present arrhythmia as pseudosinoatrial block. In cases where a long pause is apparently considered as sinoatrial block, the ST segment and the T wave of the preceding sinus beat must be compared with other T waves and carefully scrutinized for even the slightest aberration. In such circumstances the diagnosis of blocked PACs should be suspected when a patient presents with apparent sinus bradycardia but also shows PACs elsewhere in the ECG. Pseudosinoatrial block is in itself not an indication for permanent pacing; however, correct diagnosis is unmistakable with intracardiac recordings.

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Platypnea in the Intensive Care Unit*

A Newly Described Cause
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Platypnea in a patient with COPD developed during the subacute onset of an ileus. Arterial blood gas studies failed to document orthodeoxia. Routine treatment for COPD failed to resolve the patient's positional dyspnea, but the dyspnea rapidly resolved following resolution of the ileus. The authors postulate that impaired abdominal muscle contraction in the upright position secondary to the ileus was responsible for the development of platypnea.

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Dyspnea in the upright position that is relieved by assuming the supine position has been described in patients with severe COPD and has been termed "platypnea." Platypnea has also been described in patients with a history of a right-to-left intracardiac shunt, true vascular lung shunts, pneumonectomy or following recovery from adult respiratory distress syndrome. Without these obvious described processes and particularly in the intensive care unit, severe positional dyspnea may provide a therapeutic dilemma.

We present the case of a patient with a history of COPD but no history of positional dyspnea who presented with an acute onset of profound platypnea. None of the above-mentioned causes of platypnea were identified, and only after the diagnosis and treatment of a slowly developing adynamic ileus did her symptom of dyspnea in the upright position resolve.

CASE REPORT

A 73-year-old white woman with oxygen-dependent COPD (FEV1 = 0.54 L) presented with a 5-day history of upper respiratory tract symptoms and increasing shortness of breath. She had mild left ventricular dysfunction, hypertension, and hypothyroidism. Her medications on admission included theophylline, iopratropium bromide, albuterol, and levophedrine sodium (Synthroid, Boots Pharmaceuticals).

The patient presented in moderate to marked respiratory distress with obvious use of her accessory muscles. Her oral temperature was 38.2°C, respiratory rate was 24 breaths/min, pulse was 140 beats/min, and blood pressure was 180/60 mm Hg. Jugular venous distention was absent. There were decreased breath sounds throughout the lung fields and diffuse expiratory wheezes. Cardiac examination revealed a tachycardic regular rhythm and distant heart sounds. Bowel sounds were present. The rest of the physical examination findings were normal. The arterial blood gas analysis revealed a pH of 7.23, a PaCO2 of 54 mm Hg, and a PaO2 of 73 mm Hg on 3 L/min of oxygen delivered by nasal cannula. The chest roentgenogram revealed increased bronchial markings but no cardiomegaly, infiltrates, or effusions.

The patient was initially treated with corticosteroids, aminophylline, antibiotics, beta2-agonists, and iopratropium bromide. Initial improvement occurred in the first 24 h, but it soon became readily apparent that she suffered from marked positional dyspnea. Any movement to a position greater than 45° from supine resulted in tachycardia, tachypnea, and severe dyspnea. She began complaining of constipation, anorexia, and intermittent nausea, which was initially thought to be secondary to a high-normal theophylline level. An echocardiogram, to look for right-to-left shunting, did not reveal any obvious atrial septal defect. To document the presence or absence of orthodeoxia, arterial blood gas samples were obtained with the patient supine, at 45°, and upright. There was no orthodeoxia accompanying her platypnea, but she was unable to tolerate sitting in the upright position for more than 3 min.

The patient's symptoms of anorexia, nausea, and constipation became more intense, and were subsequently accompanied by abdominal pain with tenderness to palpation. A flat plate film of the abdomen revealed a dilated transverse colon and small bowel. A nasogastric tube was inserted to treat the adynamic ileus, which in retrospect had probably been present to some degree since the second day of her hospitalization. Approximately 2,500 ml of fluid was removed in the next 12 h. Her abdominal complaints resolved and, more significantly, her platypnea ceased.

DISCUSSION

Platypnea in a patient with severe COPD was first described by Altman and Robin. They hypothesized that the combination of increased alveolar pressure and lower pulmonary artery pressure in the upper lung zones during

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the upright position produce regions that act as respiratory
dead space (diffuse zone I phenomenon). Michel et al.,
reporting on a patient with COPD and platypnea, felt that
the platypnea was related to severe hypoxia as a result of
poor regional ventilation-perfusion matching, which was a
consequence of a poor hypoxic vascular adaptive mecha-
nism.

Our patient certainly had severe COPD, but no previous
symptoms of platypnea. The patient's positional dyspnea
seemed to worsen as the ileus progressed, and it was not
until we treated the ileus that the platypnea dramatically
resolved. Because the ileus was not readily apparent initially,
and because the platypnea was so new in onset, we
attempted to rule out other causes of platypnea. Although a
tilt-table two-dimensional echocardiogram with peripheral
venous contrast medium was not performed to rule out a
right-to-left shunt,4 no obvious shunting was noted on routine
echocardiography nor did orthodeoxia occur with the pla-
ypnea. Another cause of platypnea, true vascular lung
shunts secondary to chronic liver disease or congenital
arteriovenous fistulas, has also been described,5 but our
patient had no evidence of either.

Besides the aforementioned hypothesized explanations
for platypnea in COPD patients, Sharp6 has also proposed
that the supine position in COPD patients allows the fluid
contents of the abdomen to push the diaphragm cephalad,
which would allow the diaphragm to assume a more favorable
position on its length-tension curve and thus assume a larger
percentage of the work of ventilation. Furthermore, active
abdominal muscle contraction during expiration has been
shown in COPD patients, and proposed as a mechanism by
which the diaphragm is pushed cephalad to improve the
length-tension status of the muscle.7 Also, Tobin8 notes that
elastic energy stored in abdominal muscles during expiration
may aid in the subsequent inspiratory effort. One could
postulate then that our patient, when at baseline health (ie,
without an ileus), was able to use abdominal muscle contrac-
tion in the upright position to improve her ventilation by
the mechanisms just described, and thus had no symptoms
of platypnea. However, with the onset of an ileus and
subsequent abdominal pain and distension, this abdominal
muscle contraction was neutralized and whatever previous
gain she had from the combined abdominal muscle con-
traction-diaphragmatic displacement was lost. Her ileus would
not affect her dyspnic symptoms when supine, however,
because in this position the abdominal contents themselves
would act favorably on the length-tension curve of the
diaphragm, as previously described.

While the explanation for this patient's platypnea is
speculative, no other cause for her positional dyspnea was
readily identifiable, and nothing but the simple treatment
of her ileus resulted in a dramatic relief of her platypnea.
The mechanism for possible interactions between an ileus,
COPD, and the acute onset of platypnea seem valid. It
would seem prudent then to add this combination of events,
particularly in the intensive care unit, to the small but
growing list of the causes of platypnea.

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