Hypokalemia Associated With Infra-His Mobitz Type Second Degree A-V Block

To the Editor:

Electrolyte disturbances may exert profound effects on myocardial impulse initiation, conduction, contractility, repolarization, and rhythmicity.1 Hypokalemia has been shown to prolong A-V conduction in pigs fed a low K+ diet and in the isolated frog turtle, and rabbit hearts when exposed to low K concentration.2,3 In contrast, in large numbers of patients with low K+ significant changes in the atrioventricular conduction could not be observed.4

The purpose of this report is to show intermittent conduction disturbances at infra-His level associated with iatrogenic hypokalemia.

A 75-year-old woman with weakness, palpitation, arrhythmia, hypotension, and congestive heart failure developed intermittent Mobitz I type of second degree AV-block. During sinus rhythm her ECG has shown left bundle branch block associated with a first degree A-V block (P-R: 0.24 s). Before her hospitalization, she used diuretics regularly with inadequate potassium intake. She was not receiving digitalis. His' bundle recordings taken when serum K was 3.1 mEq/L (Fig 1) revealed Mobitz 1 type second degree AV-block distally to His' bundle, with a 3:2 atrioventricular conduction ratio. Nota bene the progressive lengthening of H-V interval from 85 ms to 100 ms before the blocked beat distally to H without change of A-H interval in conducted sinus beats. After correction of potassium level to 4.5 mEq/L persistent sinus rhythm was observed with a normalized P-R interval of 0.18 s on conventional 12-lead ECG.

We are unaware of a similar report in the literature. Our case is of interest not only because of occurrence of Mobitz 1 type of second degree A-V block at infra-His' level, which itself is very rare,5 but the clinical circumstances compellingly suggest that there was some correlation between impairment of impulse conduction and potassium level.

These abnormalities of intraventricular conduction has shown progression with hypokalemia and disappeared when potassium level was normalized.

After correction of hypokalemia there was no His' bundle recording. Although the P-R interval normalized on the surface, ECG persistence of H-V prolongation cannot be ruled out.

Electrophysiologic changes, e.g. hyperpolarization, increase of rate of rise of phase O, and prolongation of action potential, occur at different levels of hypokalemia and cause either retardation or enhancement of conduction depending on the substrate.

Thus, hypokalemia appeared to potentiate subclinical conduction abnormalities in our case, however, pre-existent intraventricular conduction defect such as the presence of left bundle branch block does not exclude the possibility that the spontaneous progression of unilateral intraventricular conduction disorders to bilateral resulting intermittent Wenckebach type H-V block.

Gabor Veress, M.D., F.C.C.P.,
Hungarian Heart Center,
Balatonfüred, Hungary

**Figure 1.** Intracardiac recordings/HBE=His' bundle electrograms, HRA=high right atrial electrograms/with simultaneous I-II-III ECG leads in a patient with left bundle branch block and Wenckebach type H-V block.
Pulmonary Embolism and Occult Right Ventricular Infarction

To the Editor:

We have read with great interest the article by Adams et al relating to the elevation of MB isoenzyme fraction of creatine kinase (CK-MB) after pulmonary embolism as an important contribution to the knowledge of manifestation of occult right ventricular infarction.

We have recently treated a 69-year-old woman who was admitted to the hospital because of a syncpe without prodromal symptoms of ischemic heart disease. She developed progressive acute respiratory failure, so she was transferred to the critical care unit, where the laboratory findings were as follows: blood pressure 80/50 mm Hg, pulse rate 135 L/min, temperature 37.5°C, arterial blood gases (fraction of inspired oxygen 0.40) Po2 43 mm Hg, Pco2 35 mm Hg, pH 7.46, HCO3 25 meq/L, SaO2 82 percent, P(A-a)O2 199 mm Hg, CK 730, and CK-MB 20. An ECG showed sinus tachycardia, right axis deviation associated with complete right bundle branch block (pattern S1Q3T3), ST depression in the precordial lead with T-wave inversion in 3 and aVF. Chest roentgenogram showed unilateral right pleural effusion and bilateral alveolar and interstitial infiltrates without cardiomegaly.

Given the information summarized above, pulmonary artery pressures were measured using a Swan-Ganz catheter; pulmonary pressures were pulmonary artery pressure (PAP) 60/25 mm Hg, pulmonary capillary pressure (PCC) 18 mm Hg, and pulmonary artery diastolic pressure (PAD) 16 mm Hg despite the x-ray film depiction of pulmonary edema. A two-dimensional echocardiogram (2D-ECG) showed tricuspid regurgitation, maximum velocity (Vmax) 3.13 m/s, tricuspid gradient 39 mm Hg, pulmonary artery pressure 49 mm Hg, right ventricular posterior wall akinesis, and normal left ventricular function. The patient was suspected of pulmonary thromboembolism underwent a pulmonary arteriogram procedure with a Swan-Ganz catheter showing vessel cutoff and filling defects, finding severe hypoperfusion of the upper and lower right lobes. Once the diagnosis was set, in situ thrombolytic therapy with 1 million IU urokinase was started.

Posterior evolution showed improvement of the hemodynamic alterations with PAP 24/12 mm Hg, PCC 10 mm Hg, PAD 5 mm Hg, pulse rate 100 L/min, blood pressure 120/80 mm Hg. Arterial blood gases were pH 7.53, Po2 80 mm Hg, Pco2 53 mm Hg, HCO3 29 m eq/L, SaO2 98 percent (FlO2 0.40). A 2D-ECG revealed tricuspid regurgitation, Vmax 2.6 m/s, tricuspid gradient 20.9 mm Hg, PAP 90 mm Hg, and right ventricular posterior wall hypokinesia.

Clinical and laboratory findings are similar to those exposed by Adams et al,1 which confirms the importance of monitoring CK-MB and 2D-ECO in patients with pulmonary thromboembolism in which ECG has excluded the initial diagnosis of ischemic heart disease. With this, we consider that occult right ventricular infarction secondary to pulmonary thromboembolism will be no more considered as an isolated case, to become a situation to worry about not only in the acute state but also as prognostic incidence.

F. J. Andrade de la Cal, M.D.,
J. M. Aguado Borruey, M.D., and
J. M. Narvaez Bermejo, M.D.,
Critical Care Unit; and
A. Peres Garrido, M.D.,
Cardiology Service;
Hospital General de Merida,
Badajoz, Spain

REFERENCE


Exercise Testing in Evaluating High-risk Patients for Resection

To the Editor:

We read with interest the article by Holden et al2 on preoperative risk assessment for pulmonary resections. While the article states that arterial blood gases were obtained on all patients, these are not listed. We wonder if any of the patients who had complications or death had elevated PaCO2 levels2 This is generally held to be a relative contraindication to any pulmonary resection surgery.

James W. Adams, II, M.D., F.C.C.P., and
Patrick B. Hazard, M.D., F.C.C.P.,
Memphis Critical Care Associates,
Memphis, Tennessee

REFERENCES

1 Holden DA, Rice TW, Stelman K, Meeker DP. Exercise testing, 6-min walk, and stair climb in the evaluation of patients at high risk for pulmonary resection. Chest 1992; 102:1774-79

To the Editor:

Doctors Adams and Hazard raise an important issue regarding the significance of an elevated PaCO2 in the preoperative evaluation of the patient who will undergo thoracic surgery. The 1990 position paper by the American College of Physicians states: "Severe chronic obstructive pulmonary disease increases the risk associated with lung resection and persistent elevation of arterial Pco2 to >45 mm Hg suggests a very high risk relative to the benefit of the procedure."1 Carbon dioxide levels were <45 mm Hg in all 11 patients experiencing no or only minor complications in our study (range 35-42.5 mm Hg), whereas 1 of the 5 patients who died had a PaCO2 of 47 mm Hg.2 A second patient with a PaCO2 of 44.6 mm Hg was extubated in 24 hours after a pneu-