Below is a discussion of tamponade physiology:

Right ventricular diastolic and right atrial systolic collapses are seen with pericardial tamponade. During ventricular systole the ventricular pressure is high preventing ventricular collapse and favoring collapse of the low pressure right atrium. During diastole the ventricular pressure falls and right ventricular diastolic collapse occurs.

Reciprocal respiratory changes can be explained as follows: fluid in not compressible and with pericardial tamponade the total pericardial volume (heart chambers plus pericardial fluid) is fixed. If right ventricular volume is high then left ventricular volume will be low and vice versa. During *spontaneous inspiration* intrathoracic pressure decreases and the thin walled right ventricle dilates increasing venous return. This increases right ventricular filling, and transtricuspid flow velocities. As the right ventricle fills right ventricular pressure exceeds left ventricular pressure and the interventricular septum shifts to the left compressing the left ventricle. This LV compression decreases transmitral inflow and decreases left ventricular stroke volume (TVI_{LVOT}). This is the mechanism that explains why systemic pressure, transmitral inflow, and LV stroke volume all decrease with spontaneous inspiration. The opposite occurs with spontaneous expiration.

POSITVE PRESSURE VENTILATION and TAMPONADE: positive pressure ventilation is more complicated in patients with tamponade physiology. Institution of positive pressure ventilation in tamponade patients following induction of general anesthesia can cause a profound decrease in left ventricular stroke volume resulting in hemodynamic collapse. That is certainly the most important clinical info. If a spontaneously breathing patient takes a deep breath and holds is for a few seconds while you are palpating their radial pulse, and that pulse disappears (pulsus paradoxus) then be very careful and consider maintaining spontaneous ventilation until the fluid is drained.

Interestingly, as described above, these patients have exaggerated reciprocal respiratory changes to LV and RV filling during spontaneous ventilation but don't have increased reciprocal respiratory changes in ventricular filling and stroke volume following institution of positive pressure ventilation. Normal euvolemic patients have reciprocal changes in RV and LV filling with spontaneous *and* positive pressure ventilation.

Spon inspiration → increased RV filling + decreased LV filling.

Spon expiration → decreased RV filling + increased LV filling.

The opposite happens in normally breathing euvolemic patients undergoing positive pressure ventilation. Tamponade causes an exaggeration in the changes seen during spontaneous ventilation. However the effects during positive pressure ventilation are NOT exaggerated. Indeed the effects during positive pressure ventilation are actually attenuated as described by Jana A. Faehnrich et al. in the reference below (2). These investigators looked at transmitral inflow velocity variation with respiration during positive pressure ventilation in intubated dogs before and after inducing tamponade with warm saline. They found that transmitral inflow velocities (a surrogate for predicting LV filling and thus LV stroke volume) normally varies during positive pressure ventilation. This variation is the inverse of that seen during spontaneous negative pressure ventilation. Positive pressure inspiration increases early transmitral filling (by compressing the pulmonary venous system and increasing left atrial pulmonary venous return) for the first few beats following the onset of positive pressure inspiration. This is followed by a decrease in transmitral inflow during positive pressure expiration. In cardiac tamponade things are different. During inspiration spontaneously breathing patients have an exaggerated increase in transtricuspid inflow velocities during negative pressure inspiration and an exaggerated decrease in transmitral inflow. The opposite occurs with spontaneous expiration. However,

unlike spontaneously breathing patients (neg pressure ventilation) who have exaggerated respiratory variation in transmitral flow, tamponade patients undergoing positive pressure ventilation have diminished changes in transmitral inflow velocities compared to control patients undergoing positive pressure ventilation without tamponade.

The authors surmise that this occurs because the severity of diastolic filling impairment caused by the compressive pericardial fluid collection is the predominant factor that limits cardiac performance and respiratory variations in pleural and pericardial pressures have little impact on left heart filling which is already critically compromised by tamponade.

However, if you remember nothing else from this discussion/feedback remember that these patients can have profound hypotension following institution of positive pressure ventilation and thus may benefit from maintaining spontaneous ventilation.

References:

1.Textbook of Clinical Echocardiography, by Otto 3rd edition page 262.

2.Effects of Positive-Pressure Ventilation, Pericardial Effusion, and Cardiac Tamponade on Respiratory Variation in Transmitral Flow Velocities
Journal of Cardiothoracic and Vascular Anesthesia, Vol 17, No 1 (February), 2003: pp 45-50, Jana A. Faehnrich,et.al