



The Relationship Between the Components of Pulmonary Artery Pressure Remains Constant Under All Conditions in Both Health and Disease*

Raheel Syeed, MB; John T. Reeves, MD; David Welsh, PhD; David Raeside, MD; Martin K. Johnson, MD; and Andrew J. Peacock, MD

Background: The relationships among systolic pulmonary artery pressure (SPAP), diastolic pulmonary artery pressure (DPAP), and mean pulmonary artery pressure (MPAP) have been found to be constant in humans breathing air, at rest, while supine. It would be important for those studying the pulmonary circulation if this relationship were maintained under other circumstances, such as change in posture, during exercise, or after pharmacologic manipulation. In particular, it would be useful if the relationship were maintained when treating pulmonary hypertension because this would allow different methods of measurement to be compared, such as SPAP from echocardiography and MPAP from right heart catheterization.

Methods: Data were reviewed from both healthy subjects and those with pulmonary hypertension ($n = 65$) who had a micromanometer-tipped, high-fidelity pulmonary artery catheter inserted for between 6 and 36 h in the Scottish Pulmonary Vascular Unit between 1997 and 2003. The 5-min averages, while the patient was supine at rest, were analyzed by linear regression to compare the response of SPAP and DPAP with MPAP.

Results: There were linear relationships (measured in millimeters of mercury) of SPAP with MPAP ($\text{SPAP} = 1.50 \text{ MPAP} + 0.46$), and DPAP with MPAP ($\text{DPAP} = 0.71 \text{ MPAP} - 0.66$). These were maintained with a high degree of accuracy following changes in posture and activity.

Conclusions: SPAP, MPAP, and DPAP were strongly related, and these relationships were maintained under varying conditions. This finding will allow comparison between invasive and noninvasive descriptions of pulmonary hemodynamics found in the literature.

(CHEST 2008; 133:633–639)

Key words: ambulatory monitoring; pulmonary artery pressure; pulmonary hypertension

Abbreviations: CTEPH = chronic thromboembolic pulmonary hypertension; DPAP = diastolic pulmonary artery pressure; MPAP = mean pulmonary artery pressure; SPAP = systolic pulmonary artery pressure

Understanding the relationships among the components of pulmonary artery pressure could be important for investigators studying the lung circulation, and for clinicians managing patients with pulmonary hypertension. For example, when investigators measure systolic pressures from the velocity of the tricuspid regurgitant jet using Doppler echocardiography, they report only right ventricular systolic pressure, which relates closely to systolic pulmonary artery pressure (SPAP). Investigators measuring pulmonary artery pressure directly at heart catheterization usually report only the mean pulmonary artery pressure (MPAP). As a result, integration of the

two bodies of literature has been difficult because the relationship of MPAP to SPAP has not been known for different patient populations.

Two studies^{1,2} have demonstrated strong corre-

For editorial comment see page 592

lations among SPAP, diastolic pulmonary artery pressure (DPAP), and MPAP in supine subjects at rest during right heart catheterization. Chemla et al¹ summarized their data by the following equation:

$$\text{MPAP} = 0.61 \text{ SPAP} + 2 \text{ mm Hg.}$$

This finding has been demonstrated in both adults and children, and across a range of different causes of pulmonary hypertension, including arterial, venous, and chronic thromboembolic causes. It is not known whether the relationships found with the patient in the supine position and at rest also hold in other circumstances. The aim of this study was to assess the relevance of the predictive equations derived with patients at rest to measurements of pulmonary artery pressure when hemodynamics were altered by changes in posture, during exercise, or following the administration of medication to vasodilate the pulmonary arteries.

MATERIALS AND METHODS

Study Design and Subjects

This was a retrospective analysis of high-fidelity digital pulmonary artery pressure data previously acquired from an indwelling micromanometer-tipped catheter in patients undergoing clinical evaluation at the Scottish Pulmonary Vascular Unit.^{3,4} Data collected between 1997 and 2003 from 65 patients are presented (37 women and 28 men; mean [\pm SD] age, 59.1 \pm 13.4 years) [Table 1]. The pressures given in Table 1 were point values measured at the time of insertion of the catheter. In this subject cohort, 12 patients were healthy, and the other 53 patients were categorized into eight different etiologic classes. The catheter remained in the pulmonary artery from 6 to 36 h, allowing the recording of pressure during the patient's usual activities. Ethical approval was provided by the West Ethics Committee, North Glasgow NHS University Trust. All subjects gave informed consent for ambulatory pressure measurements as part of their clinical evaluation for suspected pulmonary hypertension.

Pressure Recording System

The right heart catheter (Gaeltec Ltd; Dunvegan, Isle of Skye, UK), and recording system, has been described in detail elsewhere.⁵ Briefly, it is a 7.5F, solid, micromanometer-tipped catheter with a bandwidth frequency of 0 to 1000 Hz. The pressure transducer is located at the tip of the catheter, making

Table 1—Details of Patients Studied by Ambulatory Micromanometer-Tipped Pulmonary Artery Catheter*

Patient No.	Diagnosis	Age, yr	Gender	SPAP/DPAP, mm Hg	MPAP, mm Hg
1	Normal	55	F	16/5	10
2	Normal	67	F	20/7	11
3	Normal	32	F	18/8	12
4	Normal	62	M	19/7	12
5	Normal	55	F	20/8	12
6	Normal	60	F	20/8	12
7	Normal	66	M	23/11	15
8	Normal	56	M	23/13	17
9	Normal	71	F	27/12	17
10	Normal	37	F	25/13	18
11	Normal	65	F	28/14	20
12	Normal	40	F	25/13	18
13	CHD	41	F	110/57	77
14	COPD	65	F	27/12	19
15	COPD	67	M	36/20	26
16	COPD	66	F	54/18	31
17	COPD	67	F	66/28	42
18	COPD	81	F	73/27	45
19	COPD	66	F	73/31	47
20	COPD	58	M	64/36	47
21	COPD	61	F	79/35	52
22	COPD	65	M	81/41	55
23	CTD	71	F	21/8	13
24	CTD	68	F	42/14	23
25	CTD	65	F	34/19	25
26	CTD	62	F	39/18	26
27	CTD	50	M	46/16	27
28	CTD	56	M	54/22	33
29	CTD	69	F	54/26	36
30	CTD	66	F	61/24	37
31	CTD	40	F	56/35	42
32	CTD	62	F	63/36	46
33	CTD	78	F	76/35	51
34	CTD	60	M	79/36	51
35	CTD	65	F	80/38	54
36	CTD	63	M	96/36	54
37	CTD	41	F	124/52	78
38	CTD	46	F	74/32	48
39	CTEPH	74	M	34/18	24
40	CTEPH	78	M	80/31	49
41	CTEPH	75	M	68/37	49
42	CTEPH	51	M	86/35	53
43	CTEPH	71	M	85/33	55
44	CTEPH	60	F	85/45	59
45	CTEPH	69	M	94/47	63
46	ILD	72	M	57/23	36
47	ILD	59	F	71/30	45
48	ILD	34	F	75/47	57
49	ILD	49	F	102/49	69
50	PoPH	48	F	22/11	17
51	PoPH	31	M	54/24	36
52	PoPH	30	M	70/30	45
53	PVH	74	M	52/19	30
54	PVH	55	F	66/33	46
55	PVH	75	M	76/37	52
56	IPAH	54	F	58/30	40
57	IPAH	59	F	31/18	23
58	IPAH	24	M	70/37	48
59	IPAH	77	M	78/36	51

(Continued)

*From the Scottish Pulmonary Vascular Unit (Mr. Syeed, and Drs. Welsh, Johnson, and Peacock), Western Infirmary, Glasgow, Scotland, UK; the Department of Pediatrics (Dr. Reeves), University of Colorado Health Sciences Center, Denver, CO; and the Department of Respiratory Medicine (Dr. Raeside), Victoria Infirmary, Glasgow, Scotland, UK.

Mr. Syeed and Dr. Raeside were supported by the National Services Division, Scottish Executive, Scotland.

The authors have reported to the ACCP that no significant conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Manuscript received June 3, 2007; revision accepted October 5, 2007.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml).

Correspondence to: Andrew J. Peacock, MD, Scottish Pulmonary Vascular Unit, Western Infirmary, Dumbarton Rd, Glasgow G11 6NT, Scotland, UK; e-mail: a.peacock@udcf.gla.ac.uk

DOI: 10.1378/chest.07-1367

Download English Version:

<https://daneshyari.com/en/article/2903890>

Download Persian Version:

<https://daneshyari.com/article/2903890>

[Daneshyari.com](https://daneshyari.com)