

The Sharp End

Special Edition



May Special Edition

Deactivating the
Activated Charcoal
Reflex: Thinking
before charcoaling
Chip

Rational O2 Use: A
breath of fresh air.
Andy

Turnin' off the tap:
Less is more in fluid
resuscitation
Joe Alcock

These discussions are the
opinions and ramblings of
the authors, designed to
stimulate thought and
discussion.

This is in no way and
should not be considered a
practice guideline of any
type

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Welcome to the May Special Edition vol. 9

While we all found our way into medicine for various reasons, likely, the most common is because we want to help. We want to help our patients in their time of need. We want to cure them, comfort them, fix them... we want to do something. And when we can't do something, or worse, we are told not to, it can be frustrating. So ingrained is this desire to do something, that we can lose sight of the words of Hippocrates and of the Fat Man: FIRST, DO NO HARM and the later, the 13th rule of the House of God - THE DELIVERY OF GOOD MEDICAL CARE IS TO DO AS MUCH NOTHING AS POSSIBLE. In this special edition, we are going to look at 3 common therapies that have recently been in the literature with the thought that less just might be more. ..

Activated Charcoal – Friend or Foe

Having spent the last 5 years as a medical toxicologist, I have seen the afore mentioned frustration play out daily in the care of the poisoned patient. The typical scenario is one of intentional OD, usually with the most readily pills accessible: anti-depressants, analgesia or something that is found in the medicine cabinet. Occasionally these can be highly toxic; most of the time they are not. Serious clinical effects occur in less than 5% of acutely poisoned pt ([Greene et al.](#)).¹ That however does not prevent many in emergency medicine from jumping into “ must do something” mode. Antidotes in toxicology are few, so for most overdoses, we are left with good supportive care...and activated charcoal, as our options.

Activated charcoal (AC), having been around in the medical literature since the 1700's, remains a highly debated topic in emergency medicine and toxicology. AC was once given universally in response to any overdose that presented to the ED, however, that approach has been challenged. [The Position statement for single-dose activated charcoal](#)² of both the *American Academy of Clinical Toxicology* and the *European Association of Poisons Centres and Clinical Toxicologists* states: **AC should not be routinely used.** Its use has never been shown to change clinical outcomes.

There are multiple RCTs as well as multiple case reports used to supports both its use and its potential dangers . The review of these individually is beyond the scope, and word count, of this monologue.

There have been two recent reviews that summarize published data and are both worth a read. [Indications for single-dose activated charcoal administration in acute overdose](#) by G Isbister in 2011 *Curr Opin Crit Care*,³ and [Activated Charcoal for Acute Poisoning: One Toxicologist's Journey](#) by K. Olson in the 2010 *J Med Toxicol*.⁴ Both review multiple expert opinions of the go/no go for AC.

Many of my registrars and colleagues believe I am anti-AC. I am not. I am anti- “AC as a knee jerk reaction to every OD that comes in the door”. Each specific pt and the specific xenobotic they ingested must be considered. A risk assessment must be done. It is the responsibility of the clinician prescribing the AC to understand the basic pharmacology and toxic potential of the drug they are giving it for. A good resource is the [Toxicology Handbook](#) by Murray et al.⁵

I believe you will find in 9 out of 10 pts, the risk of giving AC will outweigh any perceived benefit. (i.e. it should not be given). Giving AC for a zolpidone OD (low toxicity) only to have your otherwise stable pt have a slight drop in LOC and aspirate AC is not only wrong, but doing harm.... NOT giving AC to a colchicine OD (in which > 0.8 mg/kg approaches 100% mortality) is wrong as well.

The goal of this monologue is not to convince you to not use AC, but rather challenge you to not reflexively give AC to everyone of your ODs. A good start is the table below. It is adapted from *Olsen's* paper When you consider the patient, the drug, the potential course and other options, if you find yourself in the red, with even just one, I would NOT recommend AC.

- Chip

AC risk assessment	AC favorable/consider	AC to risky or not needed
Patient	- Presents with in 1 hr of OD - Awake, alert and cooperative - Airway protected*	- Altered♦ or refusing AC
Substance	- Known to be absorbed by AC - High toxicity (i.e colchicine or CCB) - Sustained release with significant potential toxicity	- Low toxicity (ie. benzos, SSRIs, sleep aids, most antipsychotics) - Poorly absorbed
Other		- Antidote available (i.e. paracetamol)

*Endotracheal intubation does not guarantee a 100% protected airway.

♦If not altered upon presentation, potential to become altered must be considered

Adapted from Olson⁴

- Greene SL et al. Acute poisoning: understanding 90% of cases in a nutshell. [Postgrad Med J. 2005 Apr;81\(954\):204-16](#)
- Chyka PA, et al Position paper: Single-dose activated charcoal. [Clin Toxicol \(Phila\). 2005;43\(2\):61-87](#)
- Isbister GK, Kumar VV. Indications for single-dose activated charcoal administration in acute overdose. [Curr Opin Crit Care. 2011 Aug;17\(4\):351-7](#)
- Olson KR. Activated charcoal for acute poisoning: one toxicologist's journey [Med Toxicol. 2010 Jun;6\(2\):190-8](#)
- [Toxicology Handbook 2nd Edition](#) Lindsay Murry

Fluid resuscitation in trauma: Is less more?

After more than a decade in practice in emergency medicine, I often advise trainees that in addition to understanding which medications or interventions are needed for a given patient, they should also consider why they should *not* perform these treatments. Although it is hard to weigh risks and benefits in a life threatening situation, we owe it to our patients to understand the risks involved with emergency procedures. Moreover we have an ethical obligation to withhold treatment when the risks outweigh the benefits.

Unlike a chest tube or arterial puncture, procedures in which the risks are easily understood, some interventions that we routinely do in the emergency department and prehospital setting are so deeply ingrained that we scarcely think about them. One example of this is the reflexive habit of administering several liters of crystalloid intravenously to trauma patients. We have all been taught to fluid resuscitate these patients. But is our zeal to restore a patient to a normal blood pressure and heart rate too much of a good thing?

Most of us would recognize the danger of giving fluids to a patient in congestive heart failure, but what about trauma? If we suspect a patient might have a hemorrhage, many physicians unquestioningly order fluids, often in large amounts, usually delivered by large bore IV catheters.

This community standard of care carries over to the prehospital environment as well. Paramedics generally provide intravenous fluids to trauma patients. Nobody is surprised when a patient arrives in the ED with a half-empty bag of normal saline, regardless of whether or not a patient is bleeding. Nobody blinks an eye when the nurse spikes another bag of normal saline and starts another bolus.

Despite the widespread assumption that saline and other crystalloids are generally good for bleeding patients, a variety of studies suggest that the practice of restoring fluid volume to patients with blood loss is not benign.

In 1994, Bickell *et al* published a paper titled [Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries](#)¹ that was a well-designed, large prospective study of 600 patients with penetrating trauma that raised doubts about the use of pre-hospital fluids. It showed that penetrating trauma patients who received crystalloid were more likely to die than those who received none.

VARIABLE	IMMEDIATE RESUSCITATION	DELAYED RESUSCITATION	P VALUE
Survival to discharge — no. of patients/total patients (%)	193/309 (62)*	203/289 (70)†	0.04
Estimated intraoperative blood loss — ml‡	3127±4937	2555±3546	0.11
Length of hospital stay — days§	14±24	11±19	0.006
Length of ICU stay — days§	8±16	7±11	0.30

*95 percent confidence interval, 57 to 68 percent.

†95 percent confidence interval, 65 to 75 percent.

‡The estimated intraoperative blood loss was calculated for patients who survived the operation: 268 in the immediate-resuscitation group and 260 in the delayed-resuscitation group.

§The lengths of stays in the hospital and intensive care unit (ICU) were calculated for patients who survived the operation: 227 in the immediate-resuscitation group and 238 in the delayed-resuscitation group.

From Bickell¹

Another subsequent randomized trial titled [A randomised controlled trial of prehospital intravenous fluid replacement therapy in serious trauma](#)² published in 2000 *Health Technol Assess.* has not led to widespread changes in treatment.

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Now nearly twenty years after these studies, recommendations to incoming emergency medicine trainees has hardly changed: give two liters of crystalloid for low blood pressure, and then start a transfusion of blood products.

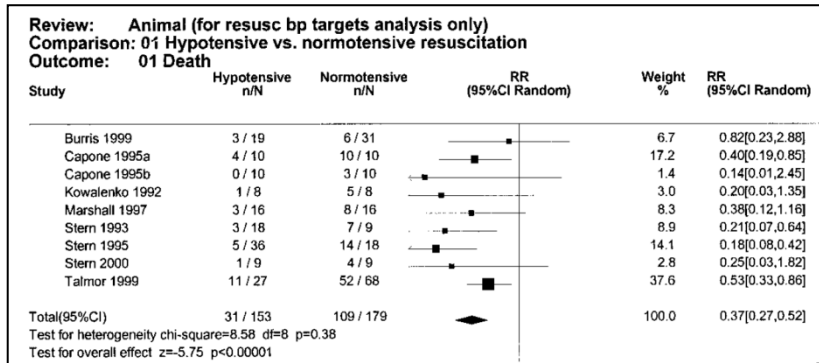
Paramedics still infuse intravenous saline when trauma patients have low blood pressure. While intravenous fluids do reliably increase blood pressure, a recent retrospective study published in Feb 2011 in *Ann Surg* titled [Prehospital intravenous fluid administration is associated with higher mortality in trauma patients: A national trauma data bank analysis](#)³ found that trauma patients who received pre-hospital fluids were 11% more likely to die than those who did not receive them. This recent study was large in scope (examining records of 776,234 patients) and is likely to change the way we treat low blood pressure in trauma. So what about the question: does moderately lowered blood pressure have a survival benefit after injury? It turns out that this is not a new idea.

In 1918 in *JAMA* in an article titled **The preventive treatment of wound shock**, Cannon wrote:

“Injection of fluid that will increase blood pressure has dangers itself. Hemorrhage in a case of shock may not have occurred to a marked degree because the blood pressure has been too low and the flow too scant to overcome the obstacle offered by the clot. If the pressure is raised before the surgeon is ready to check any bleeding that may take place, blood that is sorely needed may be lost.”

In a study of aortic injury in pigs, published in a 2003 *J Trauma* article titled [Blood pressure at which rebleeding occurs after Resuscitation in swine with aortic injury](#)³ investigators found that increasing the systolic blood pressure to normal destabilizes clots and causes re-bleeding. A review published in Feb 2011 *Am Surg* titled [Damage control resuscitation: From emergency department to the operating room](#)⁴ reports on how some trauma centers have lowered death rates by following a protocol of “permissive hypotension”, minimizing the use of fluids in trauma patients with low blood pressure in the emergency department.

Another review in 2003 *J Trauma* titled [Fluid resuscitation strategies: A systematic review of animal trials](#)⁵ reviews experiments using animal models supports this approach. In animals, fluid resuscitation was helpful only during near-exsanguination. In less severe hemorrhage, fluid resuscitation increased mortality. In the 9 available animal studies of permissive hypotension, this “hypotensive resuscitation” strategy improved survival in animal studies.



From Mapstone⁵

The most recent paper of hypotensive resuscitation was published in 2011 *J trauma* titled [Hypotensive resuscitation strategy reduces Transfusion requirements and severe postoperative coagulopathy in trauma patients with hemorrhagic shock: Preliminary results of a randomized controlled trial](#).⁸ was a trial of hypotensive resuscitation in trauma patients. It confirmed that a lower mean arterial pressure (50 mm Hg, instead of 65 mm Hg) decreased intraoperative and postoperative mortality, though the survival benefit at discharge fell short of statistical significance.

TABLE 9. Timing of Deaths

	MAP = 50 mm Hg (n = 44)	MAP = 65 mm Hg (n = 46)	p
Died in OR	5	2	0.26
Died within 24 h of ICU admission	1	8	0.03
Total deaths <24 h	6	10	0.32
Died 1-10 d after ICU admission	2	2	1.00
Died >10 d after ICU admission	2	1	1.00
Total deaths >24 h	4	3	1.00
Overall deaths at 30 d	10	13	0.55

From Morrison⁸

One benefit to hypotensive resuscitation is the decreased incidence of coagulopathy from dilution of clotting factors. Dilution of clotting factors is one of the most frequently cited potential dangers of crystalloid overtreatment, particularly in the military medicine literature (see 2003 *J Trauma* titled [Searching for the optimal resuscitation method: Recommendations for the initial fluid resuscitation of combat casualties](#).⁹ David Lounsbury, author of the Army textbook, [War Surgery in Afghanistan and Iraq](#) was recently quoted in the *New York Times* saying “Traditional hemorrhage treatment — giving intravenous saline solution to restore blood pressure — actually killed patients...because it diluted clotting factors.”¹⁰

In addition to dilution of clotting factors and destabilization of clots by restoring blood pressure, crystalloid boluses can exacerbate systemic inflammation. Rhee and colleagues have shown, using animal models, that provision of crystalloid or artificial colloids causes marked up regulation of pro-inflammatory cytokines and cellular injury markers. Because hemorrhage itself induces a systemic inflammatory state that contributes to shock syndrome, exacerbating inflammation with normal saline is probably a bad idea.

As reported in the 2010 *Lancet* article titled [Critical care: Advances and future perspectives](#),¹¹ it is notable that many of the advances of the last two decades have involved doing less, not more, for critically ill patients in the emergency department and intensive care unit. For example, physicians no longer hyperventilate patients after head trauma. Pulmonary artery catheterization are rarely used in the intensive care unit. Tight glycemic control and selective gut decontamination for the critically ill are now thought to do more harm than good. Perhaps aggressive crystalloid fluid resuscitation in the prehospital setting and emergency department will be the next to go.

Despite the above evidence on permissive hypotension for penetrating trauma, there is less consensus on fluid use in blunt trauma. There is also a strong consensus that **hypotension in brain injury is bad**.

For those intersted in more on this topic check out one of the most underutilized resources in EM: [FreeEmergencyMedicineTalks.net](#). Dr. Williams discusses the pros and cons of permissive hypotension at the [MEMC-VI](#) in 2011. Also, the superstud at [Life in the Fast Lane](#) just had a brief review of haemorrhagic resuscitation this March that is worth a look.

-Andy

A 2007 study published in *Am Surg* titled [Reanalysis of prehospital intravenous fluid administration in patients with penetrating truncal injury and field hypotension](#)¹² showed that the recommendations of Bickell and colleagues are not followed in a large urban center in the United States. The reluctance of EMTs and emergency providers to incorporate the findings of Bickell may be explained by the intense concern that a hypotensive patient causes in emergency health care workers. Doing *nothing* seems contrary to our training and can generate cognitive dissonance, not to mention negative feedback from our peers.

In many hospitals, withholding fluids in such a situation would be deemed a delay in care, a mistake. However, once we discover that treatments that we once valued are ineffective, it offers a chance to re-examine our assumptions about the nosology of illness.

In the setting of trauma, restoration of normal vital signs with crystalloid fails to improve outcomes. These findings suggests that in vertebrates, moderately low blood pressure contributes to clot formation and hemostasis. Excessive IV fluids may be harmful because they interfere with well-honed mechanisms of hemostasis that are the product of millions of years of evolution.

Definitive answers to the question of when to use fluids, what kind, and how much, during ED trauma resuscitation will require additional large randomized trials. Meanwhile, it is reasonable to raise questions when the nurse spikes another liter of saline on the next trauma patient. Is this treatment going to help or hurt? Although it might seem to fly in the face of the emergency ethos, this might be a situation where it is better to do less, not more.

-Joe Alcock



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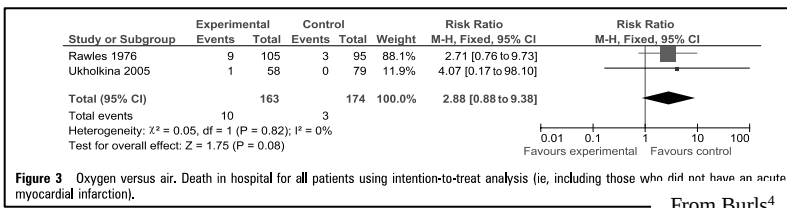
Rational Oxygen Therapy in the Emergency Department

Like giving fluid to hypotensive patients, giving oxygen to every ill-looking ED patient is a traditional practice that has proven difficult to break. Not surprisingly, the evidence for using oxygen for most ED situations is very weak.

While most people would agree that using oxygen for a minor viral illness or a fractured ankle is unlikely to help, the “basic science” rationale for treating myocardial ischemia with inhaled oxygen in order to improve the myocardial tissue oxygenation is more plausible. However, despite giving oxygen for angina for over 100 years, and the widespread recommendations for its use for “cardiac” chest pain over the last 60 years, there are many physiologic and patient-based reasons to suspect it may be doing more harm than good.

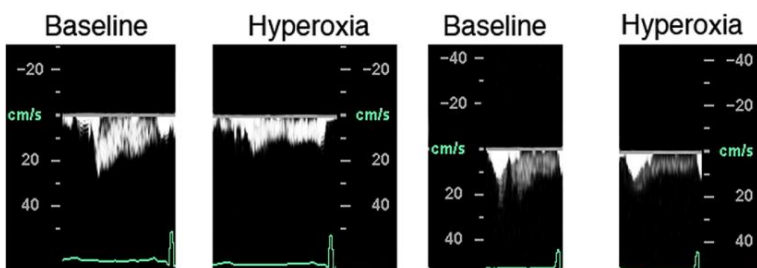
In 1950, JAMA published an article titled [One hundred percent oxygen in the treatment of acute myocardial infarction and severe angina pectoris](#)¹ that showed 100% oxygen caused increased and longer duration ECG findings of ischemia. Since then, there have been multiple studies that show the potentially detrimental physiological effects of high flow oxygen, but there have been few large patient-oriented trials that show benefit of supplemental oxygen. The *Cochrane Review* published a systemic review in 2010 titled [Oxygen therapy for acute myocardial infarction](#)² that concluded “There is no conclusive evidence from randomized controlled trials to support the routine use of inhaled oxygen in patients with acute AMI.” The accompanying editorial, titled [Oxygen therapy in acute myocardial infarction- too much of a good thing?](#)³ clearly opines that we are likely killing patients with our indiscriminate use of oxygen.

In 2011, *Emerg Med J* published an article titled [Oxygen therapy for Acute myocardial infarction: a systematic review and meta-analysis](#)⁴ that looked through 2529 articles before reviewing three randomized trials of oxygen versus air on suspected myocardial infarction that compared the outcomes of death, pain, and complications. These three trials had enrolled 387 patients and showed a trend toward increased death in the oxygen arms of the trials.



A landmark paper, titled [Revisiting the role of oxygen therapy in cardiac patients](#),⁵ was published in 2010 in *J Am Coll Cardiol* describes the mechanisms by oxygen could be harmful. They describe the coronary vasoconstriction due to hyperoxia, increased reactive oxygen species, changes in K⁺ and Ca⁺⁺ channels, angiotension II, and endothelin-1, and other vasoconstrictors. Additionally, the article briefly reviews animal studies that suggest oxygen decreases myocardial oxygen consumption, decreases capillary density, decreases oxygen diffusion, and causes a misdirection of blood flow and a reduction of oxygen consumption. They conclude that “the use of oxygen is clearly appropriate and advisable to treat hypoxia... excessive use of supplemental oxygen could potentially lead to worse outcomes.” Another paper published in *Am Heart J* in 2009 titled [Systematic review of studies of the effect of hyperoxia on coronary blood flow](#),⁶ concludes high-concentration oxygen has the potential to cause harm.

Normal Heart Transplant



Pulsed Doppler Recording of Left Anterior Descending Coronary Artery in Healthy Subjects and Cardiac Transplant Patients Blood flow velocity is significantly decreased in both groups during hyperoxia. (From Farquhar⁶)

Every paper that analyzes the utility of oxygen concludes that “additional research is needed.” Luckily for us our bros in Wellington were industrious enough to be conducting a prospective trial. They published their study in the Feb 2012 *Am Heart J* titled [High-concentration versus titrated oxygen therapy in ST-elevation myocardial infarction: a pilot randomized controlled trial](#).⁷ They took 136 STEMI patients (without hypoxia or shock) and randomized them to get either high-concentration O₂ via face mask or oxygen titrated to achieve a pulse ox of >93%. Neither the mortality, the troponin, or infarct mass was any different in either group.

Victoria Ambulance in Melbourne is currently conducting a prospective, multicenter, randomized controlled trial study that they described in a Mar 2012 *Am Heart J* article titled: [A randomized controlled trial of oxygen therapy in acute myocardial infarction Air Verses Oxygen In myocarDial infarction study \(AVOID Study\)](#).⁸ They intend to enroll 490 STEMI patients without hypoxia to either oxygen or no oxygen therapy. By collecting mortality and other clinical data, this study will hopefully be able to provide some evidence on the benefit or harm of oxygen. Another trial, which compares 10 lpm of O₂ to room air on STEMI patients, is titled [Supplemental Oxygen in Catheterized Coronary Emergency Reperfusion \(SOCCER\)](#),⁹ and is hoping to start recruiting soon in Sweden.

Until these results come back, you have to ask yourself if you would like to base your practice on a 1939 *JAMA* paper titled paper titled [One hundred per cent oxygen: indications for its use and methods of its administration](#),¹⁰ or modern data, multiple systematic reviews and several national guidelines.

Oxygen still is appropriate treatment for **HYPOXIA** (and *pneumothorax, carbon monoxide poisoning, dive injuries, and procedural sedation*), but there is very little evidence for it's use in the ED for SEMTI, chest pain, dyspnea, shock, or nearly any other ED condition that you can name. Giving oxygen to *non-hypoxic* patients is a potentially dangerous and likely wasteful treatment. Because of this, we should think very hard before reflexively starting it on our patients.

-Andy

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The British Thoracic Society's [2008 Guideline for Emergency Oxygen Use in Adult Patients](#) sums up a rational oxygen strategy pretty well:

- Oxygen is a treatment for **hypoxemia**, not breathlessness.
- Start oxygen on **critically-ill** patients (triage 1).
- Attempt to achieve normal or near-normal saturation on most acutely ill patients by maintaining s O₂Sat between **94-98%**.
- When using oxygen, use pulse oximetry.
- Reduce oxygen for those patients without hypoxia.
- Beware of the risks of oxygen on patients with chronic hypercarbia but generally maintain them between 88-92%.

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