Inferior vena cava obstruction and heart failure with preserved ejection fraction

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Disclosure

Speaker name:

Tim Sebastian

I have the following potential conflicts of interest to report:

- ☐ Consulting
- ☐ Employment in industry
- ☐ Stockholder of a healthcare company
- ☐ Owner of a healthcare company
- ☐ Other(s)

- ■ I do not have any potential conflict of interest

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Inferior vena cava obstruction

- Wide variability in clinical presentation
- Many reasons for IVC obstruction excist:
  - Caused by doctors: IVC disruption: Filters, clips, sutures); Umbilical catheters in neonates
  - Not caused by doctors: Congenital anomalies, tumors, fibrosis, idiopathic thrombosis
- 25-50% of cardiac output reach the heart through the IVC

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Cardiac Output Response to Exercise in Patients with Inferior Vena Caval Ligation

By Murray A. Varat, M.D., Noble O. Fowler, M.D., and Robert J. Adolph, M.D.

SUMMARY

Studies were carried out to determine whether hemodynamic alterations may follow inferior vena caval (IVC) ligation. Five patients, aged 29 to 49 years, who had been operated upon 2 to 5 years previously, and five normal controls, aged 39 to 44 years, were studied at rest and during graded submaximal treadmill exercise. There were no significant differences in heart rate, right atrial pressure, brachial arterial pressure, oxygen consumption, or cardiac output between the patients and normals in the supine rest and head-up tilt positions. During treadmill exercise the heart rate and oxygen consumption were comparable in the two groups, but the cardiac output response was definitely diminished in the patient group. This was reflected in the exercise factor, that is, the increase in cardiac output per 100 ml increase in oxygen consumption. The mean exercise factor was 406 ml for the patient group and 726 ml for the controls, a significant difference ($P < 0.001$).

It is concluded that the cardiac output response to upright exercise may be impaired in patients with IVC ligation. Inadequate venous return following IVC ligation may explain the persistent postoperative exertional dyspnea that has been noted in our patients.

CHEST

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Impaired Exercise Tolerance After Inferior Vena Caval Interruption

Todd D. Muller M.D., F.C.C.P. * , Bruce A. Staats M.D. *

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Four patients were evaluated for persistent dyspnea eight months to four years after inferior vena caval interruption for treatment of pulmonary emboli. Maximal exercise testing with gas-exchange analysis was performed. All patients attained less than 64 percent of predicted maximal oxygen uptake. Peak exercise heart rates were 85 percent or greater of predicted values. Arterial hypoxemia was not observed. The ratio of dead space to tidal volume (VD/VT) decreased with exercise, and the ratios of maximal exercise ventilation to maximal voluntary ventilation ($\dot{V}E$/MVV) were less than 67 percent. These results suggest a cardiac rather than a ventilatory limitation to exercise. Inadequate venous blood return to the heart is the likely mechanism for the impaired exercise performances.

„Cardiac output response to upright exercise may be impaired in patients with IVC ligation. Inadequate venous return may explain persistent postoperative exertional dyspnea.“

„Inadequate venous blood return to the heart is the likely mechanism for the impaired exercise performance.“

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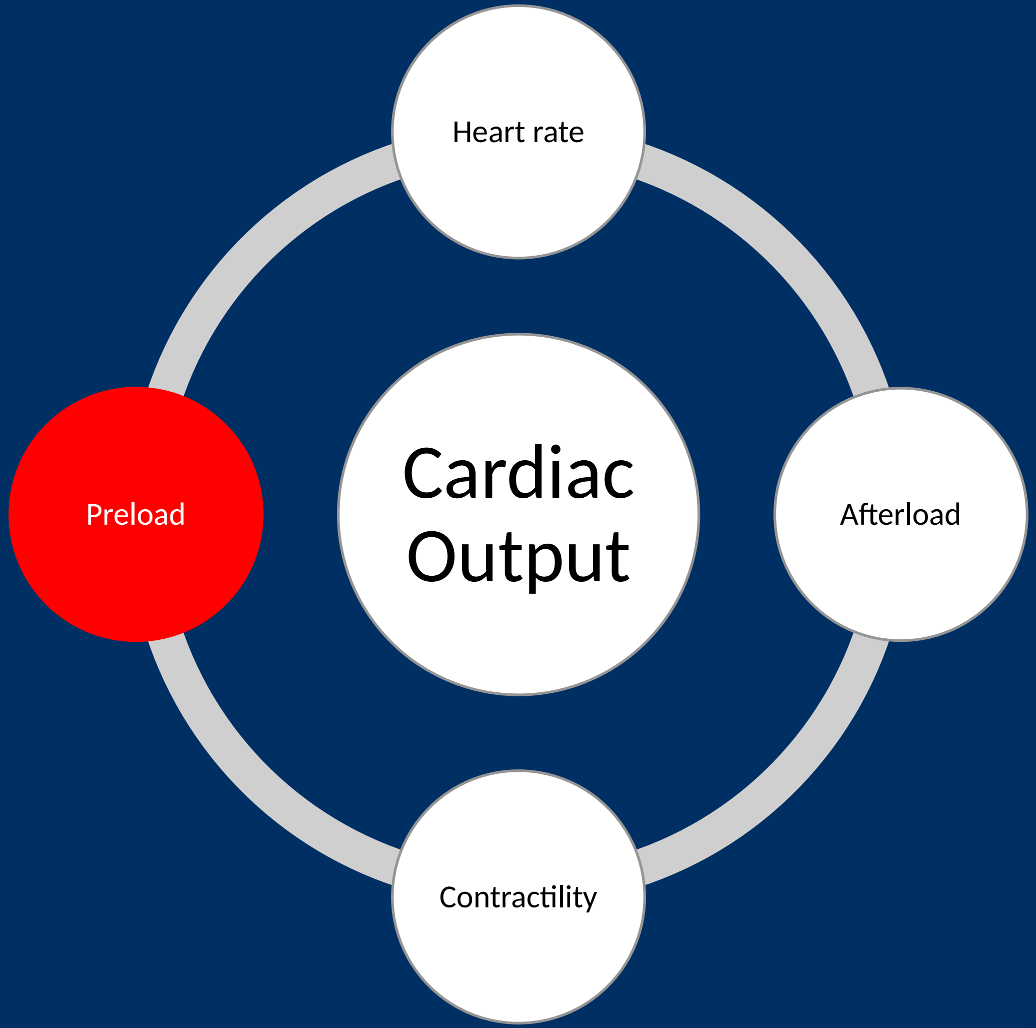
Is exertional dyspnea due to

IVC occlusion reversible?



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From the Swiss Venous Stent Registry: Inclusion criteria for a subgroup analysis

- Stent therapy for IVC obstruction with/without iliac vein involvement
- Left ventricular ejection fraction >50% in TTE.
- CPET (spiroergometry) before and after therapy

Patient characteristics	N=17
Mean age	45 ± 15 years
Female	29%
NYHA Stage II	53%
NYHA Stage III	18%
Procedural data	
Number of implanted venous stents, mean ± SD	4.1 ± 1.5
Proximal stent landing zone in -suprarenal IVC -infrarenal IVC, N (%)	53% 47%
Iliac kissing stents, N (%)	88%
Distal stent landing zone in -Common femoral vein	47%

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Baseline Test	
Sufficient subject effort during testing (1 criteria fulfilled: >85% HR, RER >1.1; Lactate >4.0 mmol/L	74%
Functional capacity (based on VO2 peak) Mild Impairment (70-84%) Moderate Impairment (50-69%)	 18% 59%

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Anaerobic threshold	Peak exercise
Time point at which metabolic acidosis and the associated changes in pulmonary gas exchange occur.	Time point of maximum ability of the cardiovascular system to deliver oxygen to exercising skeletal muscle

→ May better reflect changes in cardiac hemodynamics in patients with initially low exercise capacity

i) Does not require maximum subject effort (which may not be achieved due to leg symptoms)

ii) Improvement of peak exercise values obtained from CPET may not be related to improved cardiopulmonary function but to reduced leg symptoms

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CPET parameters at anerobic threshold	Before intervention	After intervention	Mean difference (95% CI)
Work rate, W	92.71 ± 28.83	107.35 ± 30.84	14.65 (-0.69; 29.98)
Heart rate, beats per minute	128.12 ± 26.46	119.41 ± 15.57	-8.71 (-18.02; 0.60)
Minute ventilation, L/min	36.98 ± 9.48	41.62 ± 7.45	4.64 (0.62; 8.66)
VO_2, mL/kg body weight	13.84 ± 4.71	15.64 ± 4.49	1.80 (0.32; 3.27)
- VO_2, percent of predicted	47.32 ± 13.08	54.42 ± 12.26	7.10 (2.07; 12.14)
- O_2 pulse, mL/beat	**9.25 ± 2.14**	**11.20 ± 2.52**	**1.95 (1.12; 2.78)**

O_2 pulse was used as a surrogate of cardiac output

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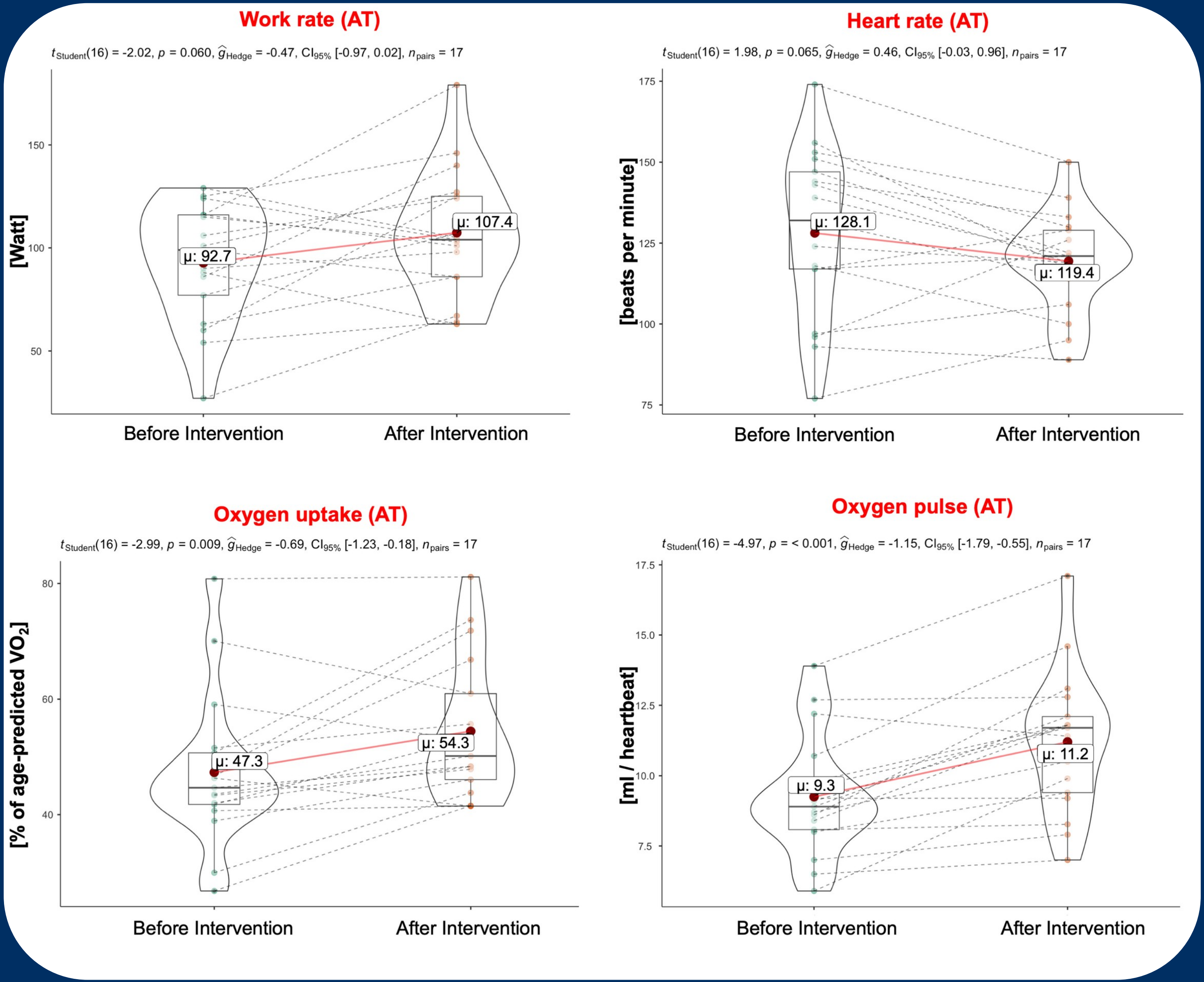

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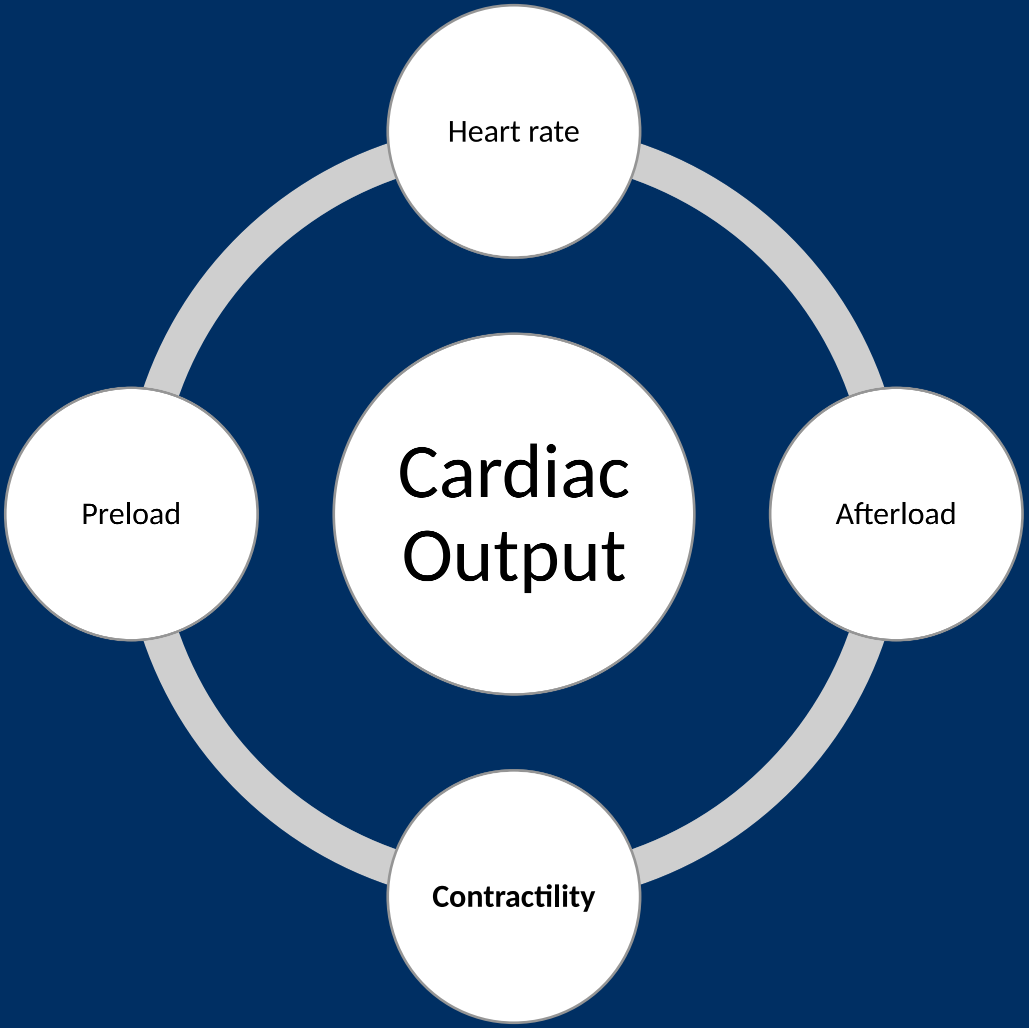
NYHA stage	Before	After
NYHA I	7	12
NYHA II	9	5
NYHA III	3	0
NYHA IV	0	0

Decrease in heart rate despite increased work load and oxygen uptake



No structural changes of the heart in TTE (aortic valve stenosis, ventricular dilatation or other signs of increased afterload)

Preserved LV-EF (TTE)

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