibrillation and esmolol in patients with ventricular arrhythmias.[14-17] All patients suffering an electrical injury require a thorough examination with a high suspicion for atypical injury patterns. All patients should be placed on cardiac telemetry and undergo electrocardiogram (ECG), regardless of symptoms. In the setting of low-voltage electrocutions, if the patient has minimal to no cutaneous injuries and does not have arrhythmias or ischemic changes noted on ECG, they can be discharged after initial evaluation. Continuous cardiac monitoring is not required as the risk of developing a lethal arrhythmia after an initially normal ECG is low.[18]

If significant cutaneous damage or electrocardiographic abnormalities are present, the patient needs evaluation by a burn specialist and telemetry monitoring. Patients with the injuries noted in Table 1 and those who suffer an electrical injury of >1000 V AC require transfer to a burns center according to the International Society of Burns Injuries 2018 Guidelines.[19-20]

| Indications for Transfer to Burn Center |
|---|
| Partial thickness burns > 10% total body surface area (TBSA) |
| Burns involving the face, hands, feet, or genitalia |
| Third degree burn |
| Electrical/lightning burns |
| Chemical burns |
| Inhalation burns |

*Table 3: Indications for transfer to a burn center*

Patients with high voltage injuries should have laboratory testing to assess for organ injury, including a renal panel, creatinine kinase, troponin, urinalysis, and urine myoglobin. The emergency provider should also evaluate for signs of compartment syndrome and rhabdomyolysis. Patients with full-thickness circumferential burns or burns extensively involving the thorax may require escharotomy. Patients with myoglobinuria, burns affecting >20% TBSA, and full-thickness burns >12% TBSA are at increased risk of requiring fasciotomy in the first 24 hours.[21]

The approach to fluid resuscitation for patients with electrical burns does not differ from patients who have suffered chemical or thermal burns. The Parkland formula, based on TBSA affected, is an appropriate starting point. However, electrical burns may disproportionately affect the deep tissues. As such, it is prudent to use urine output to guide fluid resuscitation, with a urine output goal of 1 – 2cc/kg/hr.

## Disposition

In general, asymptomatic patients who have suffered a low-voltage shock can safely be discharged if there are no concerning findings on history, physical examination, and ECG. Patients who suffer a high-voltage shock should be admitted for a period of observation, even if all components of the ED evaluation are reassuring.[1]

## Conclusion

The evaluation and management of patients who have suffered electrocution is challenging, as presentations range from asymptomatic to refractory cardiac arrest. Important historical features include type of current (AC or DC), voltage, and duration of exposure. Electrocuted patients are especially susceptible to cardiac arrhythmias, neurologic injury, rhabdomyolysis, and compartment syndrome. Even in the well-appearing patient, a thorough evaluation and high index of suspicion for injury is paramount, as electrocuted patients often have atypical injury patterns.

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vidual ingestions makes the clinical course difficult to predict and the risk of co-ingestions presents one of many confounders. Common co-ingestions include ethanol, other antidepressants (e.g. SSRI, SNRI), benzodiazepines, acetaminophen and salicylates.[7] Given these difficulties, the ECG has become an important tool for assessing the severity of the ingestion. While an effort should be made to assess a patient's baseline ECG, a newly prolonged QRS duration of greater than 100 ms is associated with increased risk of seizure activity and a QRS duration greater than 160 ms is associated with increased risk of ventricular arrythmia.[13] Attention should also be given to the morphology of the QRS complex in aVR as the presence of an R wave greater than 3 mm or a R/S ratio greater than 0.7 suggests an increased risk of seizure and arrhythmia.

### Treatment

Early studies demonstrated benefit of increasing the serum concentration of sodium to overcome the sodium channel blockade as well as induced serum alkalization to reduce the unbound fraction of biologically active medication.[18-20] One can achieve both goals via administration of sodium bicarbonate, which has become the cornerstone of treatment for TCA overdose. Realtime feedback on the effectiveness of this intervention can be observed in narrowing of the QRS complex on ECG, as was observed in this case (Figures 1, 2). This is commonly achieved by administering a bolus of 1-2 milliequivalents per kilogram (mEq/kg) of sodium bicarbonate followed by initiating a bicarbonate infusion, with a goal serum pH of 7.45 – 7.55. A bicarbonate solution for infusion may be prepared by adding 3 amps of sodium bicarbonate to 1 liter of D5W. Commonly, infusions are started at 2 -3 mL per kilogram per hour (mL/kg/hr). If sodium bicarbonate is unavailable, administration of hypertonic saline can be used as an alternative agent to mitigate the effects of sodium channel blockade.[7,8]

Given the antimuscarinic activity of TCAs and associated slowing of gastrointestinal motility, additional intervention may be directed at limiting further drug absorption via gastric lavage or administration of activated charcoal (1 gram/kilogram orally, max 50 grams) if these can be completed within one hour of the ingestion.[7,8]

Hypotension emerges as a result of both decreased cardiac performance and peripheral vasodilatation via alpha blockade. Vasopressors are often necessary in significant overdoses. Epinephrine and norepinephrine are favored for their combined cardiac and vasoconstrictive properties. Dopamine is less favorable as its use assumes adequate endogenous stores of norepinephrine, which may already be depleted in the severely poisoned patient.[7] In one case report, an infusion of angiotensin II was useful in treating refractory shock in a doxepin overdose.[21] Vasopressin and methylene blue may have a role in refractory vasoplegic shock, given mechanisms of action that do not rely on adrenergic receptor agonism, but evidence for their use in TCA overdose is lacking.

Patients with TCA overdose are at very high risk for cardiac arrhythmia. Prevention of arrhythmias is best achieved by treating any proarrhythmic conditions including hypoxia, acidemia and hypokalemia.[22] Should the patient develop a persistent tachyarrhythmia such as ventricular tachycardia, most antiarrhythmics are contraindicated as they will promote further compromise to cardiac conduction. Lidocaine, however, presents an interesting intervention. While also exerting its effect via sodium channel blockade, lidocaine appears to competitively inhibit TCA binding and dissociates quickly resulting in a net normalization of phase 0 depolarization.[23]

Seizures commonly occur as a result of the neuroactive properties of TCA medications, including norepinephrine reuptake inhibition, GABA antagonism and anticholinergic activity. Benzodiazepines are recommended as a first-line treatment for seizures secondary to TCA overdose. There are reports for the use of phenytoin and levetiracetam as additional measures for refractory seizures. Most patients with severe toxicity will require intubation for airway protection and ventilatory support and therefore propofol has been utilized for the maintenance of sedation and has the added benefit of increasing GABAergic tone for resolution of seizure activity[7,24,31] Based upon these findings, one could reasonably pursue a plan for initial management of seizures with benzodiazepine followed by propofol added in the larger context of comprehensive resuscitation including definitive airway control.

Several alternative treatments for TCA toxicity have been explored but have not proven sufficiently beneficial to alter standard practice. The use of extracorporeal filtration including hemoperfusion, hemodialysis and forced diuresis has not proven effective as TCA medication is highly protein bound.[8] The development of antibody-based therapies proved unsuccessful primarily because the quantity of required antibody would be clinically untenable.[7] There is anecdotal evidence for the use of lipid emulsion therapy, however, when evaluated experimentally and in review, the outcomes have been less favorable.[24-28]

## Summary

TCA toxicity has high mortality, and patient prognosis is dependent upon early identification and aggressive treatment. The foundations for treatment include assessment for pathognomonic ECG changes suggesting severity of toxicity followed by administration of sodium bicarbonate to achieve a narrowing of the QRS and maintenance of serum alkalemia. Gastric lavage or charcoal may be beneficial if given early in the ingestion and the patient's airway is protected. To that end supportive measures are likely to include intubation for airway protection and ventilatory strategies to promote a mild alkalosis as well as interventions to address hypotension, seizure activity and other metabolic derangements such as hypokalemia. Major complications from overdose will be apparent at 6 hours post ingestion[8] and will invariably include evidence of cardiac conduction defect, arrhythmia, altered mental status and seizures followed by cardiovascular collapse.[30] Additional complications include respiratory arrest, pulmonary edema, aspiration pneumonia, anoxic encephalopathy, hyperthermia and acute renal failure.[8] Should resolution of cardiac toxicity be achieved as well as other high acuity complications, further observation with telemetry is recommended for a minimum of 24 hours.[7]

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## Subclavian Steal Syndrome

*Continued from page 3*

nomenon when there is subclavian artery stenosis or an occlusion proximal to the vertebral artery, leading to distal hypoperfusion. To compensate for this hypoperfusion, blood shunts from the contralateral vertebral artery, proceeding in a retrograde fashion through the ipsilateral vertebral artery to supply the vasculature distal to the occlusion. However, the distal subclavian artery is now 'stealing' blood from vertebral artery that would otherwise be solely perfusing the head and neck. This adaptation is oftentimes asymptomatic; however, if the stenosis or occlusion is severe enough, it can lead to positional and demand hypoperfusion of both the ipsilateral upper extremity and the brain, resulting in a cluster of symptoms collectively known as SSS.

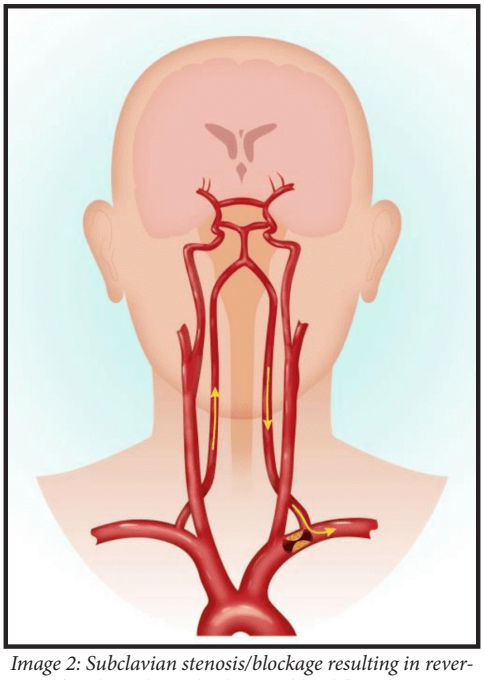

*Image 2: Subclavian stenosis/blockage resulting in reversal of ipsilateral vertebral artery blood flow direction[18]*

Subclavian artery stenosis and occlusion affects males more commonly than females. Although atherosclerotic thrombus formation is by far the most common etiology, it can also be attributed to an embolus, arteritis, anatomic abnormalities in the cervical ribs, or repairs from congenital cardiac anomalies.[1] SSS is three times more likely to occur on the left side compared to the right. This difference is thought to be attributed to the relatively higher shear force experienced by the left subclavian artery and the resultant increase in left-sided atherosclerotic disease.[1] Despite the high prevalence of atherosclerotic disease in the United States (US), SSS remains a rare entity, with an incidence reported between 0.6 and 6.4% of all adults.[2] Less than 3% of all patients with peripheral artery disease (PAD), and less than 10% of patients with confirmed subclavian stenosis will manifest syndromic symptoms.[1,3] This rarity may be attributed to the wide vascular network of collateral alternatives that exists in the upper limb, as well as the low prevalence of PAD in the upper extremities.

### Clinical Presentation

SSS commonly presents with unilateral arm claudication defined by pain, fatigue, or sensory change with exertion. Symptoms may also be provoked with specific movements, such as rotation of the head, which compresses the vertebral artery, and movement of the affected arm. Although less common, when cerebral perfusion is reduced, symptoms include those due to vertebrobasilar insufficiency, including vertigo, visual changes, ataxia, numbness, and syncope.[4] Presence of these symptoms usually indicates coexisting cerebrovascular disease,[5] and when these symptoms are present upon initial evaluation, the patient has a higher risk of developing a posterior circulation stroke in conjunction with their SSS.[6] The initial differential should be kept broad to include SSS, thoracic outlet syndrome, stroke, peripheral neuropathy, peripheral vascular disease, or musculoskeletal injury.

### Diagnostic Evaluation

Systolic blood pressure discrepancy and reproduction of symptoms with upper extremity activity are highly suggestive of SSS. Symptoms are more common in those with a pressure differential of greater than 20-40 mmHg between the two arms, and these patients are more likely to require intervention.[1] Exercise of the affected arm may actually result in disappearance of the radial pulse or reproduction of neurologic symptoms.[7]

Imaging is required to make the formal diagnosis and exclude other etiologies. Duplex ultrasound is a non-invasive imaging modality to evaluate for significant subclavian pathology and retrograde flow through the ipsilateral vertebral artery.[8] While one study posited that a peak systolic velocity (PSV) greater than 240cm/second was predictive of greater than 70% arterial stenosis, a consensus on diagnostic criteria in the upper extremity does not yet exist.[9] In the lower extremities, stenosis is estimated by the ratios between the PSV within the occlusion to just distal to the occlusion. Higher grades of subclavian stenosis are more frequently associated with both continuous and intermittent vertebral artery flow reversal on ultrasound. Additional maneuvers, such as a reactive hyperemia test, may be utilized to detect occult or intermittent subclavian steal.[10] In a reactive hyperemia test, a blood pressure cuff is applied to the affected extremity, inflated above systolic blood pressure, and left in place for three to four minutes. This functions as a temporary tourniquet and results

in distal vasodilation of the extremity. When the blood pressure cuff is released, distal blood flow dramatically increases to fill the dilated arteries and may precipitate occult retrograde flow detectable on ultrasound, resulting in symptoms of SSS.[10] Additionally, transcranial doppler can be used to evaluate reversal of flow within the basilar artery, which may be predictive of development of SSS.[11]

The diagnostic utility of duplex ultrasound can be limited by its anatomical restrictions. Ultrasound may miss stenosis in the segment of the subclavian artery obscured by the clavicle. Additionally, retrograde vertebral artery flow is not always caused by subclavian steal if there is stenosis in the vertebral artery origin itself, and to differentiate between the two on ultrasound is difficult. CTA and MRA are considered confirmatory in those with abnormal ultrasound, or as initial and confirmatory diagnostic test when ultrasound unavailable.[1]

These modalities provide further information on the vasculature of the head and neck, as well as surrounding structures which may be involved in the pathologic process, such as in the case of anatomically anomalous ribs or compressive mass effect.

### Treatment

Severity of symptoms, rather than the extent of disease seen on imaging, usually dictates the degree of intervention required.[12] Patients with asymptomatic subclavian stenosis are frequently managed with risk factor modification, as it is indicative of widespread atherosclerotic disease and is independently associated with increases in mortality (OR 1.4).[13] Such modifications are similar to that of general atherosclerotic disease and include treatment of hypertension and dyslipidemia, glucose control, and smoking cessation.

Patients with presumed atherosclerotic origin are typically placed on lifelong antiplatelet medication to prevent further plaque accumulation, whereas anticoagulation is preferred for those with a presumed embolic etiology.[15] Similar medical management strategies are applied to those with moderate to severe symptoms who are poor surgical candidates. For patients who are surgical candidates and who have significant stenosis with symptomatic steal, endovascular intervention versus surgical bypass are the most common therapeutic procedures. As angioplasty and stenting have become more widely available, they are gradually becoming preferred over surgical bypass due to their effectiveness and relatively minimally invasive nature.[16,17] Symptomatic patients presenting to the emergency department require admission for urgent management while asymptomatic patients with incidental finds require timely outpatient follow-up.

## Summary

Subclavian Steal Syndrome is an uncommon disease process resulting from the proximal occlusion of the subclavian artery. Occlusion of the proximal subclavian artery leads to blood flow reversal in the ipsilateral vertebral artery shunting blood into the affected limb. This is most commonly asymptomatic but can result in symptoms of arm claudication and vertebrobasilar insufficiency. Clinical suspicion should be high when there is greater than 20-40 mmHg in difference in right and left upper extremity pressures and symptoms are reproducible on exam. Subclavian stenosis and retrograde vertebral artery flow can be observed using duplex ultrasound and confirmed using CTA or MRA. Once confirmed, vascular surgery should be contacted for further management and surgical evaluation.

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