
Minneapolis
Heart Institute
Foundation
GRAND ROUNDS

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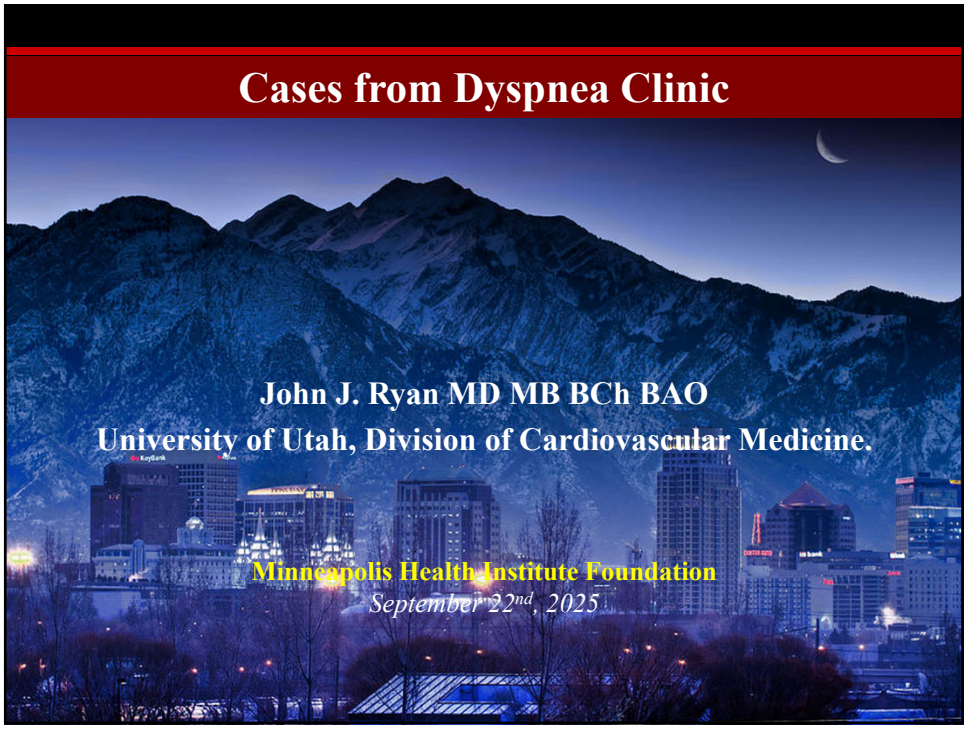
Cases from Dyspnea Clinic
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Minneapolis Health Institute Foundation
September 22nd, 2025

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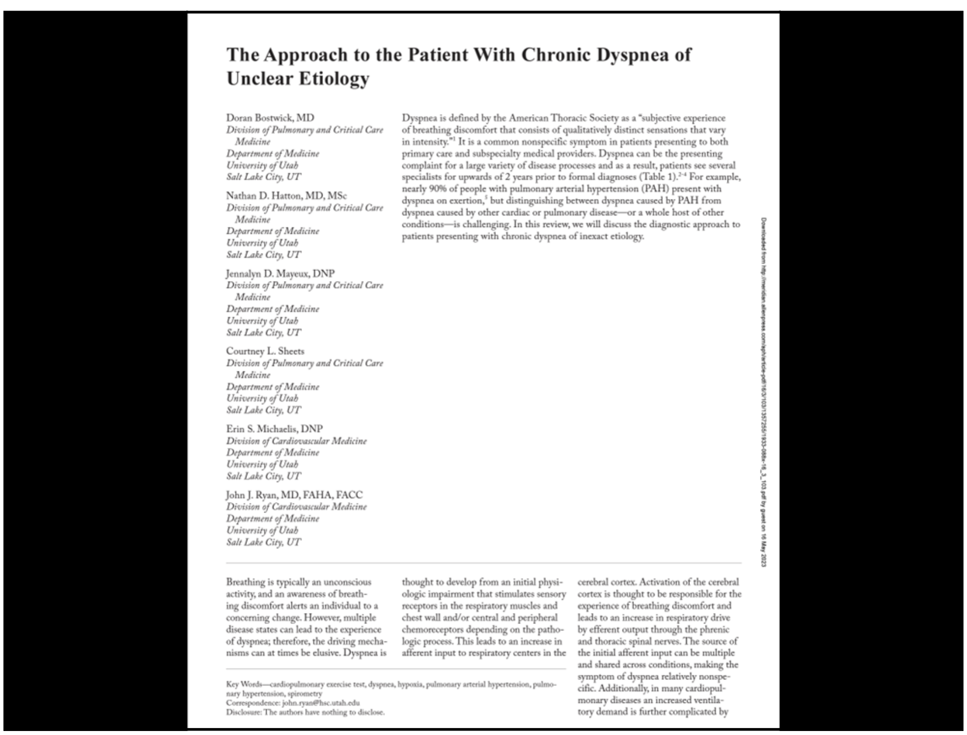

The Approach to the Patient With Chronic Dyspnea of Unclear Etiology

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Dyspnea is defined by the American Thoracic Society as a "subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity."[1] It is a common nonspecific symptom in patients presenting to both primary care and subspecialty medical providers. Dyspnea can be the presenting complaint for a large variety of disease processes and as a result, patients see several specialists for upwards of 2 years prior to formal diagnoses (Table 1).[2-4] For example, nearly 90% of people with pulmonary arterial hypertension (PAH) present with dyspnea on exertion,[5] but distinguishing between dyspnea caused by PAH from dyspnea caused by other cardiac or pulmonary disease—or a whole host of other conditions—is challenging. In this review, we will discuss the diagnostic approach to patients presenting with chronic dyspnea of inexact etiology.

Breathing is typically an unconscious activity, and an awareness of breathing discomfort alerts an individual to a concerning change. However, multiple disease states can lead to the experience of dyspnea; therefore, the driving mechanisms can at times be elusive. Dyspnea is thought to develop from an initial physiologic impairment that stimulates sensory receptors in the respiratory muscles and chest wall and/or central and peripheral chemoreceptors depending on the pathologic process. This leads to an increase in afferent input to respiratory centers in the cerebral cortex. Activation of the cerebral cortex is thought to be responsible for the experience of breathing discomfort and leads to an increase in respiratory drive by efferent output through the phrenic and thoracic spinal nerves. The source of the initial afferent input can be multiple and shared across conditions, making the symptom of dyspnea relatively nonspecific. Additionally, in many cardiopulmonary diseases an increased ventilatory demand is further complicated by

Key Words—cardiopulmonary exercise test, dyspnea, hypoxia, pulmonary arterial hypertension, pulmonary hypertension, spirometry
Correspondence: john.ryan@hsc.utah.edu
Disclosure: The authors have nothing to disclose.

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Case MW

- A 19-year-old female from Idaho presented in respiratory failure after blood transfusion for severe menorrhagia.
- The patient had been raised previously in a rural mountain community that did not engage in medical care. She had been experiencing cyanosis and syncope for 2 years before emancipating herself from her family.

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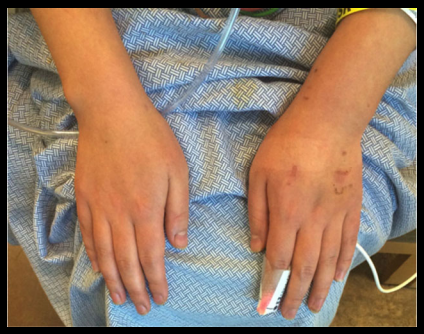
On exam

- On transfer to the cardiovascular intensive care unit, the patient was interactive.



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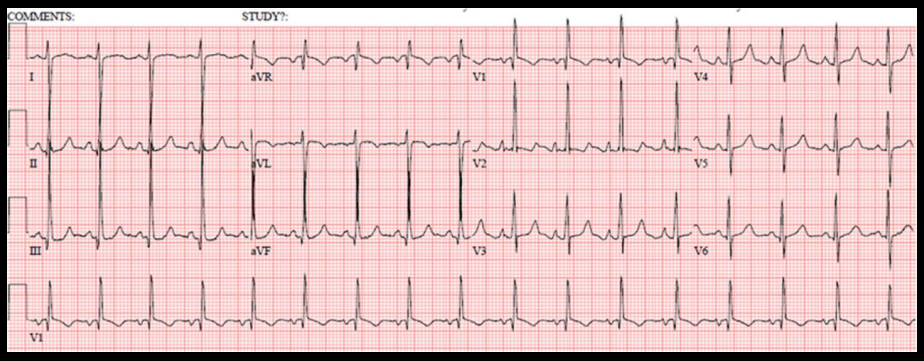
ABG

Variable	Right Radial ABG	Left Radial ABG
pH	7.399	7.418
$PaCO_2$, mmHg	33.7	34.7
PaO_2, mmHg	86.6	51.7
Carboxyhemoglobin, %	2.0	1.6
Methemoglobin, %	0.8	1.3

7

ECG



NSR, RAD, RVH

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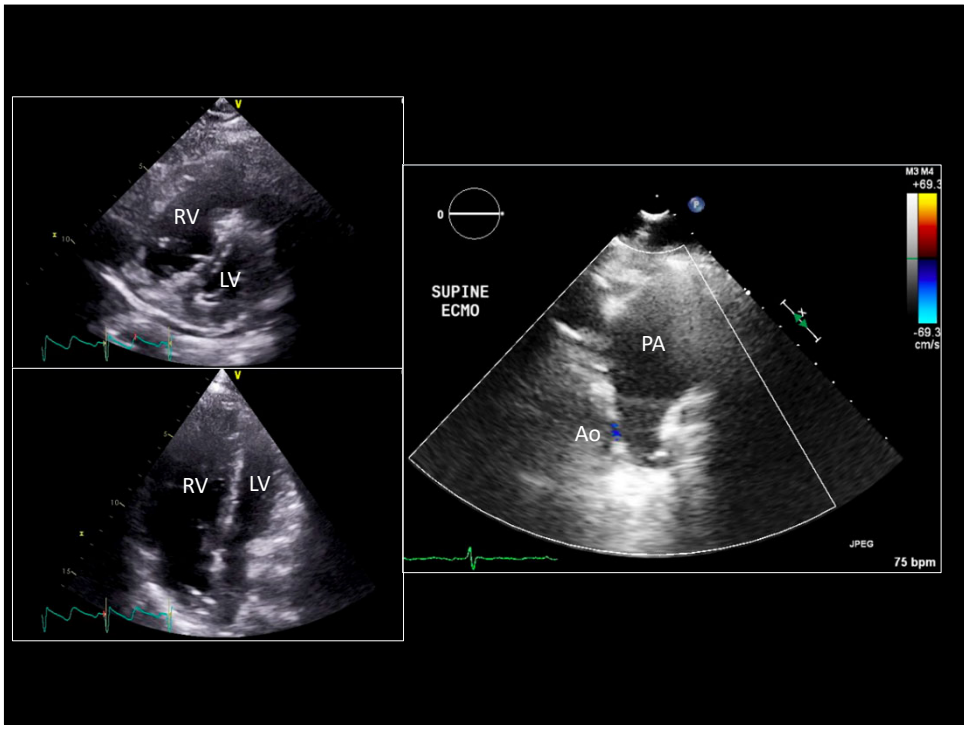

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Right heart catheterization

RA (mmHg)	13
PA (mmHg)	92/60, 71
PCWP (mmHg)	10
CO (L/min)	3.8
PVR (WU)	16
Systemic BP (mmHg)	105/60

SVC sat: 68%
PA sat: 62%
PV sat: 90%
Peripheral sat: 78%

No significant step-up in oxygen saturation from the main pulmonary artery to the branch vessels.

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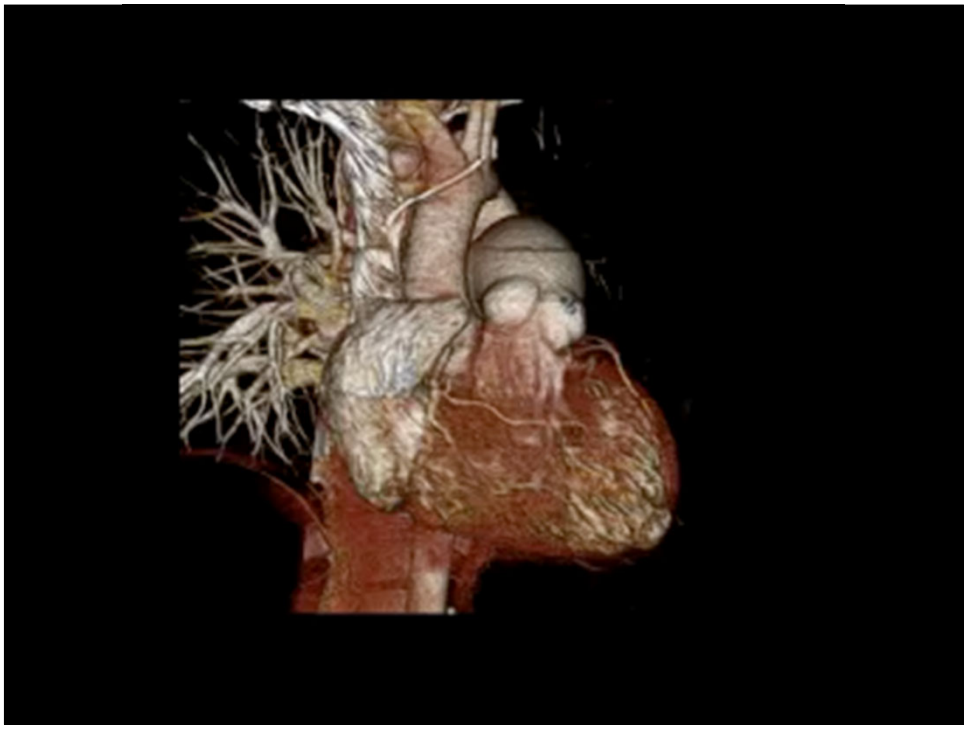

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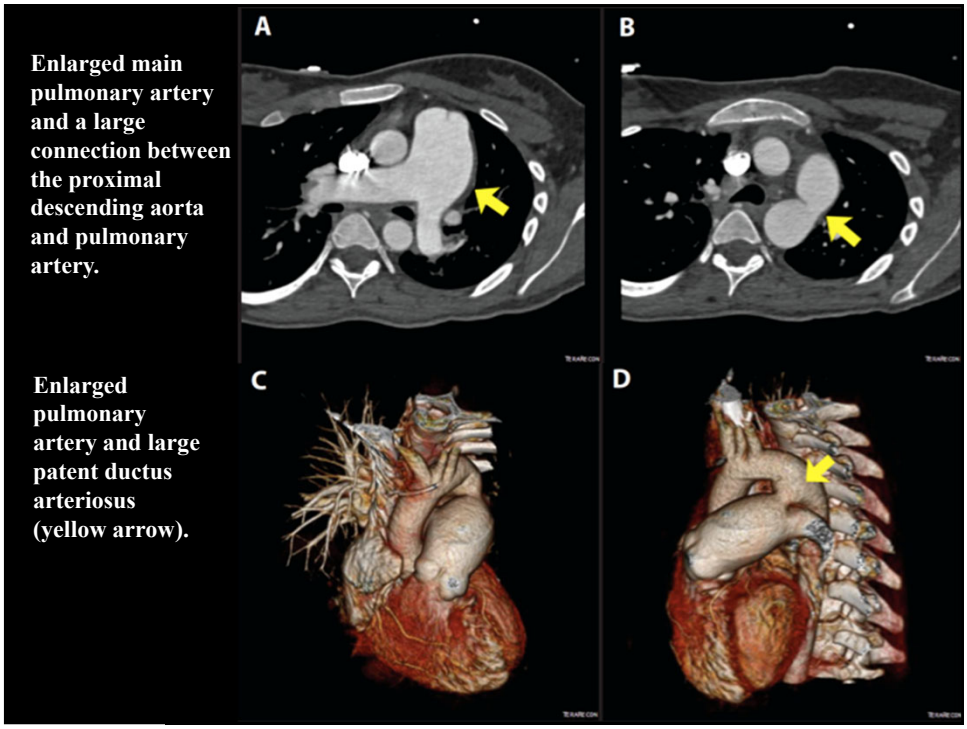
Enlarged main pulmonary artery and a large connection between the proximal descending aorta and pulmonary artery.
A
B
Enlarged pulmonary artery and large patent ductus arteriosus (yellow arrow).
C
D

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• Initial contrast opacification of the right atrium and ventricle.
• This is followed by filling of the pulmonary artery and descending thoracic aorta before opacification of the left ventricle.
• Continued imaging demonstrates filling of the left ventricle, aortic root, and ascending aorta.
NOT FOR MEDICAL USAGE

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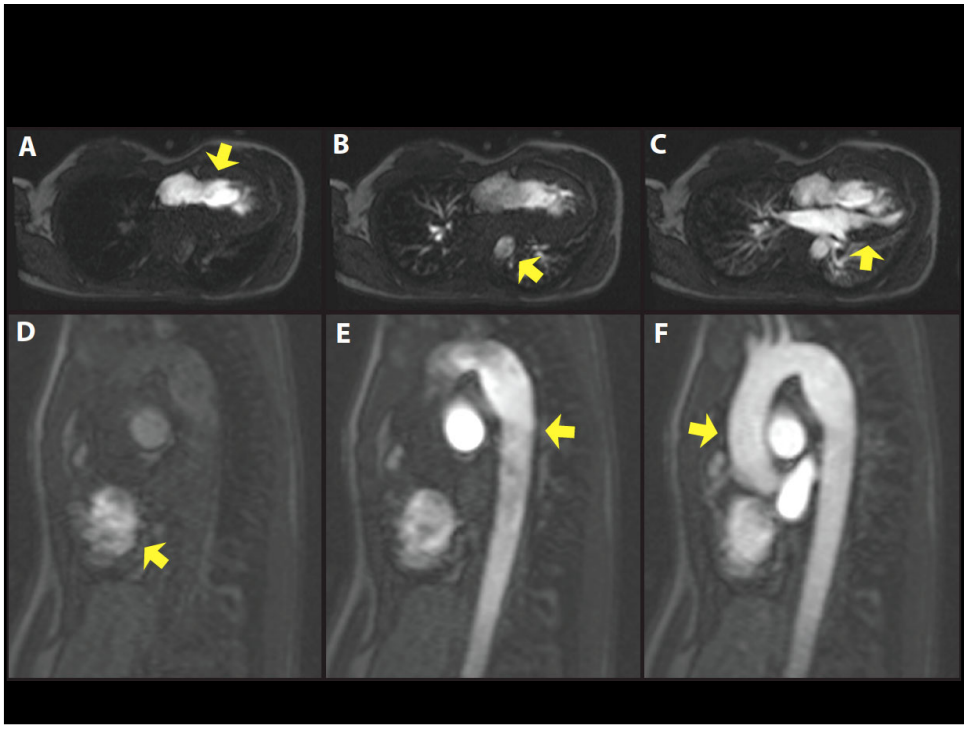
A
B
C
D
E
F

14

Eisenmenger Syndrome With Unrepaired Patent Ductus Arteriosus

- Started on oral and parenteral PAH therapies.
- Referred for lung transplant.
- 10 years later, she is doing well on PAH-specific therapies.

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Case HH

- 47-year-old Caucasian male former Mixed Martial Arts (MMA/UFC) fighter from Nevada presented to liver clinic for elevated liver function tests and a 35-pound weight loss associated with nausea, vomiting, and diarrhea.

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On exam

- JVP of 15 cm.
- Normal S1 and S2 without additional sounds .
- Hepatomegaly with a firm liver edge 4 cm below the right costal margin.
- Trace peripheral edema

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PMH

- Concussions.
- Hypertension.
- Chronic pain related to musculoskeletal injuries and fractures secondary to MMA competition.
- Right shoulder surgery.
- Foot surgery.
- 20-year history of performance enhancing drugs, including testosterone that ceased 8 months prior.

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Work up

- ALT: 1155 U/L
- AST: 219 U/L

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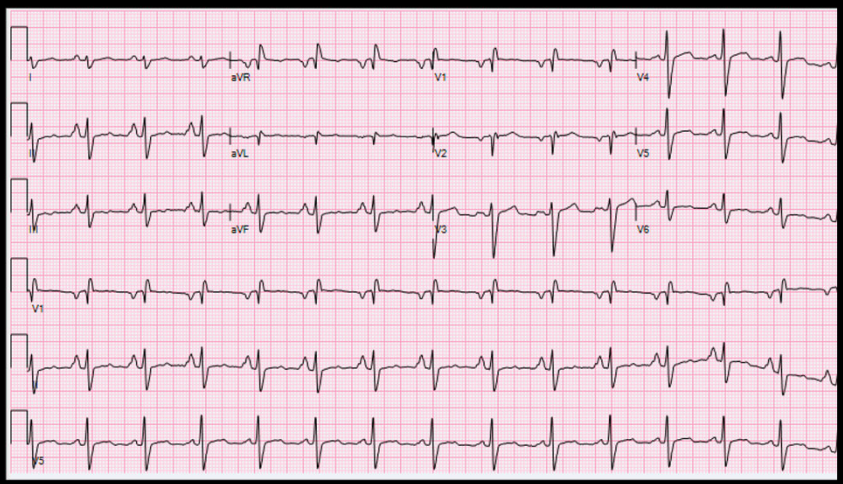
ECG



NSR, RBBB

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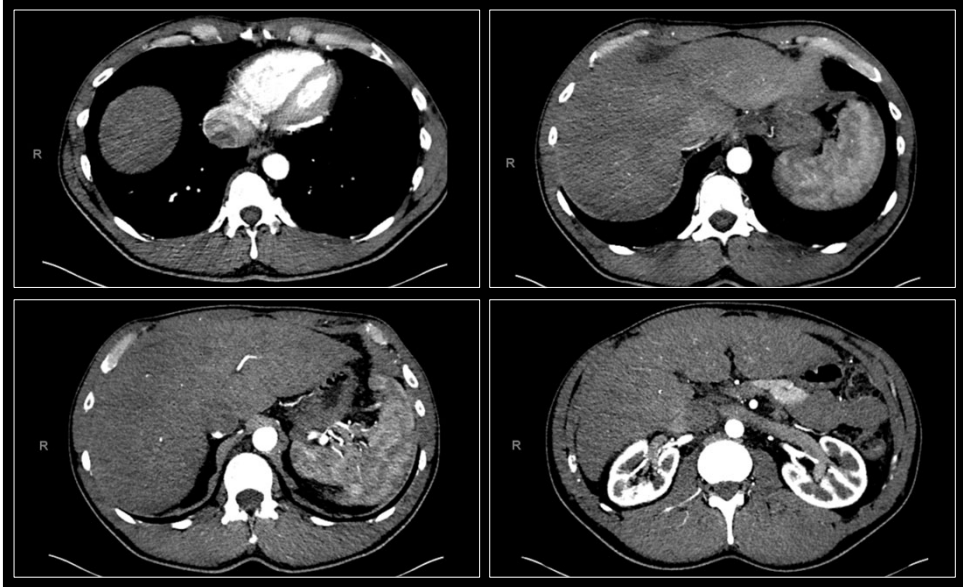
CT liver



Hepatomegaly with steatosis, ascites,
and dilated inferior vena cava.

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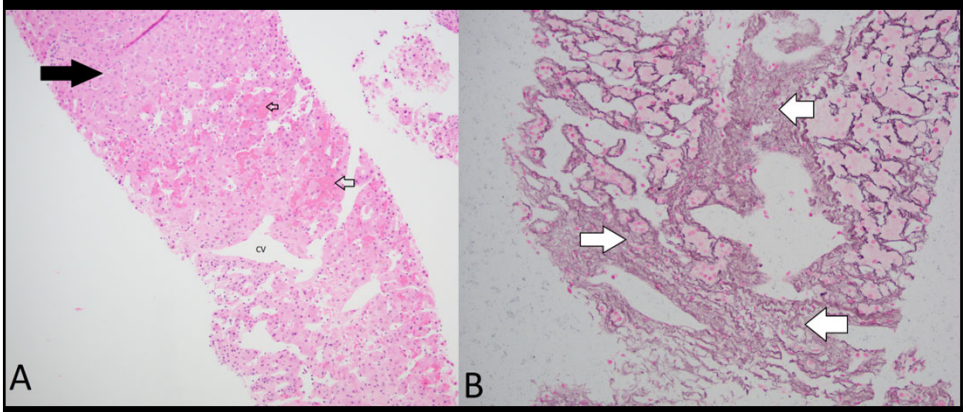
Liver biopsy: pathology



A) Histologic slides of this liver biopsy show centrizonal congestion and dilation with extravasated blood (open arrows) surrounding the central vein (CV). Compare this with normal hepatic lobular architecture (black arrow).

B) There is loss of hepatocytes around the central vein, highlighted by an increase in reticulin staining (white arrow).

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Liver biopsy: Haemodynamics

- Right atrial pressure: 12 mmHg.
- Free hepatic vein pressure: 12 mmHg.
- Wedged sinusoidal pressure: 13 mmHg.
- Hepatic vein portal vein gradient: 1 mmHg.

Post hepatic portal hypertension secondary to right heart failure

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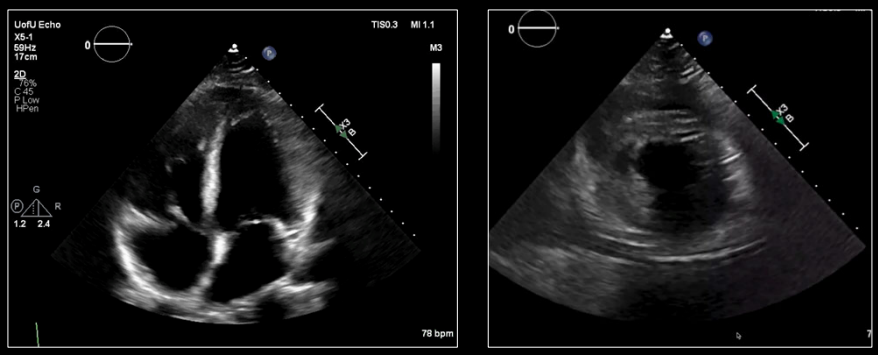
Echocardiogram



Abnormal septal motion suggestive of ventricular interdependence

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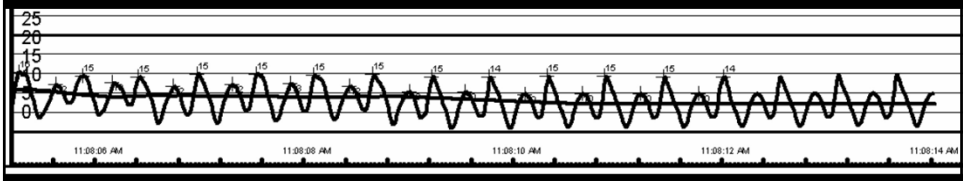
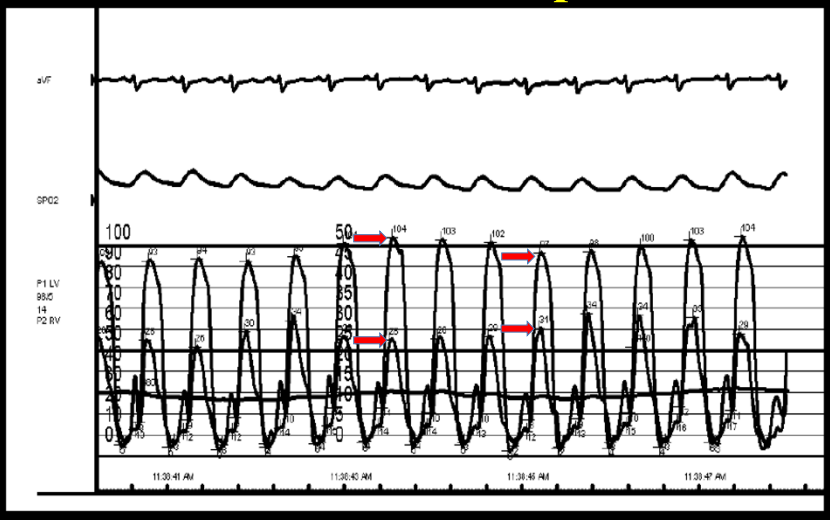
Cardiac Catheterization: Right atrial pressure



Elevated RA pressure with a steep *y* descent

25

Cardiac Catheterization: Simultaneous RV/LV pressure



Right ventricle-left ventricle discordance, consistent with constrictive physiology

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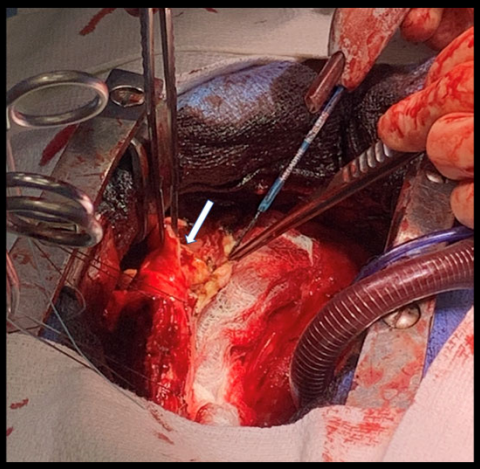
Pericardiectomy: calcification and thickening of pericardium



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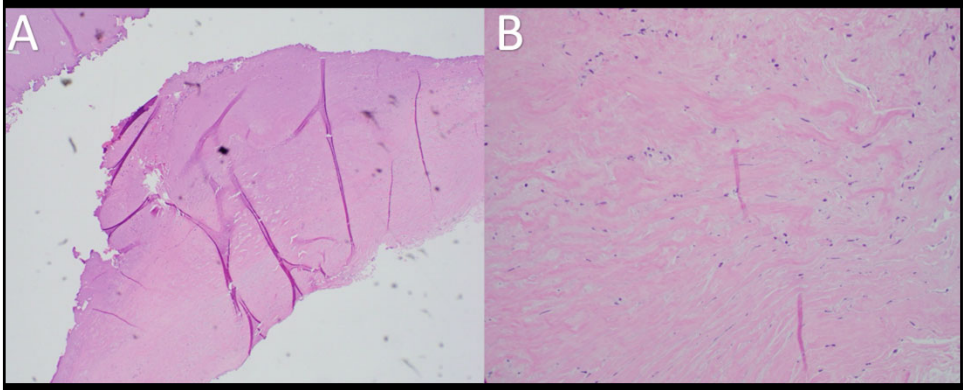
Histology



Chronic pericarditis:
Dense fibrosis without much chronic inflammation point towards repeated trauma.

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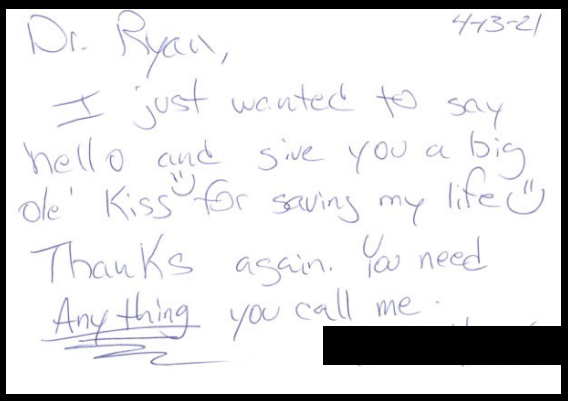
One year follow-up



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Case NN

- 18-year-old male from Idaho noticed new onset, increased shortness of breath.
- Resolved with rest but escalated with progressively less exercise.

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- One month into his symptoms he woke up with significant right arm swelling.
- Seen by PCP who sent him to Urgent care.
- He was told that he was sleeping on it wrong, was prescribed an inhaler and no evaluation performed.
- Family were concerned and took him to Emergency Department.

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US of Right Upper Extremity

INDICATION: Right upper extremity swelling TECHNIQUE: Two-dimensional real-time imaging, color flow imaging, and Doppler spectral analysis were performed. COMPARISON: No comparisons FINDINGS: Extensive occlusive thrombus throughout the right subclavian, axillary and proximal basilic veins with some extension into the internal jugular vein. There is also thrombus in 1 of the proximal brachial veins. Cephalic vein, antecubital vein and the forearm cephalic and radial veins are patent.

Impression: Extensive thrombus in the right upper extremity as described above. DATE OF

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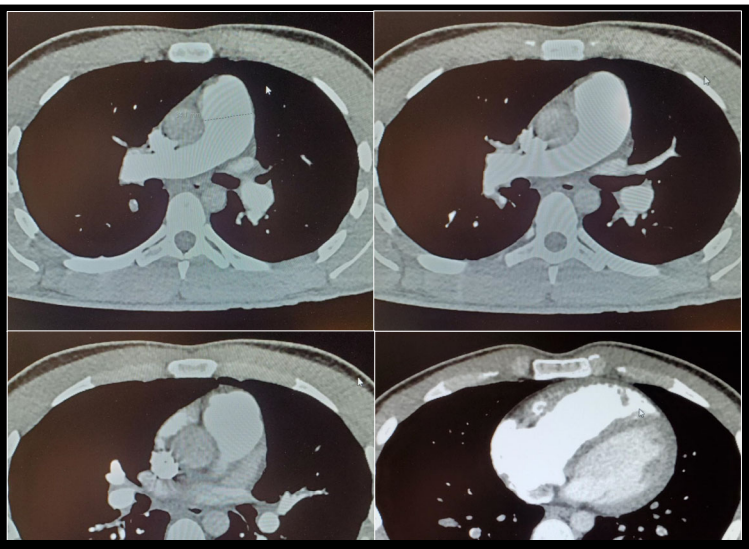

1. **Overall moderate load of pulmonary embolism with bilateral emboli seen throughout the segmental and subsegmental vessels in every lobe.**
2. **Subtle findings suggesting right heart strain with flattening of the interventricular septum and slight prominence of the right ventricle.**
3. **Mild ground-glass opacity in the perihilar lungs bilaterally. This may represent some pulmonary hemorrhage or edema.**

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- *"Patient underwent catheter directed tPA thrombolysis over 24 hours and was discharged on clopidogrel and rivaroxaban. Since this intervention, the patient's arm swelling and SOB has resolved."*

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Why would a young man develop upper extremity DVT and PE?

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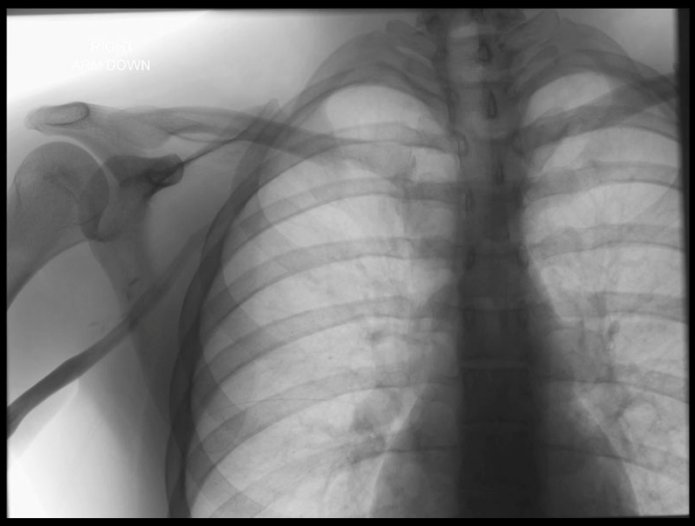
Upper extremity Venogram (1)



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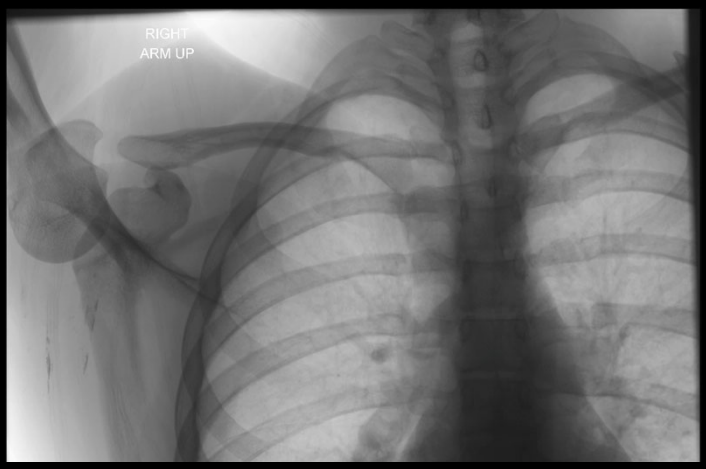
Upper extremity Venogram (2)



- With arms adducted there is a mild stenosis of the subclavian vein at the juncture of the first rib and clavicle. No thrombus is identified.
- With arm above the head, there is severe stenosis of the subclavian vein at the juncture of the first rib and clavicle with opacification of numerous collateral veins.

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Diagnosis

Paget-Schroetter Syndrome
- Venous thoracic outlet syndrome causing upper extremity DVTs.

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Referred for first rib resection

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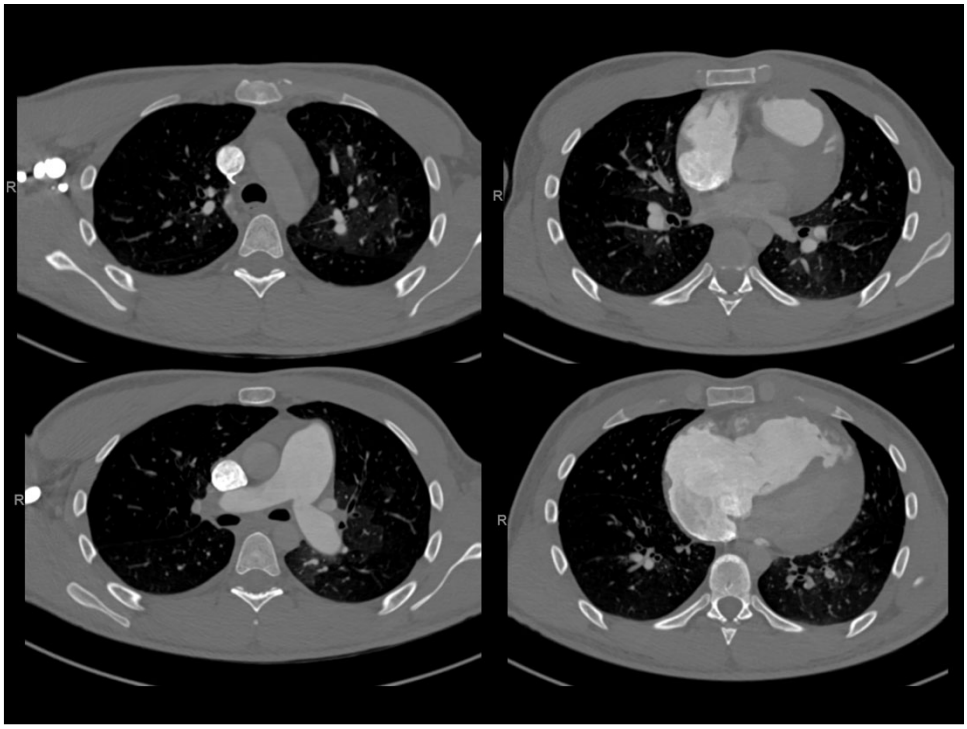

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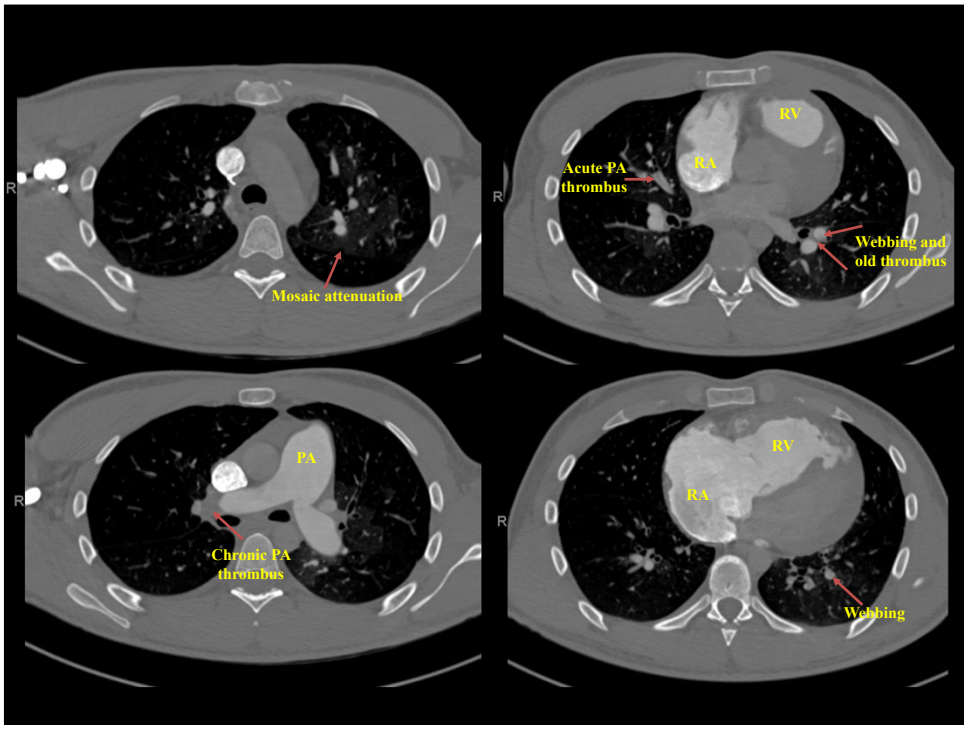
Mosaic attenuation
Acute PA thrombus
RV
RA
Webbing and old thrombus
PA
Chronic PA thrombus
RV
RA
Webbing

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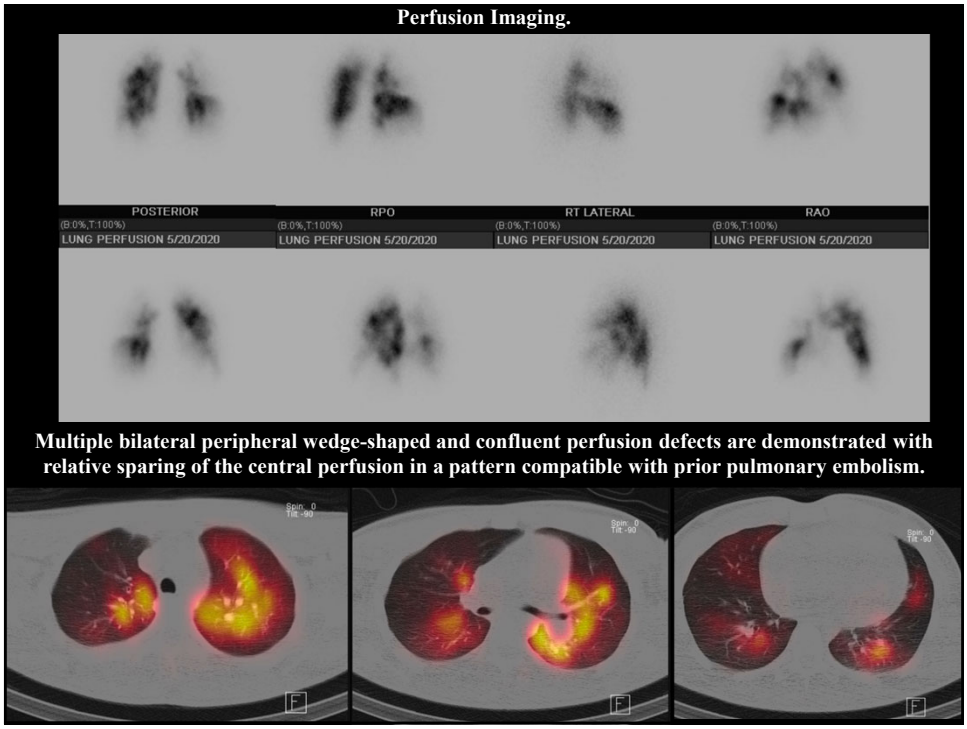
Perfusion Imaging.
POSTERIOR
RPO
RT LATERAL
RAO
LUNG PERFUSION 5/20/2020
Multiple bilateral peripheral wedge-shaped and confluent perfusion defects are demonstrated with relative sparing of the central perfusion in a pattern compatible with prior pulmonary embolism.

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Right heart catheterization

RA (mmHg)	13
PA (mmHg)	84/41, 55
PCWP (mmHg)	22
CO (L/min)	2.97
PVR (WU)	11
Systemic BP (mmHg)	111/69
PA sat	45%
Ao sat	92%

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Diagnosis

Paget-Schroetter Syndrome

↓

Chronic Thromboembolic Pulmonary Hypertension.

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Treatment?

- Pulmonary Endarterectomy?
- Rib resection(s)?
- Pulmonary Endarterectomy + Rib resection(s)?

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Reviews

Atrial Septostomy: A Contemporary Review

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ABSTRACT

Pulmonary arterial hypertension (PAH) is a rare disease, but it boasts significant morbidity and mo
Although remarkable achievements have been made in the medical treatment of PAH, there is a r
invasive or surgical procedures in patients with progressive disease despite optimal medical therapy
no access to such therapy. Atrial septostomy creates a right-to-left intracardiac shunt to decompre
overloaded right ventricle. Despite significant advances to validate and improve this palliative procedu
well as recent reports of improved outcomes, it is only slowly being adopted. This article aims to det
history, indications, contraindications, procedural techniques, and outcomes of atrial septostomy. W
also shed light on some of the newer interventions, inspired by the same physiological concept, that are
evaluated as potential palliative modalities in patients with PAH.

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ASAIO Journal 2025 Adult Circulatory Support

PLACE: Multicenter Study for Right Ventricular Failure on Mechanical Cardiocirculatory Supports

Valeria Lo Coco,* Michele Di Mauro,* Antonio Loforte,†‡ Thomas Fux,§¶ Dominik Wiedemann,‖ Tom Verbelen,# Lars Mikael Broman,** Jamila Kremer,†† Matteo Pozzi,‡‡ Koji Takeda,§§ Udo Boeken,¶¶ Yih-Sharng Chen,‖‖ Paolo Masiello,## Dominik J. Vogel,*** Jacinta J. Maas,††† Andrea Ballotta,‡‡‡ Federico Pappalardo,§§§ Kasia Hryniewicz,¶¶¶ and Roberto Lorusso*‖‖‖, On behalf of PLACE Study Group

Isolated acute right ventricular failure (aRVF) is associated with poor prognosis in different scenarios. In severe conditions, temporary mechanical cardiocirculatory support (tMCS) is required. PLACE is an international, retrospective, multicenter registry including 17 centers that investigated patients affected by isolated aRVF and treated with various types of tMCS from January 2000 to December 2020. The registry included 644 (69.6% males, mean age: 55 years) patients. The most frequent etiologies were post-left ventricular assist device implantation (LVAD) and postcardiotomy shock. These patients received mostly mechanical circulatory support (MCS) and veno-arterial extracorporeal membrane oxygenation. Mean tMCS duration was 9 days, weaning was achieved in 70.5% of the patients, and the major cause of death on support was multiorgan failure (50.5%). The mortality rate was 45 and 48.4% in-hospital and at 3 month follow-up, respectively. Multivariable logistic regression analysis identified age, aRVF due to acute pulmonary hypertension, bilirubin level, and oliguria or anuria at tMCS implantation as risk factors for in-hospital mortality. Conversely, aRVF after LVAD was found to be associated with a lower risk of early mortality. In-hospital and 3 months mortality occurred in less than half of the aRVF-supported subjects. Furthermore, several preimplant aspects such as age, organ function, and type of tMCS are independently associated with in-hospital and 3 month mortality. *ASAIO Journal* 2025; 71:290–299

From the *Department of Cardio-Thoracic Surgery, Maastricht University Medical Centre, Maastricht, the Netherlands; †Department of Cardiac Surgery, Sant' Orsola University Hospital, Bologna, Italy; ‡Department of Surgical Sciences, University of Turin, Turin, Italy; §Department of Molecular Medicine and Surgery, Karolinska Institute, Stockholm, Sweden; ¶Department of Perioperative Medicine and Intensive Care, Karolinska University Hospital, Stockholm, Sweden; ‖Department of Cardiac Surgery, Medical University of Vienna, Vienna, Austria; #Department of Cardiac Surgery, University Hospitals Leuven, Leuven, Belgium; **Extracorporeal Membrane Oxygenation Centre Karolinska, Karolinska University Hospital, Stockholm, Sweden; ††Department of Cardiac Surgery, University Hospital Heidelberg, Heidelberg, Germany; ‡‡Department of Cardiac Surgery, Louis Pradel Cardiologic Hospital, Lyon, France; §§Department of Cardiac Surgery, Columbia University Irving Medical Center, New York, New York; ¶¶Department of Cardiac Surgery, University Hospital Düsseldorf, Düsseldorf, Germany; ‖‖Department of Cardiovascular Surgery,

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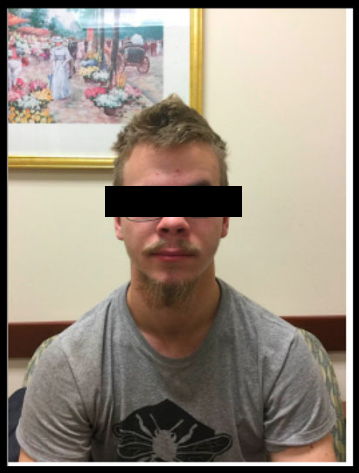
Rib resection – 6 month follow-up

RA (mmHg)	13	RA (mmHg)	7
PA (mmHg)	84/41, 55	PA (mmHg)	63/15, 31
PCWP (mmHg)	22	PCWP (mmHg)	15
CO (L/min)	2.97	CO (L/min)	5.8
PVR (WU)	11	PVR (WU)	2.75
Systemic BP (mmHg)	111/69	Systemic BP (mmHg)	134/71
PA sat	45%	PA sat	58%
Ao sat	92%	Ao sat	92%

48

Diagnosis



- Reviewed the case when Stephen Archer gave GR.
- He commented that his shoulders appeared raised.
  - *"Could this be Klippel-Feil Syndrome?"*
  - a rare congenital condition characterized by the abnormal fusion of any two of the cervical vertebrae.

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Genetic testing

- Heterozygous for a Variant in MYO18B.
- Pathogenic variants in MYO18B have been associated with autosomal recessive Klippel-Feil Syndrome 4, with myopathy and facial dysmorphism.

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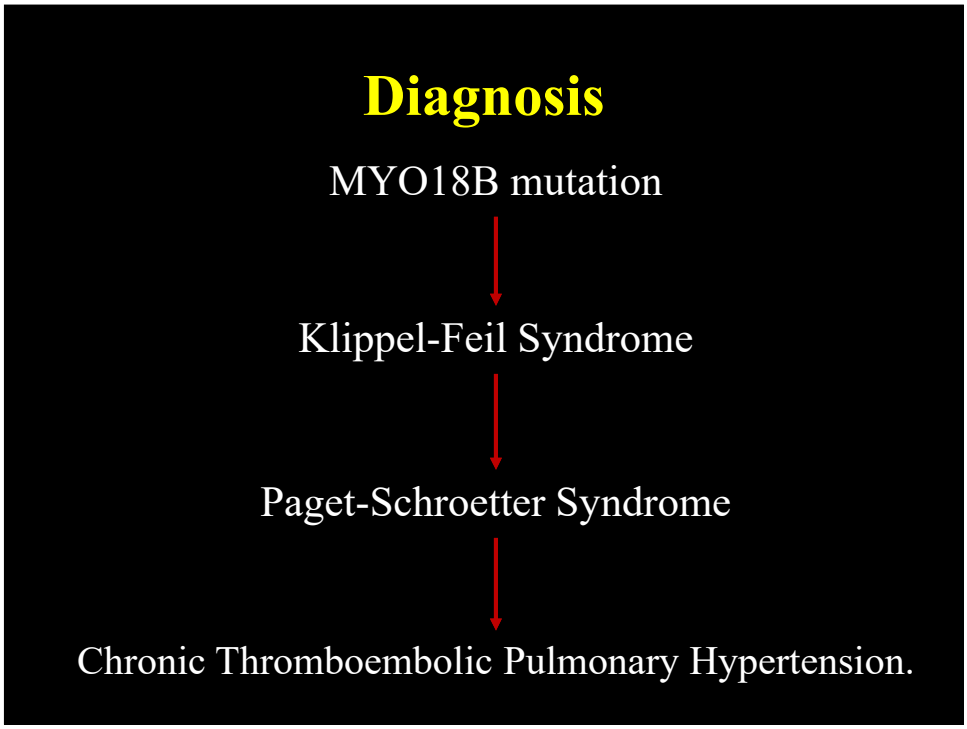
Diagnosis
MYO18B mutation
Klippel-Feil Syndrome
Paget-Schroetter Syndrome
Chronic Thromboembolic Pulmonary Hypertension.

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