They suggested that alveolar gas can dissect proximally along the bronchovesicular bundle into the mediastinum, then inferiorly into the retroperitoneal space and into the vascular compartment below the diaphragm via the perirenal, splanchnic, or subcutaneous veins.[2] The findings of mesenteric gas and pulmonary arterial gas in the patient reported here are compatible with this proposed mechanism.

Our patient had evidence of both venous and arterial gas embolism. Large amounts of venous gas can overwhelm the lung's ability to filter the gas[6] and can produce an arterial gas embolism (paradoxical embolism) in the absence of a communication between the right and left sides of the heart. Once the gas reaches the arterial circulation, cerebral infarction, as well as coronary artery occlusion, may occur.

The pathophysiology of gas embolism includes right atrial and right ventricular distention, pulmonary artery obstruction, impaired gas exchange due to intrapulmonary shunting, production of vasoactive mediators, and activation of neutrophils. Oxygen radicals may contribute to the lung injury.[7]

This patient did not die with the initial event that we believe involved venous gas embolism; that event occurred the day following randomization when she precipitously became hemodynamically unstable and sustained a right ventricular infarction. We suspect venous embolism produced partial obstruction of the pulmonary artery with foam. This, we believe, caused increased pulmonary arterial pressures and increased pulmonary vascular resistance and contributed to a right ventricular infarction due to increased right ventricular afterload and a decreased cardiac output. Arterial gas embolism clearly occurred the following day, manifested by brain death. Alternatively, arterial embolism may have been the cause of the right ventricular infarction. This possibility seems less likely, however, because there was no evidence of left ventricular infarction with the initial event.

We were perplexed by the pulmonary arterial gas found at necropsy until the article by Bricker et al[2] was published (our case occurred in 1991). Their discovery of inferior venal caval gas in ARDS patients supported with PPV provides an explanation for the pulmonary arterial gas in our patient.

In conclusion, this case report provides antemortem and postmortem evidence of venous and arterial gas embolism as a consequence of PPV. We do not know the frequency of such problems, but data suggest that the frequency of venous and arterial gas embolism is higher than previously suspected.[2]

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Elevation of Peak Expiratory Flow by a "Spitting" Maneuver*

Measured With Five Peak Flowmeters

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Study objective: **To determine if peak expiratory flow (PEF) is higher using incorrect technique versus correct technique with five marketed peak flowmeters.**

Design: **Randomized, nonblinded study.**

Setting: **University pulmonary medicine clinic.**

Patients: **Twenty adults with clinically stable asthma.**

Interventions: **After inhaling 2 puffs of albuterol via a valved aerosol holding chamber (Aerochamber), patients were instructed over the next 15 min in correct and incorrect (a "spitting" action) technique when using peak flowmeters. Order of use of five peak flowmeters and correct vs incorrect technique was random.**

Measurements and results: **PEF (percentage of personal best) was recorded for best of three attempts with correct and incorrect technique. Each peak flowmeter had a statistically significant elevation in PEF with incorrect technique. The range for elevation in PEF using incorrect technique was 12.4 to 68.2% above the PEF with the subject using correct technique.**

Conclusion: **Each of the five marketed peak flowmeters had a significant elevation in PEF when a "spitting action" was used. Clinicians need to instruct patients carefully regarding correct technique when using peak flowmeters.**

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Key words: asthma; peak expiratory flow; peak flowmeters

Abbreviations: PEF=peak expiratory flow

Home monitoring of peak expiratory flow (PEF) is a basic component of the National Institutes of Health guidelines[1] as well as the Global Initiative[2] guidelines for management of asthma. Since colored-zone management[2] with PEF can be helpful to patients and clinicians in making decisions regarding appropriate action, accurate measurements are of obvious importance. "Green zone" is ≥80% of personal best; "yellow zone," 50-79%; and "red zone," <50%.[1] Connolly[3] reported elevation in PEF (*ie*, >30% increase over PEF when using correct technique) due to accelerating the airflow from the mouth in an adult patient who used a "spitting action" with the tongue and buccal musculature. To our knowledge, there are no studies to verify if several commonly used peak flowmeters will give elevation in PEF due to acceleration in the mouth.

Methods

We enrolled 20 clinically stable adult patients aged 25 to 50 years with moderate persistent or severe persistent asthma who were receiving inhaled corticosteroid therapy.[2] After giving informed consent for this Institutional Review Board-approved study, patients were allowed to enter the study only if the following criteria were met: their PEF was now >80% of personal best (set within the past year), their need for albuterol was <4 times daily for the past week, and they had no emergency department visits or hospitalizations for the past 4 weeks. Patients were recruited through the private practice of pulmonologists at the university as well as from the university student body or employees. Patients with chronic bronchitis or emphysema, clinically significant cardiac disease or other pulmonary disease, and psychoses were excluded. In addition, patients who were unable to use peak flowmeters correctly or to perform the "spitting action" despite instructions were excluded.

After a medical history was taken and a brief physical examination was performed, each patient received albuterol, 2 puffs via the Aerochamber (Monaghan Medical Corp; Plattsburgh, NY). The study was performed after bronchodilator use to minimize risk that repeated performance of PEF could trigger bronchospasm. Over the next 15 min, patients were instructed regarding incorrect technique via the "spitting" maneuver, with the mouthpiece not placed well into the mouth. Although each patient was already familiar with correct technique, the investigators reviewed correct use of peak flowmeters again.[2] Instruction for both correct and incorrect techniques included demonstrations by the investigators. While verbal instruction of correct and incorrect techniques was provided, demonstrations of these techniques were emphasized. In addition, patients were coached during the maneuvers by the investigators.

Fifteen minutes after albuterol administration, patients performed PEF using 5 peak flowmeters (TruZone, Personal Best, Assess, MiniWright, and PocketPeak; see Table 1) in random order. Order of using correct vs incorrect technique also was random. The best of three attempts was recorded for correct technique[2] and for incorrect use.[3]

For each subject, device, and technique combination, percentage of change for personal best was computed as [(PEF-BEST)/BEST) · 100%]). Using three-way analysis of variance, differences among the devices for the percentage of change from personal best for the correct and incorrect techniques were compared. The percentage of elevation was the difference between the percentage changes from personal best and was the computed value from the incorrect technique minus the value from the correct technique. Using two-way analysis of variance for a complete block design (*ie*, with patients serving as blocks), the means for the percentage of elevation using the incorrect technique were tested against the null hypothesis that the percentage of elevation equals 0%. A sample size of 20 individuals was sufficient to detect a difference of 1 SD on a two-sided test at a significance level of 0.05 and a power of 0.8.

Results

When used incorrectly, each peak flowmeter tested had a significant elevation in PEF (Table 1). While each peak flowmeter had a statistically significant increase in PEF with incorrect use, the MiniWright had the least elevation in PEF. Results were the same when data were analyzed for actual PEF (L/min). Among these 20 patients, there was a wide range of PEF values (290 to 850 L/min; median, 490 L/min) using the correct technique.

Comment

Patients who accelerate air in the mouth as described by Connolly[3] and as found in this study could have clinically relevant problems in management. For example, a patient may truly be in the "red zone" (<50% of personal best) and need immediate medical attention and not receive it because incorrect use of the peak flowmeter gave a value in the "green zone" (≥80% of personal best) or "yellow zone" (50-79% of personal best). Even with the peak flowmeter that had the least elevation in PEF with incorrect technique (*ie*, MiniWright), a >10% elevation could move a patient from one zone to another, thus being clinically significant.

Why one peak flowmeter rather than another is more susceptible to the marked elevation in PEF has not been studied. One may speculate that because the MiniWright brand has a diaphragm, it has less of a rise in PEF with incorrect technique. In support of that possibility, one may note that the PocketPeak also has a diaphragm, and it had the second lowest percentage elevation in PEF. The other three instruments tested do not have a diaphragm. On the other hand, any of the

Table 1—*Peak Expiratory Flow (Percentage of Personal Best)

Peak Flowmeter	PEF (Correct)	PEF (Incorrect)	% False Rise	p Value (n=20)
Assess†	105.8±12.9	174.0±39.3	68.2±33.7	0.0001
MiniWright‡	102.9±11.9	115.4±15.8	12.4±9.3	0.0161
PocketPeak§	97.1±10.5	121.0±15.7	23.8±13.8	0.0001
Personal Best¶	99.8±11.5	143.3±28.2	43.4±24.4	0.0001
TruZone‖	113.7±28.5	156.4±41.9	42.6±38.7	0.0001

*Mean±SD. Standard errors from 3-way and 2-way analysis of variance were 3.5 and 5.0, respectively.
†Assess; HealthScan Products, Inc; Cedar Grove, NJ.
‡MiniWright; Clement Clarke Ltd; Essex, UK.
§PocketPeak; Ferarris Medical, Inc; Holland, NY.
¶Personal Best; HealthScan Products, Inc; Cedar Grove, NJ.
‖TruZone; Monaghan Medical Corp; Plattsburgh, NY.

peak flowmeters tested is absolutely acceptable for use clinically as long as correct technique is used by the patient and each instrument may be susceptible to significant elevation with use of the incorrect technique. The reason that PEF is inappropriately high as a result of the "spitting" maneuver is apparently as Connolly[3] described it; he believes that the incorrect maneuver accelerates the airflow through the mouth, with the subject using the tongue and buccal musculature.

Other investigators have shown that patients will lie to clinicians about their PEF values.[4] Some patients want to please their clinicians by reporting values obtained either by manipulating the peak flowmeter via incorrect technique (one of our patients admitted to doing this prior to the study) or by recording untrue values. Thus, regardless of which peak flowmeter is used, clinicians need to instruct patients carefully regarding correct use,[2] proper recording of PEF, and avoidance of acceleration in the mouth (*ie*, the "spitting" maneuver). Emphasizing maximum expiratory effort with the mouthpiece well into the mouth, overlaying the anterior part of the tongue, prevents acceleration of airflow in the mouth.

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ARDS and Adrenal Insufficiency Associated With the Antiphospholipid Antibody Syndrome*

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The antiphospholipid antibody syndrome (APS) is typically characterized by recurrent arterial and/or venous thromboses, miscarriages, and thrombocytopenia. There have been five reported cases of ARDS associated with primary APS. Adrenal insufficiency has also been reported as a rare complication of APS. We report a case of both ARDS and adrenal insufficiency associated with primary APS. Chest radiographs and pulmonary angiography studies revealed findings consistent with ARDS. CT scans confirmed the presence of bilateral adrenal hemorrhagic infarction. Patients with APS are at an increased risk of widespread vascular thrombosis due to the presence of circulating antiphospholipid antibodies. This case emphasizes the importance of recognizing APS and its potential for multiple organ system involvement.

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Key words: adrenal insufficiency; antiphospholipid antibodies; antiphospholipid antibody syndrome; ARDS

Abbreviations: ACTH=adrenocorticotropic hormone; AI=adrenal insufficiency; APS=antiphospholipid antibody syndrome; APTT=activated partial thromboplastin time; PT=prothrombin time

A syndrome of recurrent thrombosis associated with a biological false-positive test for syphilis was reported by Johannson and colleagues[1] in 1976; but only over the past decade has the association of recurrent vascular thromboses, fetal loss, and thrombocytopenia, combined with persistently elevated antiphospholipid antibodies, been designated as the antiphospholipid antibody syndrome (APS).[2] APS is considered to be primary in the absence of connective tissue diseases and secondary in their presence; the most common connective tissue disease is systemic lupus erythematosus.[3] The rising awareness of APS, along with its potential for multiple organ system involvement mediated by vascular thromboembolic events, has made its recognition increasingly important. Pulmonary manifestations associated with APS are well documented, but only five cases have been reported of patients with both APS and ARDS.[4,5] Adrenal insufficiency (AI) has also been reported as a rare complication of APS.[6] We report a case of both ARDS and AI associated with primary APS.

Case Report

A 33-year-old white woman with known lupus anticoagulant and Hashimoto's hypothyroidism presented to the hospital's emergency department with a 5-day history of nausea, vomiting, diffuse abdominal pain, and generalized joint pain. Her symptoms started while she was vacationing in Europe. Her medication consisted of levothyroxine and an aspirin a day. Her past medical history included testing positive for antithyroid microsomal and antithyroglobulin antibodies. Physical examination revealed the presence of tachycardia (131 beats/min), a macular violaceous discoloration of her distal phalanges, and diffuse abdominal tenderness. A leukocyte count of 11,400/μL with 80% neutrophils, 7% lymphocytes, and 9% monocytes, a hemoglobin of 10.9 g/dL, a hematocrit of 32.3%, and a platelet count of 68,000/μL were noted. The activated partial thromboplastin time (APTT) was 92.8 s with a prothrombin time (PT) of 13.3 s. Chest radiographs and abdominal series on admission were negative. Lupus anticoagulant and antiphospholipid antibodies in the form of antiphosphatidyl serine IgG antibodies were detected. Along with the thrombocytopenia, the criteria for APS were established.

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