

Vaso-Reactivity Testing with Inhaled Nitric Oxide in a Patient with Pulmonary Hypertension Undergoing Cardiac Catheterization

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Submitted: 04 June 2017; Accepted: 12 June 2017; Published: 19 June 2017

Keywords: Pulmonary Hypertension (PH), Inhaled Nitric Oxide (iNO), Pulmonary Vaso-reactivity, Drug choices, Nitric Oxide in PH crises.

Background

Inhaled low-dose nitric oxide is a selective pulmonary vasodilator. It is used in the evaluation and treatment of persistent pulmonary hypertension of the newborn [1]. And therapeutically in acute pulmonary lung injury. [2]. We describe its delivery in an awake spontaneously breathing patient with PH during Right-sided Cardiac Catheterization. A positive response is helpful in choosing appropriate medical therapy and can be lifesaving in a PH crisis perioperatively.

Pulmonary arterial hypertension (PAH) is a disorder of the pulmonary arterioles that leads to right ventricular failure and death. It is a major risk factor for complications, morbidity and mortality during anaesthesia due to a PH crisis that can lead to a cardiac arrest. PH is defined as a mean pulmonary artery pressure (PAP) of > 25mmHg at rest and a PAP of >30mmHg when exercising.

The Nice classification recognizes five groups of pulmonary hypertension [4].

- Pulmonary arterial hypertension. (Category 1)
 - Pulmonary hypertension due to left heart disease.
 - Pulmonary hypertension due to lung disease.
 - Chronic thromboembolic pulmonary hypertension.
 - Pulmonary hypertension with multiple mechanisms.
- For a diagnostic algorithm for suspected PH see [3,4].

Case study

A fifteen-year-old school girl complained of increased shortness of breath and easy fatigue becoming progressively worse over a period of a few months. Her normal everyday activities became increasingly difficult and she now experienced chest discomfort and palpitations. There was no history of syncope. On clinical examination a palpable P2 was felt and a loud P2 was heard with signs of right ventricular enlargement. She was not in right-sided heart failure. Echocardiography measurements of pulmonary artery pressures were raised. To confirm PAH right heart catheterisation is mandatory as it remains the gold standard in defining PH.

Monitoring

Standard monitors include ECG, heart rate, blood pressure and oximetry. Pulmonary artery pressures, cardiac output and other

derived measurements are done during cardiac catheterisation. Minimal sedation given, Midazolam 2mg for anxiolysis. Baseline cardiac output measurements and pulmonary artery pressure were done at rest.

An anaesthetic ADE circuit connected to a Draeger Primus anaesthesia machine was used and the patient given Oxygen at room air FiO₂ (21%). Nitric oxide (NO) is then delivered via the inlet limb of the ADE system 10-12 cms from the face mask. Several delivery systems are available commercially and the Sidewinder delivery NO Box used. Rotameters control flow and concentrations can be measured in parts per million. The inhaled NO sampling (concentration) is integrated in the delivery system.

The circuit is primed with NO and with a face mask snugly fitted the patient encouraged to breathe deeply at fresh gas flows of 4-5 litres per minute aiming at NO concentration of 25 parts per million. Repeat measurements of PA pressure were done after 10 minutes of inhaled NO. Combinations of inhaled NO and higher FiO₂ provide added pulmonary vasodilation in persons with a reactive vascular bed [5].

Results

Baseline - Cardiac Output(CO) 3.04 (l/m),
PA mean Pressure. 53 (mmHg),
Pulm. Resistance. 14 (wood),
Pulm wedge Pressure 10.4 (mmHg).

NO 25 ppm + FiO₂ 21%, CO. 3.93(l/m), PA mean Pressure. 32(mmHg), Pulm.Resistance. 5.59(wood).

Responders vary between 5-15% of all patients. They have more than a 20% decrease in Pulmonary artery pressure or pulmonary vascular resistance. In addition CO should be unchanged and the pulmonary mean pressure <40mmHg [2,6]. Wedge pressure less than 16 mmHg will exclude L heart diseases.

Inhaled NO is an Odourless Gas

Issues related to ideal dosing and safety have been raised. However its effects are largely limited to the lungs. On diffusing into the blood it is rapidly inactivated by haemoglobin to form (metHb) methemoglobin and nitrate. Nitric dioxide (NO₂) a toxic metabolite is also formed. Significant metHb or NO₂ is uncommon in patients breathing NO at less than 80ppm [3].

Management Strategies:

Vaso-reactive patients benefit from calcium channel blockers (CCB). Its empirical use is discouraged due to the systemic effects of high dose CCB.

- Other classes of drugs available for non- reactive patients include [3,7].
- Endothelin receptor antagonists.(Sildenafil)
- Phosphodiesterase inhibitors. (PDE5 inhibitors).
- Prostacyclin analogs
- Cyclic guanosine monophosphate agonist.

Mono-therapy with 1 agent with less severe disease may benefit PH patients but those with more severe disease require combination therapy targeting different pathways of the pathophysiological process [8]. The new paradigm involves combined therapies that act on different pathways with huge implications for cost containment. Primary therapy also includes diuretics, digitalis and oxygen. Lung transplant with ECMO as a bridge are the remaining surgical option. Pulmonary Hypertension in the Perioperative period [2].

Inhaled No is Useful in Ameliorating Post-Operative Pulmonary Hypertension Following

Congenital Heart Disease and Cardiac and Lung Transplants. Inhaled NO is standard treatment in children peri operatively with pulmonary hypertension and can be administered through the breathing circuit beginning at induction. In long term use precautionary measures

include monitoring levels of NO and NO2, avoiding high levels of inspired FiO2 and measuring methemoglobin levels. Pulmonary Hypertension peri-operatively requires a planned approach as peri-operative complications occur with general anaesthesia or procedural sedation. The goal is to minimize PVR and depression of Right Ventricular Function. A balanced anaesthetic technique maintaining cardiac output recommended while avoiding systemic hypotension [8, 9].

Prevent increases in PVR by avoiding hypoxia, hypercarbia, pain, acidosis and hypothermia and treating impending pulmonary hypertensive crises. Life threatening PH crises requires aggressive management [5]. The delivery of 100% oxygen and iNO is life saving as is inotropic support with low dose Dopamine. Freissen et al reviews its management in detail [8].

Dexmedetomidine an alpha 2 agonist with properties of sympatholysis and sedation has with good hemodynamic monitoring been successfully used for procedural sedation and general anaesthesia in PH disease [10]. PAH is a debilitating illness that leads to early death. In high risk patients the 1 year mortality is greater than 10 %. [7].

Pre-operative testing requires cardiac catheterisation. Inhaled NO is a useful diagnostic tool to evaluate patients likely to benefit from CCD and NO perioperative is lifesaving in PAH. It is easily accessible and recommended in major theatre environments.

Name: LEBESE NOMPUMELELO

Id:

Case: 1008493175

NETCARE MILPARK HOSPITAL
CATH LAB

Condition Summary Report

10/05/2017 11:00 - 10/05/2017 11:37

0 / [11:00] PATIENT DETAILS

PATIENT NAME	LEBESE NOMPUMELELO	
CASE ID	1008493175*	
SEX	Female	
DATE OF BIRTH	02/09/2001	
WEIGHT	63.0*	[kg]
HEIGHT	162*	[cm]
HEMOGLOBIN	19.0	[gm%]
B.S.A	1.67*	[sqm]
HEART RATE	96	[bpm]
O2 CONS. EST	213*	[ml/min]


0 / [11:00] CONDITION: *****

26 / [11:26] LEFT VENTRICLE (REST)

HEART RATE	83	[bpm]
LV BDP	5	[mmhg]
LV EDP	10	[mmhg]
LV PEAK SYST	95	[mmhg]
LV MEAN SYST	64	[mmhg]
LV MEAN DIAS	7	[mmhg]
LV MAX DP/DT	1044	[mmhg/sec]
LV MIN DP/DT	-1239	[mmhg/sec]
LV PEAK VCE	29.4	[/sec]
LV V MAX	33.1	[/sec]

19 / [11:19] RIGHT VENTRICLE (REST)

HEART RATE	76	[bpm]
RV BDP	6	[mmhg]
RV EDP	10	[mmhg]
RV PEAK SYST	76	[mmhg]
RV MEAN SYST	50	[mmhg]
RV MEAN DIAS	8	[mmhg]
RV MAX DP/DT	795	[mmhg/sec]
RV MIN DP/DT	-732	[mmhg/sec]
RV PEAK VCE	28.7	[/sec]
RV V MAX	38.2	[/sec]



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GEMS
DR : PG WILLIAMS

Netcare Milpark Hospital
PH: 100517308
CASE NO: **1008493175**
NOMPUMELELO MASEGO
HTEL: 0731582271
ADM DATE: 09.05.2017
WARD: Section 1
I.D.: 0109020765086 AGE: 15 yrs 8 mt
GENDER: F DOB: 02.09.2001
PLAN: EMERALD
DEP: 02 PAR:
PR: 1809246
MEM NO.: 001157308

18 / [11:17] RIGHT ATRIUM (REST)

HEART RATE	79	[bpm]
RA MEAN PRESSURE	8.4	[mmhg]
RA A-WAVE	11.4	[mmhg]
RA V-WAVE	9.6	[mmhg]
RA X-WAVE	6.4	[mmhg]
RA Y-WAVE	7.0	[mmhg]

21 / [11:21] PULMONARY WEDGE (REST)

HEART RATE	79	[bpm]
PW MEAN PRESSURE	10.4	[mmhg]
PW A-WAVE	11.3	[mmhg]
PW V-WAVE	13.9	[mmhg]
PW X-WAVE	6.7	[mmhg]
PW Y-WAVE	9.1	[mmhg]

27 / [11:27] AORTA (REST)

HEART RATE	75	[bpm]
AO PEAK SYST	96	[mmhg]
AO MIN DIAS	68	[mmhg]
AO MEAN PRESSURE	81	[mmhg]

29 / [11:29] PULMONARY ARTERY (REST)

PA MEAN PRESSURE	53.0	[mmhg]
T.P.R	17.41	[wood]
T.P.R INDEX	29.10	[wood m2]

29 / [11:29] MITRAL VALVE (REST)

MIV DFP	28	[sec/min]
MIV VALVE FLOW	107	[ml/sec]
MIV MEAN GRAD	2.9	[mmhg]
MIV VALVE AREA	1.66	[sq cm]
MIV AREA INDEX	0.99	[sq cm/sq m]

29 / [11:29] PULMONARY RESISTANCE (REST)

MEAN GRAD	42.6	[mmhg]
PUL RESISTANCE	14.00	[wood]
PUL RESISTANCE INDEX	23.39	[wood m2]

29 / [11:29] SYSTEMIC RESISTANCE (REST)

MEAN GRAD	72.3	[mmhg]
SYS RESISTANCE	23.77	[wood]

Continued...

29 / [11:29] SYSTEMIC RESISTANCE (REST) (Continue)

SYS RESISTANCE INDEX	39.72	[wood m2]
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29 / [11:29] CARDIAC OUTPUT FICK (REST)

HEART RATE	80	[bpm]
HEMOGLOBIN	19.0	[gm%]
O2 CONS. EST	213	[ml/min]
PA SAT	64.5	[%]
ART. SAT	92.0	[%]
CO	3.04	[l/min]
CI	1.82	[l/min/sqm]
SV	38	[ml/bt]
SI	22.76	[ml/bt/sqm]

28 / [11:28] OXIMETRY (REST)

HEMOGLOBIN	19.0	[gm%]							
O2 CAPACITY CONSTANT	1.34	[ml/g]							
O2 CAPACITY	25.5	[vol%]							
PA	64.5	[sat%]	16.4	[vol%]	[mmhg]	[mmhg]
LV	92.0	[sat%]	23.4	[vol%]	[mmhg]	[mmhg]
Used PV	92.0	[sat%]	23.4	[vol%]	[mmhg]	[mmhg]
Used SA	92.0	[sat%]	23.4	[vol%]	[mmhg]	[mmhg]
Used MV	64.5	[sat%]	16.4	[vol%]	[mmhg]	[mmhg]
Used PA	64.5	[sat%]	16.4	[vol%]	[mmhg]	[mmhg]
SBF	3.0	[l/min]	1.82	[l/min/sqm]					
PBF	3.0	[l/min]	1.82	[l/min/sqm]					
EPBF	3.0	[l/min]	1.82	[l/min/sqm]					
QP/QS	1.0								

30 / [11:30] CONDITION: *****

$$F_{iO_2} (21\%) + NO \ 25PPM.$$

34 / [11:33] AORTA (POST OXY 1)

HEART RATE	68	[bpm]
AO PEAK SYST	109	[mmhg]
AO MIN DIAS	73	[mmhg]
AO MEAN PRESSURE	89	[mmhg]

38 / [11:37] PULMONARY ARTERY (POST OXY 1)

PA MEAN PRESSURE	32.4	[mmhg]
T.P.R	8.24	[wood]
T.P.R INDEX	13.77	[wood m2]

38 / [11:37] MITRAL VALVE (POST OXY 1)

MIV DFP	28	[sec/min]
MIV VALVE FLOW	139	[ml/sec]
MIV MEAN GRAD	2.9	[mmhg]
MIV VALVE AREA	2.15	[sq cm]
MIV AREA INDEX	1.28	[sq cm/sq m]

38 / [11:37] PULMONARY RESISTANCE (POST OXY 1)

MEAN GRAD	22.0	[mmhg]
PUL RESISTANCE	5.59	[wood]
PUL RESISTANCE INDEX	9.35	[wood m2]

38 / [11:37] SYSTEMIC RESISTANCE (POST OXY 1)

MEAN GRAD	80.8	[mmhg]
SYS RESISTANCE	20.57	[wood]
SYS RESISTANCE INDEX	34.38	[wood m2]

38 / [11:37] CARDIAC OUTPUT FICK (POST OXY 1)

HEART RATE	74	[bpm]
HEMOGLOBIN	19.0	[gm%]
O2 CONS. EST	213	[ml/min]
PA SAT	70.2	[%]
ART. SAT	91.5	[%]
CO	3.93	[l/min]
CI	2.35	[l/min/sqm]
SV	53	[ml/bt]
SI	31.76	[ml/bt/sqm]

37 / [11:36] OXIMETRY (POST OXY 1)

HEMOGLOBIN	19.0	[gm%]							
O2 CAPACITY CONSTANT	1.34	[ml/g]							
O2 CAPACITY	25.5	[vol%]							
PA	70.2	[sat%]	17.9	[vol%]	[mmhg]	[mmhg]
AO	91.5	[sat%]	23.3	[vol%]	[mmhg]	[mmhg]
Used PV	91.5	[sat%]	23.3	[vol%]	[mmhg]	[mmhg]
Used SA	91.5	[sat%]	23.3	[vol%]	[mmhg]	[mmhg]
Used MV	70.2	[sat%]	17.9	[vol%]	[mmhg]	[mmhg]
Used PA	70.2	[sat%]	17.9	[vol%]	[mmhg]	[mmhg]
SBF	3.9	[l/min]	2.35	[l/min/sqm]					
PBF	3.9	[l/min]	2.35	[l/min/sqm]					
EPBF	3.9	[l/min]	2.35	[l/min/sqm]					
QP/QS	1.0								

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