Mechanisms of Cough Syncope as Evaluated by Valsalva Maneuver

A-Ching Chao,[1,2] Ruey-Tay Lin,[1,2] Ching-Kuan Liu,[1,2] Pao-Yu Wang,[3] and Hung-Yi Hsu[3]
Department of Neurology, [1]Faculty of Medicine, College of Medicine, Kaohsiung Medical University, [2]Kaohsiung Medical University Hospital, Kaohsiung, and [3]Section of Neurology, Taichung Veterans General Hospital, Taichung, Taiwan.

Successful treatment of cough syncope depends on the correction of various pathogenetic mechanisms among different patients. The valsalva maneuver (VM), which elicits hemodynamic responses mimicking coughs, has potential for investigating the individual pathogenesis of cough syncope. Eighteen consecutive patients suffering from cough-induced syncope were examined. All patients were asked to cough and to perform VM several times under continuous cerebral blood-flow velocity and blood pressure (BP) monitoring by transcranial Doppler and finger plethysmography. Eight patients demonstrated abnormal VM characterized by the absent BP overshoot following the relief of straining. Patients demonstrating abnormal VM had delayed BP recovery after cough (median, 16.4; range, 8.7–25.6 seconds) compared to those demonstrating normal VM (2.6, 1.3–3.8 seconds, $p < 0.001$). Seven of the 10 patients exhibiting normal BP overshoot during VM had stenotic arterial lesions in the cerebral or coronary circulation, whereas only one of the eight patients demonstrating absent BP overshoot had coronary artery disease (70% *vs*. 12.5%, $p = 0.025$). Other clinical profiles, body mass index, frequency of obstructive pulmonary disease and valsalva ratio did not differ between patients featuring normal and absent BP overshoot. In conclusion, the pathogenesis of cough syncope could be different between patients with normal and abnormal VM responses. Patients who had no BP overshoot during VM sustained prolonged hypotension after cough. The VM helps in discriminating among pathogenic mechanisms and guiding investigation and treatment for cough syncope patients.

Key Words: cough syncope, hypotension, intrathoracic pressure, valsalva maneuver
(*Kaohsiung J Med Sci* 2007;23:55–62)

Cough syncope is a conspicuous syndrome. Clinically, the fainting fit is always associated with a paroxysm of cough, often occurring after a few vigorous coughs. Cough syncope may result from various etiologies, such as profound arterial hypotension [1–3], cerebral hypoperfusion secondary to the corresponding increase in intracranial pressure [4–6], pulmonary vasoconstriction and hypoxemia [7], or even reflex cardiac arrhythmia [8]. Although the syndrome is usually benign, fatalities have been reported for 1–2% of such patients [9]. No specific treatment has yet been demonstrated to be universally effective. The management of cough syncope can be difficult if individual pathologic perturbations are not clearly understood, although there does not appear to be any clear consensus regarding the diagnostic workup for patients suffering from cough-induced syncope. Coughing is comparable to an exaggerated valsalva maneuver (VM) because both conditions elicit an increase in intrathoracic and intracranial pressures and a decrease in venous return [5]. The VM, which elicits hemodynamic responses similar to those produced by coughing, is a suitable test for investigating individual pathogenic mechanisms for patients suffering from cough syncope. However,

Received: May 17, 2006 Accepted: August 15, 2006
Address correspondence and reprint requests to: Dr Hung-Yi Hsu, Section of Neurology, Taichung Veterans General Hospital, 160, Section 3, Taichung-Kang Road, Taichung 407, Taiwan.
E-mail: 2941@vghtc.gov.tw

VM-induced hemodynamic changes have only been sparsely investigated in cough syncope patients in the early literature [5,7]. This study was undertaken to determine the usefulness of conducting standardized VM in elucidating the pathophysiologic mechanisms of cough syncope.

Patients and Methods

From January 1998 to June 2002 inclusively, 427 consecutive patients suffering from syncope were examined by an organized team consisting of cardiologists, neurologists, and experienced sonographers to evaluate the cause of syncope systemically. Eighteen male patients who experienced attacks of only syncope immediately after coughs were enrolled in this study prospectively. A detailed medical history was obtained from all study participants. All participants underwent detailed physical and neurologic examinations and blood evaluation including a complete blood cell count, blood chemical analyses, fasting plasma sugar, and arterial blood gas analysis. Chest radiography, spirometry, electrocardiography (ECG), echocardiography, awake electroencephalography (EEG), Duplex ultrasonography of the cervical vessels, and transcranial color-coded sonography were also performed in all participants. Significant extracranial or intracranial arterial stenosis was diagnosed according to the validated ultrasonographic criteria used in our laboratory. Twenty-four-hour holter ECG and neuroimaging studies were performed when clinically indicated.

Patients abstained from consuming alcohol-, caffeine-, or nicotine-containing products for the 10- to 12-hour period prior to undergoing further studies on hemodynamics. Medication that could interfere with the testing of autonomic function was also withheld for at least five half-lives. All measurements were performed in a temperature-controlled room with the subjects in the supine position, and were conducted in the morning, at least 2 hours subsequent to the patient having eaten a light breakfast. Transcranial Doppler ultrasonography (Multidop X; DWL Electronische System GmbH, Sipplingen, Germany) was used to monitor the blood-flow velocity of the middle cerebral artery (MCAFV) by using the usual method. Noninvasive servo-controlled plethysmography (Portapres, TNO-BMI, Amsterdam, The Netherlands) was used to measure the beat-to-beat blood pressure (BP).

First, patients were asked to cough forcefully to obtain two similar BP responses. Two reproducible VMs were performed by blowing into a mouthpiece and maintaining a pressure of 40 mmHg for a period of 15 seconds, according to methods described elsewhere [10,11]. Subsequent to performing VM, all patients were positioned in a passive, head-up tilt to 70° upright position for a period of 30 minutes to rule out the possibility of neurocardiogenic syncope or postural hypotension. There was a rest of at least 5 minutes separating each test. The institutional review board at this hospital approved the study protocol. All eligible subjects signed an informed consent form prior to their being enrolled in the study.

Data analysis

The analog signal of BP and that representing the peak velocity envelope of MCAFV were fed via an analog-to-digital converter (BNC2070 and 6020E, National Instruments, Austin, TX, USA) at a sampling rate of 200 Hz to an IBM-compatible computer, and saved on the hard disc for later analysis. Responses to the VM were divided into four main phases by the respective maximal and minimal values of BP and MCAFV as proposed by previous investigators [11–13]. The proportional changes of mean BP and mean MCAFV during each phase of the VM were estimated from the baseline values and were used for analytical purposes. We estimated recovery latencies for both MCAFV and BP, which Ferrer et al [14] used in 1991 as surrogate markers of autonomic disorder. The time periods from the trough value for phase III to corresponding baseline value (baseline latency) and the peak (overshoot latency) of phase IV were calculated (Figure 1). The time period required for BP to recover to baseline value after cough was also calculated for each individual (Figure 1C). The valsalva ratio was extracted from the maximum heart rate generated by VM divided by the lowest heart rate occurring within 30 seconds of peak heart rate [10,11].

Continuous data were expressed as median and range. The χ^2 test was used for evaluating categorical variables, and the Fisher exact test for instances in which individual counts were <5. A nonparametric Mann–Whitney U test was used to compare the difference between patients featuring a normal and those who exhibited a delayed BP overshoot.

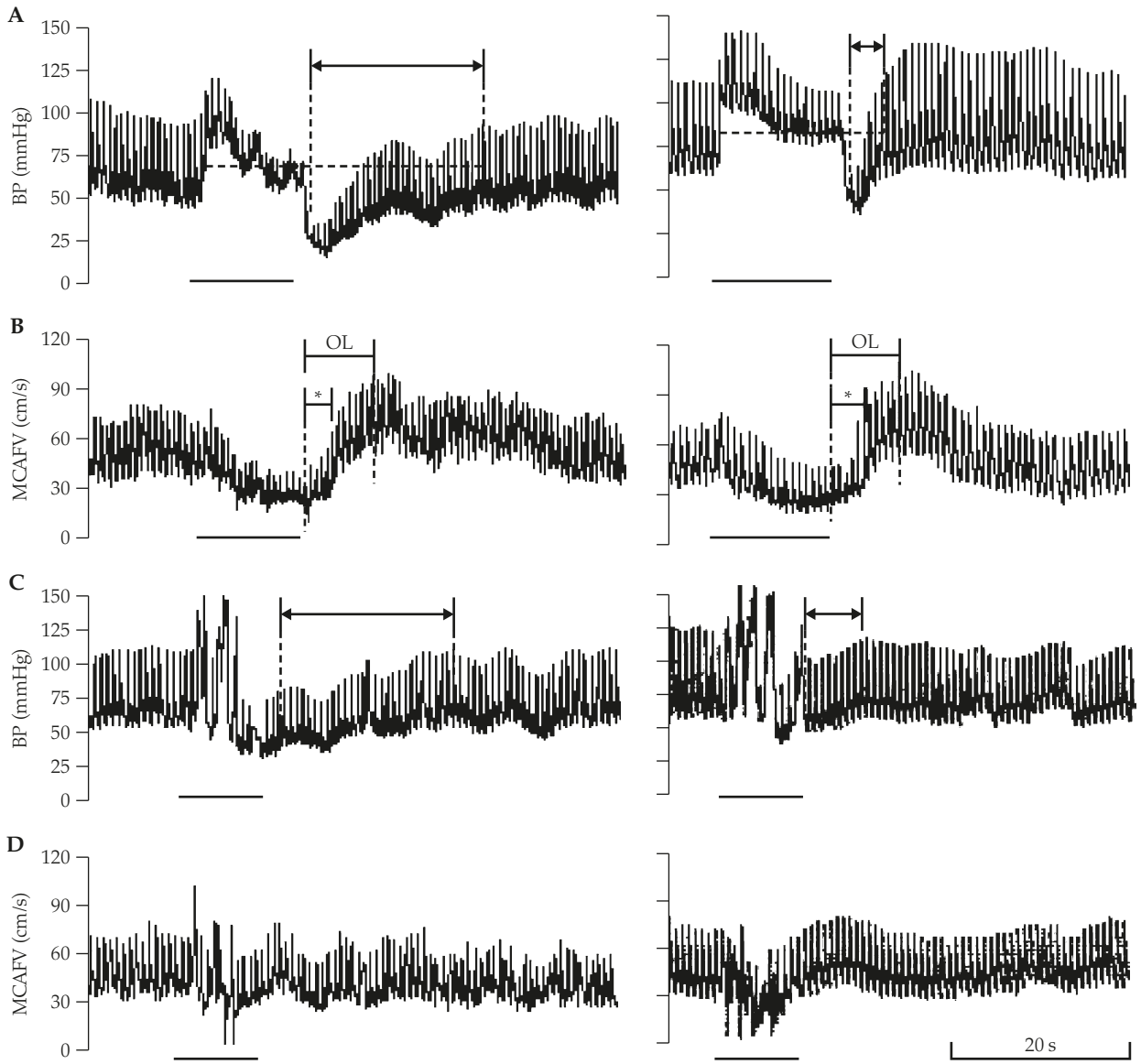

Figure 1. *Characteristic changes in blood pressure (BP) (A, C) and blood-flow velocity of middle cerebral artery (MCAFV; B, D) induced by the valsalva maneuver (VM; A, B) and by cough (C, D). Horizontal lines indicate periods of straining during VM (A, B) or cough (C, D). The left panels demonstrate the absence of BP overshoot during VM in a representative patient from group-1 patients, and the right panels show corresponding normal responses in a patient from group-2 patients (A). The time required for BP to return to baseline levels is prolonged for the group-1 patient following VM and coughing (indicated by "↔") as compared to its group-2 analog (A, C). Baseline latency (indicated by "∗") and overshoot latencies of MCAFV are similar between the two groups (B, D).*

Results

The clinical profiles of these patients are summarized in the Table. Eight patients demonstrated an abnormal VM-induced response that was characterized by absent BP overshoot following the relief of straining associated with the VM (Figure 1A, left panels). These patients were classified as group-1 patients. The other 10 patients featuring a normal BP overshoot during VM were classified as group-2 patients (Figure 1A, right panels). Age and body mass index were not different between groups ($p = 0.725$ and 0.705, respectively). Baseline BP value latencies were significantly prolonged for group-1 patients (8.7, 5.2–18.9 seconds) compared to group-2 patients (2.6, 1.3–3.8 seconds; $p < 0.001$). Group-1 patients had delayed BP recovery

Table. Summary of the clinical profiles and medical histories of study patients

Patient/ age (yr)	BMI	Medical history	COPD	CAD	Holter ECG	Echocardiography	Brain imaging	Neurosonology/ tilting table test
Group 1								
1/80	24.5	Normal	No	No	Neg	Neg	Neg	Neg
2/73	27.1	HTN	Yes	No	Neg	Neg	ND	Neg
3/80	21.5	Normal	Yes	No	Neg	Neg	Neg	Neg
4/76	22.5	Normal	No	No	Neg	Neg	Neg	Neg
5/80	26.4	Normal	Yes	No	ND	Neg	Neg	Neg, TCD-PW
6/76	22.8	Normal	Yes	No	ND	Neg	Neg	Neg
7/60	29.3	DM	No	Yes	ND	1/3 septal hypokinesia	Neg	Orthostatic hypotension
8/45	25.9	Normal	No	No	ND	Neg	Neg	Neg
Group 2								
9/76	25.4	HTN	No	Yes	ND	LVH	R watershed infarct	R MCA stenosis
10/76	24.9	HTN	Yes	Yes	ND	Inferior wall hypokinesia	Neg	Neg
11/67	22.8	Normal	Yes	Yes	Neg	Neg	Neg	L MCA stenosis
12/79	25.6	Normal	Yes	Yes	ND	Neg	ND	Neg, TCD-PW
13/81	27.5	Normal	No	No	ND	Neg	Neg	Neg
14/74	22.6	HTN	No	Yes	ND	Neg	Neg	Neg
15/69	25.2	DM	Yes	No	ND	Neg	Neg	Neg
16/72	24.2	Normal	Yes	No	ND	Neg	ND	Neg, TCD-PW
17/67	27.4	Normal	No	No	Neg	Neg	L hemispheric infarct	R vertebral, L ICA stenosis
18/67	28.6	Normal	No	No	Neg	Neg	Multiple lacunar infarcts	R ICA stenosis

BMI = body mass index; HTN = hypertension; DM = diabetes mellitus; COPD = chronic obstructive pulmonary disease, as demonstrated by spirometry; CAD = coronary artery disease, as demonstrated by coronary angiography; ECG = electrocardiography; Neg = normal or negative findings; LVH = left ventricular hypertrophy; ND = not done; R = right; L = left; TCD-PW = poor acoustic windows for transcranial Doppler; MCA = middle cerebral artery; ICA = internal carotid artery.

after cough (Figure 1C) compared to that of group-2 patients (16.4, 8.7–25.6 seconds *vs*. 4.3, 3.6–6.4 seconds; $p < 0.001$). The patterns of BP recovery after cough were quite similar to that observed after the relief of straining during VM for each individual (Figure 1). Doppler flow signals could not be detected for three patients due to the presence of poor temporal acoustic windows. The baseline and overshoot latencies of MCAFV, however, did not differ between group-1 and group-2 patients, with, respectively, baseline latencies of 1.8 seconds (1.2–3.6) versus 1.3 seconds (0.9–3.5; $p = 0.132$) and overshoot latencies of 4.1 seconds (2.9–6.1) versus 3.2 seconds (1.7–5.2; $p = 0.183$). The mean BP values of group-1 patients were 89.6 (60.7–101.5), 105.3 (74.3–126.3), 71.3 (47.7–92.8), 75.5 (47.0–98.3), 29.6 (15.7–50.6), and 82.4 mmHg (56.7–92.0) for baseline, phase I, phase IIa, phase IIb, phase III, and phase IV, respectively. The mean BP values of group-2 patients were 74.7 (61.5–101.5), 93.4 (71.7–168.3), 67.6 (53.8–115.7), 82.8 (58.6–123.3), 54.5 (33.7–99.0), and 93.9 mmHg (70.0–134.7) for the corresponding phases. The proportional changes in mean BP and mean MCAFV induced by VM are summarized in Figure 2.

The valsalva ratio was not different between group-1 (1.28, 1.05–1.69) and group-2 patients (1.48, 1.09–1.75; $p = 0.46$). None of the studied patients sustained a syncope or presyncope throughout the whole period of examination. Among all patients, none had significant abnormality on awake EEG recording. The frequency of obstructive pulmonary disease as diagnosed by spirometry was 50% for members of both groups. Seven of the patients from group-2 (70%) exhibited overt cardiac or cerebral arterial disease(s), and only one patient from group-1 (12.5%, $p = 0.025$) had coronary artery disease diagnosed by coronary angiography.

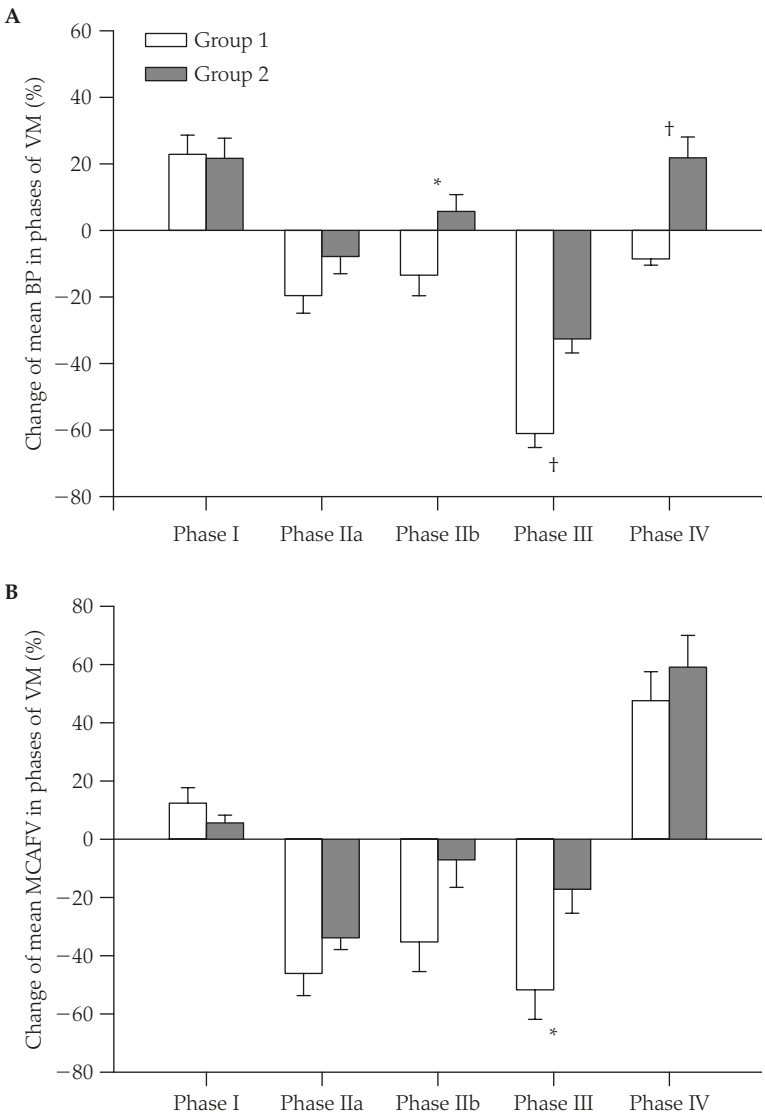

Figure 2. *Percentage changes in: (A) mean blood pressure (BP); and (B) mean blood-flow velocity of the middle cerebral artery (MCAFV) that occurred during different phases of the valsalva maneuver (VM). Group-1 patients demonstrate absent BP overshoot during phase IV of VM. Group-2 patients demonstrate normal responses during VM. *$p < 0.05$; †$p < 0.01$.*

Discussion

Our study demonstrated that a certain number of cough syncope patients revealed an abnormal VM as characterized by absent BP overshoot after the relief of straining. The absent BP overshoot was related to delayed BP recovery after a VM and a cough. Patients with absent BP overshoot also sustained prolonged hypotension subsequent to a cough, as illustrated in Figure 1C. The hypotension after paroxysmal vigorous cough of daily life should be more profound and cause syncope in patients with absent BP overshoot during VM. Our findings suggested that VM provided valuable information about differential hemodynamic responses among patients with cough syncope.

Absence of BP overshoot in response to VM in group-1 patient is an uncommon finding. Absence of BP overshoot seemed exceptional in healthy elderly subjects according to normative data of VM from a previous study [15]. Absent BP overshoot that occurs during VM has been presumed to arise as a consequence of sympathetic adrenergic dysfunction [10,12,14]. However, the majority of our study patients revealed no evidence of the presence of any sympathetic failure. Although subtle autonomic dysfunction cannot be totally excluded by completing a patient's

clinical history and by their demonstrating normal BP responses to passive head-up tilt testing, such subtle autonomic dysfunction is unlikely to cause absent BP overshoot during VM [10,12,14].

Previous investigators have described an "emphysema response" characterized by a delay in the occurrence of phase IV during VM for patients suffering from obstructive pulmonary disease [16]. This emphysema response would appear to be similar to the absent BP overshoot observed in our patients. Zema et al [17] demonstrated absent overshoot during VM in eight of 37 patients with chronic obstructive pulmonary disease. However, the emphysema response is not related to the severity of pulmonary disease and could be observed in patients with normal pulmonary function. Patients with mild congestive heart failure could have a loss of the overshoot in BP during phase IV of VM [13,17]. Only two of our 18 patients had abnormal ventricular wall motion detected by echocardiography. Absence of BP overshoot during VM should result from complex interactions among sympathetic regulation of cardiac contractibility and vasomotor tone, cardiac function, and pulmonary condition. Additional factors such as the extent of change in pleural pressure [18,19], low blood volume in alveolar vessels and intrathoracic cavity [18,20], and cardiac compression by pulmonary overdistention [21,22] might also be involved in the absence of BP overshoot for group-1 patients.

Patients with delayed BP recovery had a greater BP reduction during phases IIb and III of VM. Pulmonary blood volume, which serves as a reservoir to maintain left ventricular filling, helps to maintain BP during the straining phase of VM [23]. The greater BP reduction during phase IIb in spite of compensated tachycardia in group-1 patients might partly result from decreased pulmonary blood volume. During phase III of VM, decreased venous return to left side heart would be accentuated when intravascular volume decreases [23] and blood volume in alveolar vessels and intrathoracic cavity is low [18,19]. The accentuated BP reduction during phase III in group-1 patients suggested that more time was required for the increase in venous return to circulate through the pulmonary vasculature. The delayed filling of left side heart turned out to be the delayed BP recovery in phase IV. Typically, cough syncope occurs among overweight patients suffering from chronic pulmonary diseases, and, reportedly, most often occurs during or soon after a heavy meal [9]. Cough syncope occurs predominantly in male [9,24]. Patients with emphysema have lower pulmonary capillary blood volume [25]. A heavy meal produces redistribution of more blood in splanchnic circulation and lower blood volume in pulmonary circulation. Accordingly, low pulmonary blood volume should contribute to the occurrence of cough syncope in group-1 patients.

The valsalva ratio was not different between the two groups of patients in this study. The greater BP reduction during straining accentuated sympathetic activation and cardioacceleration in group-1 patients. The greater increase in heart rate during the straining phase resulted in normalization of the valsalva ratio in group-1 patients, despite there being no reflex bradycardia in the absence of BP overshoot. Previous investigators also demonstrated that the valsalva ratio is not a selective test of cardiovagal function [26].

The pathogenesis of cough syncope among patients featuring normal responses to VM could be different from that of patients with delayed BP recovery during VM. The frequencies of coronary heart disease or cerebral arterial stenotic lesions were greater for group-2 patients than for group-1 patients. The aforementioned arterial stenotic lesions might accentuate cerebral or cardiac ischemia during coughing for these so-affected group-2 patients [27]. Elevation of central venous and intracranial pressures might contribute to a marked reduction in cerebral perfusion during coughing for cough syncope patients who feature a normal BP overshoot [5,6]. However, our data cannot be interpreted to address this point. The relative amplitudes and latencies of the MCAFV overshoot did not differ between our two study groups. Cerebrovascular autoregulation seemed competent to maintain cerebral perfusion for all of our patients despite different BP responses.

Some limitations of this study should be mentioned. First, coughs and VM were performed in the supine position due to safety considerations. Cough syncope is less likely to occur in the supine position. BP reduction during a cough should be greater in the standing position compared to that in the supine position because of more intravascular volume pooling in lower limbs and more reduction in venous return [28]. Second, we were unable to provide invasive hemodynamic data such as central venous pressure, cardiac stroke volume or pleural pressure during VM,

and coughs. Third, our sample size was rather small given that cough syncope is a relatively uncommon condition. Further studies among more patients are warranted prior to the general application of our results.

In summary, VM may help in discriminating among pathogenic mechanisms of cough syncope, whereas it would appear likely that a patient's physical condition and medical history would fail to do so. Absence of BP overshoot during VM suggested that cough syncope resulted from prolongation of hypotension after coughs. For such individuals, attempts to keep adequate intravascular volume, to treat pulmonary disease, and to avoid heavy meals could help to reduce or eliminate syncopal attacks during coughing. On the other hand, patients who exhibited normal BP responses to VM may need to undergo additional evaluations to rule out other possible etiologies for cough syncope, such as stenosis or occlusion of the cerebral arteries or the presence of some form of organic heart disease.

References

1. Pedersen A, Sandoe E, Hvidberg E, et al. Studies on the mechanism of tussive syncope. *Acta Med Scand* 1966; 179:653–61.
2. Sharpey-Schafer EP. The mechanism of syncope after coughing. *Br Med J* 1953;4841:860–3.
3. Benditt DG, Samniah N, Pham S, et al. Effect of cough on heart rate and blood pressure in patients with "cough syncope". *Heart Rhythm* 2005;2:807–13.
4. Mattle HP, Nirkko AC, Baumgartner RW, et al. Transient cerebral circulatory arrest coincides with fainting in cough syncope. *Neurology* 1995;45:498–501.
5. McIntosh HD, Estes EH, Warren JV. The mechanism of cough syncope. *Am Heart J* 1956;52:70–82.
6. Desser KB, Harris CL, Benchimol A. Carotid blood velocity during cough studies in man. *Stroke* 1976;7: 416–8.
7. McCann WS, Bruce RA. Tussive syncope; observations on the disease formerly called laryngeal epilepsy, with report of two cases. *Arch Med Interna* 1949;84:845–56. [Illustration]
8. Lee D, Beldner S, Pollaro F, et al. Cough-induced heart block. *Pacing Clin Electrophysiol* 1999;22:1270–1.
9. Kerr A Jr, Derbes VJ. The syndrome of cough syncope. *Ann Intern Med* 1953;39:1240–53.
10. Low PA. Autonomic nervous system function. *J Clin Neurophysiol* 1993;10:14–27.
11. Low PA. Laboratory evaluation of autonomic function. In: Low PA, ed. *Clinical Autonomic Disorders: Evaluation and Management*. Philadelphia: Lippincott-Raven, 1997: 179–208.
12. Porth CJ, Bamrah VS, Tristani FE, et al. The valsalva maneuver: mechanisms and clinical implications. *Heart Lung* 1984;13:507–18.
13. Nishimura RA, Tajik AJ. The valsalva maneuver and response revisited. *Mayo Clin Proc* 1986;61:211–7.
14. Ferrer MT, Kennedy WR, Sahinen F. Baroreflexes in patients with diabetes mellitus. *Neurology* 1991;41: 1462–6.
15. Denq JC, O'Brien PC, Low PA. Normative data on phases of the valsalva maneuver. *J Clin Neurophysiol* 1998;15:535–40.
16. Mills H, Kattus AA Jr. The emphysema response to forced straining (valsalva's maneuver). *Circulation* 1958;17:65–71.
17. Zema MJ, Masters AP, Margouleff D. Dyspnea: the heart or the lungs? Differentiation at bedside by use of the simple valsalva maneuver. *Chest* 1984;85:59–64.
18. Hausknecht MJ, Wise RA, Brower RG, et al. Effects of lung inflation on blood flow during cardiopulmonary resuscitation in the canine isolated heart–lung preparation. *Circ Res* 1986;59:676–83.
19. Martin J, Jardim J, Sampson M, et al. Factors influencing pulsus paradoxus in asthma. *Chest* 1981;80:543–9.
20. Obermiller T, Lakshminarayan S, Willoughby S, et al. Influence of lung volume and alveolar pressure on reverse pulmonary venous blood flow. *J Appl Physiol* 1992;73:195–9.
21. Scharf SM. Cardiovascular effects of airways obstruction. *Lung* 1991;169:1–23.
22. Santamore WP, Heckman JL, Bove AA. Right and left ventricular pressure–volume response to respiratory maneuvers. *J Appl Physiol* 1984;57:1520–7.
23. Little WC, Barr WK, Crawford MH. Altered effect of the valsalva maneuver on left ventricular volume in patients with cardiomyopathy. *Circulation* 1985;71: 227–33.
24. Bonekat HW, Miles RM, Staats BA. Smoking and cough syncope: follow-up in 45 cases. *Int J Addict* 1987;22: 413–9.
25. Morrison NJ, Abboud RT, Muller NL, et al. Pulmonary capillary blood volume in emphysema. *Am Rev Respir Dis* 1990;141:53–61.
26. Sandroni P, Benarroch EE, Low PA. Pharmacological dissection of components of the valsalva maneuver in adrenergic failure. *J Appl Physiol* 1991;71:1563–7.
27. Linzer M, McFarland TA, Belkin M, et al. Critical carotid and vertebral arterial occlusive disease and cough syncope. *Stroke* 1992;23:1017–20.
28. Singer W, OpferGgehrking TL, McPhee BR, et al. Influence of posture on the valsalva manoeuvre. *Clin Sci (Lond)* 2001;100:433–40.

利用 Valsalva Maneuver 來研究咳嗽昏厥之致病機轉

趙雅琴[1,2]　林瑞泰[1,2]　劉景寬[1,2]　王寶玉[3]　許弘毅[3]

[1]高雄醫學大學　醫學院醫學系　內科學

[2]高雄醫學大學附設醫院　神經內科

[3]台中榮民總醫院　神經內科

欲成功地治療「咳嗽昏厥」則必先了解及處理該類病人之不同的致病機轉。Valsalva maneuver 所引起的血流動力學改變，基本上極似咳嗽所引起的變化，因此也可用作研究咳嗽昏厥致病機轉的方法。本研究納入因咳嗽而昏厥之病患，共 18 位進入研究，在以穿顱超音波及非侵入性紅外線手指血壓計 (servo-controlled infrared finger plethysmography) 連續監控紀錄血壓及腦血流之下，要求患者作 valsalva maneuver 及咳嗽數次，結果 8 位患者在鬆開壓力時並沒有正常所見之血壓回衝 (overshoot) 現象。這些病人在要求作咳嗽時，其血壓下降後回復期與 valsalva maneuver 正常者相較也有意義的延長 (median, 16.4; range, 8.7－25.6 秒比 median, 2.6; range, 1.3—3.8 秒，$p < 0.001$)。在 valsalva maneuver 正常者 10 人中有 7 人有腦血管或心血管狹窄，在 valsalva maneuver 異常者 8 人 (即血壓無回衝者)，只有 1 人有腦血管或心血管狹窄 (70% *vs.* 12.5%，$p = 0.025$)。其餘如臨床表徵，body mass index，阻塞性肺病之機率和 valsalva ratio 則在二組病人中無有差異。總結，患者在 valsalva maneuver 時無有血壓回衝者，也同時呈現咳嗽後血壓下降延遲恢復之現象。因此 valsalva maneuver 可以幫助區分咳嗽昏厥之病理機轉，進而可以成為一研究及治療患者之方法。

關鍵詞：咳嗽昏厥，低血壓，胸內壓，valsalva maneuver

(高雄醫誌 2007;23:55－62)

收文日期：95 年 5 月 17 日

接受刊載：95 年 8 月 15 日

通訊作者：許弘毅醫師

台中榮民總醫院神經內科

台中市407中港路三段160號