# RSI in the Hypotensive Patient

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## Disclaimers

- **NONE! NADA! NULUS!**
- No industry or other affiliations, no sponsors, no nobody.
- **My Goal in Life:**
  - **To be the person my dog thinks I am.**


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## Indications for RSI

- Three fundamental clinical situations in which RSI should be considered:
  1. When there is a failure of airway maintenance or protection.
  2. When there is a failure of ventilation or oxygenation.
  3. In anticipation of the clinical course.

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## Seven "P's" of RSI

1. **P**reparation
2. **P**reoxygenation
3. **P**retreatmen
4. **P**ositioning
5. **P**aralysis with induction
6. **P**lace the tube + **P**roof of Placement
7. **P**ost-intubation management

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# Special RSI Cases

- Most patients the standard "7 P's" of RSI and proceeding down that sequence fairly rapidly is appropriate and not associated with any undue risk.
- However, in a number of patients RSI intubation can be associated with precipitous and severe deterioration due to the fact one has intubated the patient.
- That creates a difficult clinical problem – the patient need their airway controlled but there are issues that often result in deterioration from being intubated.
- These patients often fall into one of three categories.

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# The "HOP" Patient

- While a majority of patients are very appropriate for the usual rapid RSI sequence it is often the first two categories of the HOP patients where one might pause before going down the RSI pathway:
  - **Hemodynamically unstable/Hypotensive** – the patient who is in shock (best measured by the **shock index = HR/SBP > 0.8**) does not tolerate the medications for RSI well and often decompensates once the thoracic cavity is pressurized and venous return falls quickly followed by cardiac output.
  - **Oxygenation** – the patient with a **Pox < 93%** has little apneic reserve and any glitch to almost immediate intubation can be disastrous aside from the fact that many such patients remain difficult to oxygenate even once they are intubated.
  - **pH** – the acidotic patient while typically presents little problem with RSI their management with the ventilator is critical and if the respiratory rate is not taken into consideration the patient becomes more acidotic and can arrest.

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# The "H" in HOP

- Of the "HOP" group of patients the ones with pH issues are an issue with managing the ventilator, another lecture in of itself.
- The patients with oxygenation problems can often be managed with meticulous pre-oxygenation, often using NIV, and such ventilator techniques as aggressive PEEP to recruit alveoli to participate to getting oxygen into the blood.
- The **hypotensive patient** is the object of our attention in this presentation today and understanding the causes of hypotension and the effect of intubation/ventilation on cardiac function can help with both managing such patients and safely RSI'ing them.

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# Cardiac Physiology of Intubation

- The major cardiac effect of intubating a patient and placing them on a ventilator has to do with the effects of pressurizing the thoracic cavity.
- One routinely increases that effect with the introduction of PEEP to aid in alveolar recruitment.
- As one increases intra-thoracic pressure venous return to the heart falls, right sided cardiac output is reduced, blood return to the left atrium is decreased, less blood is presented to the left ventricle, ejection fraction is less, left sided cardiac output falls, and perfusion along with blood pressure is reduced – often precipitously.
- This is a perfect example of the effects illustrated by the Frank-Starling Curve.

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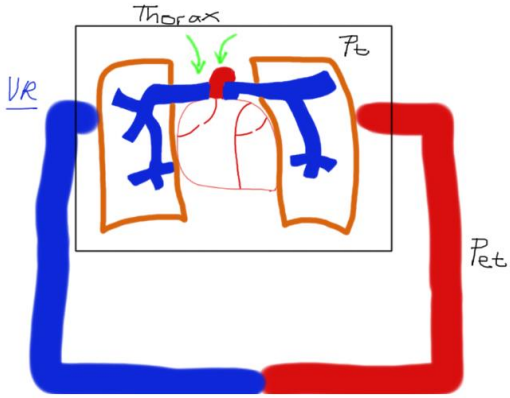
# Increasing Intrathoracic Pressure

**Venous return (VR) to the right atrium is passive due to a higher VR than RA pressure – blood flows into the RA and is pumped into the RV.**

**With increased intrathoracic pressure that VR to RA gradient drops, VR is reduced and less blood is pumped into the RV, less RV distension reduces RV stroke volume and RV cardiac output.**




**With a decrease in RV cardiac output there is a fall in blood return from the lungs and LA filling is reduced, this leads to reduced LV filling and distension.**

**The reduction in LV distension results in a lower LV stroke volume and cardiac output falls leading to impaired systemic perfusion, hypotension, and eventual seizure/coma/death.**

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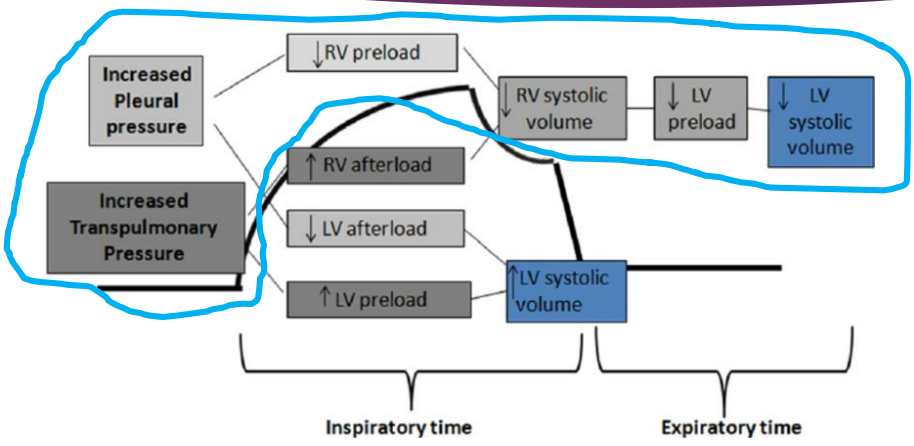
# Increasing Intrathoracic Pressure




**This is a more complex diagram but it does illustrate the relationship of increased pleural pressure, RV preload and SV impacting on LV preload and SV with the result be a fall in CO and perfusion.**

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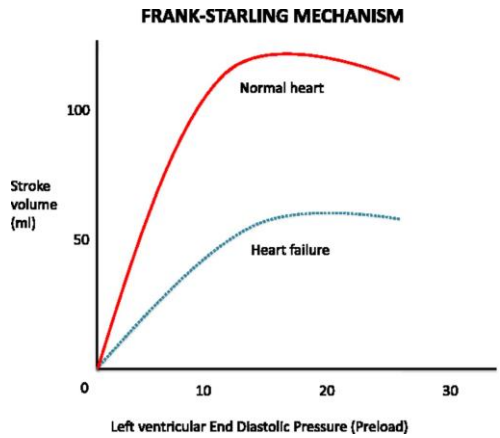
# Frank-Starling Curve




**This is the classic Frank-Starling curve of the relationship between end diastolic pressure (EDP) and stroke volume (SV).**

**Within broad parameters as EDP increases cardiac muscle contraction increases as does SV and thus CO.**

**This falls off with excessive EDP and the failing heart is less able to augment SV for a given increase in EDP.**

*This curve is for the LV but applies to the RV in the same manner.*

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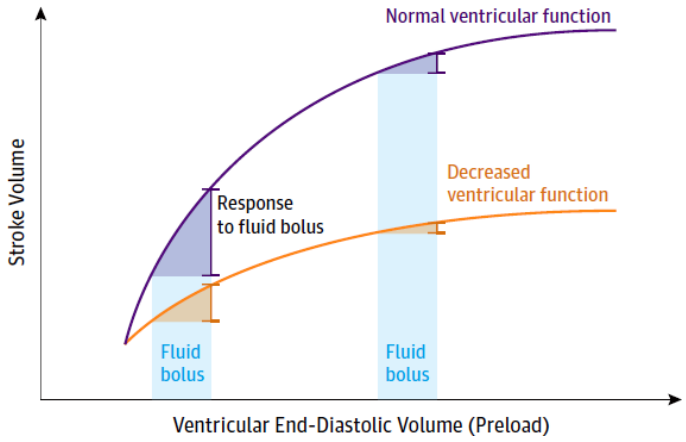
# Frank-Starling Curve - Fluids




**This shows how the administration of fluids can help as EDV (preload) moves the heart rightward and upward on the curve resulting in an increase in SV and thus CO.**

**This is less effective in the heart with a decreased ventricular function.**

**Again, this falls off with excessive ventricular EDV.**

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# Causes of Hypotension

- There are many causes of hypotension but the ones most common to effect RSI are:
  - Fluid/blood losses.
  - Cardiac dysfunction.
- Determining which cause is most applicable in a given patient may not be a simple as one would like:
  - Obvious hemorrhage or profuse vomiting/diarrhea – great, easy to determine etiology.
  - AMI on EKG along with symptoms of ACS – you know what you are dealing with.
  - Not uncommonly you do not know why the patient is in your department, you do not know the patient's name or history or medications but – they need emergent airway management – **and the patient is hypotensive**.

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# DSI

- How to address this subset of patients who need to be intubated but are likely to be adversely effected by the RSI sequence is best addressed with **DELAYED SEQUENCE INTUBATION.**
- First described by Dr. Scott Weingart* and the concept is essentially the usual RSI 7 P's but not in the usual 1 to 7 sequence with one step being followed essentially immediately after the next.**
- In the best case scenario of hypotension one can take some time to address the issue as one is preparing to intubate the patient as well as anticipating the potential for hypotension post-intubation – i.e. the patient does not need a crash intubation.

***Weingart SD, et al. Ann Emerg Med 2015;65:349.**

****Walters BL, Delayed Sequence Intubation, presented Critical Care in the ED conference, 3/2018.**

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## RSI Sequence in the Hypotensive Patient

- One starts with the usual RSI sequence:
  - **Preparation** – get all your staff together, everyone briefed on goals and steps, meds available and being drawn up (more on that later).
  - **Preoxygenation** – must be meticulous to extend the "safe apnea time", use NRB at 30 L/min + NC at 15 L/min, use of NIV can pressurize the thoracic cavity and exacerbate the hypotension.
  - **Pretreatment** – this is the main area where one delays the usual quick movement from step to step in the RSI sequence and involves several considerations.

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## Pretreatment in the Hypotensive Patient

- **Pretreatment**:
  - Fluids – even small amounts of an isotonic fluid can make a large difference in addressing not just the hypotension but post-intubation hypotension.
  - Establishing adequate IV's – 2 large bore peripheral, consider placing a "crash" central line, often femoral.
  - Blood replacement in the patient who is hypotensive from blood loss, O neg, and again even small amounts can make a big difference in both hypotension, post intubation hypotension, and oxygen carrying capacity.
  - Sedation might be necessary in selected patients and the drug of choice for this is **ketamine 0.5-1 mg/kg** – effective, least hypotensive effect of any sedative, analgesic and sedating, can proceed with an additional dose just before paralysis.

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## Pretreatment in the Hypotensive Patient

- **Pressors** –
  - Might be necessary in the severely hypotensive patient.
  - Infusion of a bolus of isotonic fluids would be prudent prior to starting a pressor.
  - Norepinephrine is the most commonly recommended pressor in both septic shock and undifferentiated shock, epinephrine is typically the second pressor recommended, dopamine is not generally recommended because of its tendency to cause arrhythmias, and phenylephrine is best if tachycardia occurs with either norepinephrine or epinephrine.
  - May need to give a **push-dose pressor** first while obtaining an infusion of a desired pressor as well as during the RSI sequence and/or post-intubation.

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## Pretreatment in the Hypotensive Patient

- **Push-dose pressors -**
  - Should be on hand in any hypotensive patient undergoing RSI.
  - Can be used both before actual intubation and/or post-intubation to manage hypotension as it is readily available and titratable.
  - Two agents are used for this – phenylephrine and epinephrine.
  - Phenylephrine comes in a ready-to-go pre-filled syringes.
  - Epinephrine can be made up using the 10:9:1 method of mixing up – 10cc syringe, 9cc's NS, 1cc epinephrine from a cardiac ampule = 100mcg/cc.
  - Dose is more a clinical judgement than a hard and fast amounts – 1-4cc's every 30-90 seconds is typical.

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# Next RSI Step - Positioning

- Positioning the patient would be as usual in the RSI sequence.
- Positioning can be most important in the obese patient – remember the *"ear to sternal notch"* position.


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# Paralysis with Induction

- The next step in the RSI sequence is as usual with the **administration of a paralytic** with an **induction agent** to sedate the patient just prior to paralysis.
- The recommended induction agent for the hypotensive patient is ketamine – cardiovascularly stable, unlikely to cause hypotension, may cause a modest rise in BP and pulse:
  - Dose = 2mg/kg, if one has given some ketamine to sedate the patient prior to paralysis given an additional amount to bring the patient up to 2 mg/kg.
- Etomidate is also an acceptable choice for RSI in the hypotensive patient, however the issue of adrenal suppression is not totally resolved and ketamine has been shown to be equally effective and is probably a better choice.

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# Rubber Meets the Road - Intubating

- The RSI steps to intubation include:
  - The patient is now sedated or on their way, it is time to paralyze and intubate.
  - **Paralysis** is with either the depolarizing agent succinylcholine (1.5 mg/kg) or a non-depolarizing agent like rocuronium (1 mg/kg).
  - **WAIT** at least a full minute before attempting to intubate to allow the paralyzing agent to fully work and provide the best intubating conditions with a fully paralyzed patient.
  - **Verify your tube placement** – ETCO2 detector, continuous ETCO2 monitoring, auscultation, and CXR.

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# Ventilator Settings

- Ventilator settings are extremely important in the hypotensive patient.
- Again this revolves around the problem of pressurizing the thoracic cavity and interfering with venous return to the right heart.
- Suggested considerations in ventilator settings would include:
  - Tidal volume – start at 6 ml/kg IBW, over distention of the lungs will impair venous return.
  - FiO2 – set according to the patient's oxygen status, if not intubating for hypoxia one can set the FiO2 to 30-40%.
  - PEEP – increases intrathoracic pressure, for the hypotensive patient start with a PEEP of 0, as the BP comes up (hopefully?) one can add PEEP back slowly.
  - Respiratory rate – starting at 14-16/minute is a reasonable starting point, look at the flow/time curve and plateau pressures and adjust accordingly.

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# Auto-PEEP, Plateau Pressure

- In order to lessen the impact of increasing intrathoracic pressure with intubation attention to both auto-PEEP and plateau pressure is necessary.
- Auto-PEEP occurs when there is a longer than normal expiratory phase such that the ventilator begins the next breath before the patient fully expires – this leads to increasing PEEP with each breath.
- Plateau pressure reflects alveolar pressure and is measured by pausing the ventilator at full inspiration and measuring that pressure.
- Looking at the flow/time graph of the ventilator illustrates these points.

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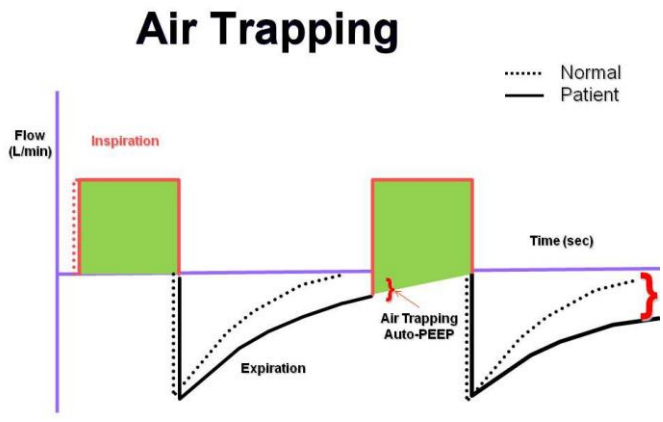
# Auto-PEEP, Plateau Pressure




**This flow/time graph shows both the normal patient (dotted line) where full expiration occurs before the next breath is initiated.**
**Air trapping is seen (solid line) where inspiration begins but full expiration has not occurred – this is auto-PEEP.**

**Solution: first decrease respiratory rate and/or one can increase expiratory time by giving the patient more time to expire by changing the I:E ratio from the usual 1:2 to 1:3 or 1:4.**

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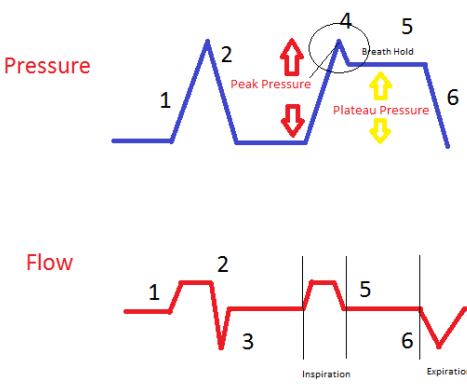
# Auto-PEEP, Plateau Pressure




Here we see the pressure/time graph with the ventilator pausing at peak inspiration, essentially the patient is holding their breath. The pressure drops a bit from peak inspiration to the plateau pressure (PP) that correlates with alveolar pressure. The goal is < 30 cmH2O. If the plateau pressure is > 30 one can be excessively distending the alveoli and worsening venous return.

Solution to PP > 30: reduce TV, if you started at 8 ml/kg go to 7 or 6, re-measure PP, keep reducing until the PP is < 30 or you hit 4 ml/kg, you may need to accept some permissive hypercapnia.

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# Summary

- RSI in the hypotensive patient is a significant challenge as the process of RSI may worsen hypotension, decrease perfusion, and lead to the inevitable consequences of multiorgan failure.
- The issue primarily revolves around the fact that by intubating the patient and placing them on a ventilator there is a concomitant increase in intrathoracic pressure.
- This leads to reduced return to the right side of the heart, decreased right sided cardiac output, that then results in decreased blood return to the left side of the heart, and finally falling cardiac output – Frank-Starling curve.

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# Summary

- To address this issue relies on altering the usual step-wise sequence of RSI that typically flows from one step to the next quickly.
- Delaying that usual sequence to address the hypotension can take a number of steps:
  - Establish adequate IV's up to and including a central line.
  - Fluids – isotonic fluids or blood depending on the etiology of the hypotension.
  - Pressors – particularly if cardiac dysfunction is the source of hypotension that might be necessary – either by infusion or push-dose pressor.
- Positioning, paralysis with induction, and passing/verifying the endotracheal tube is as usual.

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# Summary

- Setting the ventilator in the hypotensive patient becomes the next challenge:
  - Tidal volume – err on the side of a lower tidal volume so as reduce the chance of over-distending the lung/alveoli – 6 ml/kg.
  - Oxygenation – in response to if the patient needs a high FiO2 as in any other intubated patient.
  - Set PEEP to zero initially – as things settle down you might add small amounts.
  - Assess the time/flow curve looking to prevent auto-PEEP, the patient needs to fully expire before the next breath, adjust RR and/or I:E ratio.
  - Plateau pressure reflects alveolar pressure and the goal is < 30 cm/H2O, if greater than that reduce tidal volume.

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# Summary

- Frequent reassessments as in any critically ill intubated patient.
- One might need an arterial line, do not hesitate to place one.
- Judicious re-adjustment of the ventilator as things may improve, generally the first thing I do is add back some PEEP.

**THANK YOU FOR YOUR KIND ATTENTION – QUESTIONS?**

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**I hope your day is a nice one like this day a few weeks ago up on the Manistee Rive.**

**I hope all your RSI's go well.**

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