factors because, despite continuation of these drugs, clinical and biologic normalization persisted, even when corticosteroid drugs were progressively tapered off.

Hypereosinophilic syndrome appears improbable in the absence of other organ involvement.

The protracted clinical evolution, the peripheral eosinophilia and especially the dramatic improvement following therapy with corticosteroids are indicative of chronic eosinophilic pneumonia (CEP). Yet, the characteristic, peripherally located infiltrates on chest radiographs\(^5\) were absent in our patient. Open lung biopsy, however, confirmed this diagnosis.

We therefore conclude that in CEP, as in other types of interstitial lung disease, chest film findings may remain normal despite the presence of distinct histologic and functional abnormalities.

Even in such cases, open lung biopsy may not be required for the diagnosis of CEP, since marked eosinophilia in the bronchoalveolar lavage fluid, together with a compatible clinical picture, appears to be very characteristic. Indeed, the eosinophilic count is less in asthma or in histiocytosis X and is not accompanied by typical systemic symptoms.\(^6\)

Hunninghake et al\(^7\) described two types of pathologic lavage fluid patterns, i.e., increased lymphocytes or increased neutrophils. We suggest that there may also exist an eosinophilic type of alveolitis, in which the eosinophils are the main inflammatory and immune effector-cells, which may even cause cytotoxic effects by their cationic proteins.

## References


## Transient Left Posterior Hemiblock during Prinzmetal's Angina Culminating in Acute Myocardial Infarction*

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A 49-year-old man had transient left posterior hemiblock during Prinzmetal's angina with inferior ST-segment elevations; subsequently, left posterior hemiblock reappeared associated with acute inferior myocardial infarction. The electrocardiographic and electrophysiologic aspects of these findings are discussed.

Transient defects in intraventricular conduction described in Prinzmetal's angina have been limited to occasional bifascicular block,\(^1\) left anterior hemiblock,\(^2,3\) and left posterior hemiblock.\(^4\) We report a case of transient left posterior hemiblock during an attack of Prinzmetal's angina and, subsequently, during an acute inferior myocardial infarction. To our knowledge, we are unaware of other similar clinical observations.

## Case Report

A 49-year-old man of heavy body build with a previous history of chest pain of three weeks' duration was admitted because of chest pain at rest radiating to both arms. Physical examination showed only a faint midsystolic ejection murmur. The patient's blood pressure was 90/70 mm Hg, and his pulse rate was 55 beats per minute.

The electrocardiogram on admission (Fig 1A) during the chest pain showed normal sinus rhythm at 55 beats per minute. The QRS duration was 0.10 second, and the frontal QRS axis was directed to about +100°. There was a QR pattern with ST-segment elevation in leads 2, 3, and aVF and an rS pattern in leads I and aVL. Reciprocal ST-segment depression occurred in leads 1, aVL, and V₆. Following sublingual administration of nitroglycerin, the chest pain subsided. An ECG (Fig 1B) when the patient was free of symptoms showed normal sinus rhythm at 100 beats per minute, frontal QRS axis to about +65°, and QRS duration of 0.08 second. There was a negative T wave in leads 2, 3, and aVF without ST-segment displacement. The chest X-ray film and serial cardiac enzyme levels were normal. Treatment with nifedipine (20 mg every six hours) and isosorbide dinitrate (120 mg four times daily) was begun.

![Electrocardiogram showing absence of left posterior hemiblock and ST-segment elevation in leads 2, 3, and aVF. B, Electrocardiogram shows absence of left posterior hemiblock and ST-segment elevation.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21384/)
Twenty hours later, the patient had severe chest pain lasting 30 minutes and not responding to administration of nitroglycerin, which was relieved with morphine sulfate. The ECG (Fig 2A) during pain showed normal sinus rhythm at 95 beats per minute, frontal QRS axis to +110°, and QRS duration of 0.10 second. There was a QR pattern with ST-segment elevations in leads 2, 3, and aVF and an rS pattern in leads 1 and aVL. Subsequent enzymatic elevations were typical of acute myocardial infarction. Two days later, the ECG (Fig 2B) revealed normal sinus rhythm at 60 beats per minute and frontal QRS axis about to -20°. There were a Q wave, ST-segment elevations, and an inverted T wave in leads 2, 3, and aVF. The patient made an uneventful recovery and was discharged ten days later.

**DISCUSSION**

In 1973, Gorfinkel et al. described a transient left posterior hemiblock, although incomplete (QRS axis to +70°), in a patient with Prinzmetal’s angina who had 90 percent obstruction of the right coronary artery. Since then, transient left posterior hemiblock has not been specifically mentioned, but we have found four cases in a review of reports of Prinzmetal’s angina with inferior ST-segment elevations. Two out of four patients who developed this disturbance in conduction had subtotal obstruction of the right coronary artery. These four cases and our patient show transient right QRS axis deviation compatible with the diagnosis of left posterior hemiblock, that is: (1) a shift of the main QRS forces to the right, between +90° and +120°, causing tall R waves in leads 2, 3, and aVF and deep S waves in leads 1 and aVL; (2) a small but definite change in the direction of the initial 10-msec to 20-msec QRS vectors, which are shifted superiorly and to the left (since in our case a Q wave was already present in the inferior leads in the absence of complete left posterior hemiblock, the appearance of the latter did not change the initial vector of the QRS complex; (3) left posterior hemiblock provokes an S–Q in pattern by shifting the initial QRS forces to the left and superiorly, whereas the terminal force is directed to the right and inferiorly; and (4) the QRS interval is slightly prolonged, usually no more than 0.02 second.

Studying the very early changes in the QRS axis and ST segment following experimental coronary arterial occlusion, Conrad et al. showed three important changes in leads overlying the affected area: (1) the "injury"-related ST-segment changes; (2) the "injury"-related intramural (local) block; and (3) the increase in the size of the R wave, which is also dependent on the modifications of conduction produced by the "injury." If the affected zone is in the inferior myocardial wall, these modifications will result in a slight increase in QRS duration, as well as in the height of the R wave in leads 2, 3, and aVF. The latter, in turn, "pulls down" the terminal portions of the ventricular complex (S wave) in lead 1. Therefore, some degree of right QRS axis deviation occurs. As a consequence of these changes, the surface ECG will show some of the features attributed to left posterior hemiblock, as in our case and in the reports described previously.

The appearance of a "new" (small) q wave in the inferior leads (or the accentuation of a previously present q wave) suggests left posterior hemiblock, but it should be remembered that most inferoapical infarctions also involved the diaphragmatic portions of the interventricular septum. The latter may allow the more anterior (left-to-right) vectorial forces to increase in magnitude in a superior (caudal) direction.

In summary, whether these electrocardiographic features seen in our case reflect left posterior hemiblock or an acute "local" block in the posteroinferior wall of the left ventricle requires more clinical investigation.

**REFERENCES**

Respiratory Arrest following First Dose of Timolol Ophthalmic Solution

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Within 30 minutes of the administration of his first dose of timolol ophthalmic solution, a 67-year-old man with stable chronic obstructive pulmonary disease experienced severe dyspnea leading to respiratory arrest. He recovered after endotracheal intubation and mechanical ventilation. Patients with bronchospastic pulmonary disease who are candidates for therapy with timolol ophthalmic solution should receive their first dose under medical supervision and should have continued close medical follow-up for as long as they receive timolol.

Timolol maleate, a nonselective beta-adrenergic blocker, is used as an ophthalmic solution for the treatment of glaucoma. It possesses the therapeutic advantage of lowering intraocular pressure without changing pupillary size or causing diminution of vision, side effects which are prominent with miotic medications such as pilocarpine. Over half the reports of adverse reactions to timolol ophthalmic solution reflect systemic beta blockade due to absorption from the conjunctiva. Thus, therapeutic doses of timolol ophthalmic solution may cause a significant decrease in airflow in patients with bronchospastic pulmonary disease. The present case is important to note because the patient suffered a potentially fatal respiratory arrest within 30 minutes of his first dose of timolol ophthalmic solution.

CASE REPORT

A 67-year-old man had been receiving outpatient treatment for chronic obstructive pulmonary disease, non–critical calcific saccular stenosis, essential hypertension, and glaucoma. His medicines included hydrochlorothiazide, theophylline sustained-action tablets, and metaproterenol sulfate, both orally and by inhalation. Pulmonary function tests, performed seven months prior to admission, demonstrated moderate obstructive disease with air trapping. The forced vital capacity was 3.2 L and the forced expiratory volume in one second was 1.76 L (55 percent). There was no significant change in flow rate following the administration of bronchodilator drugs. He had been clinically stable, except for worsening glaucoma.

On the day of admission, he had been seen in the ophthalmology clinic, and, because of progression of glaucoma despite pilocarpine therapy, timolol ophthalmic solution (0.5 percent) was prescribed. That evening, within approximately five minutes of his first dose of one drop in each eye, he noted the acute onset of shortness of breath. His symptoms were not relieved by three of four puffs from his metaproterenol inhaler. Over the next ten minutes, his dyspnea progressed rapidly, and his wife noted that he was markedly cyanotic. She summoned the paramedics; when they arrived, the patient was unresponsive and apneic. Assisted ventilation with an Ambu bag and supplemental oxygen were begun, and he was transferred to a nearby community hospital.

When he arrived in the emergency room, the pulse rate was 120 beats/min and the blood pressure was 250/120 mm Hg. There was no effective spontaneous respiration. The arterial blood gas levels were pH, 6.59; PCO_2, 141 mm Hg; and PO_2, 71 mm Hg. Following insertion of an endotracheal tube under morphine sedation, mechanical ventilation was begun. He received intravenously-administered aminophylline and steroids and aerosolized isethionate hydrochloride. Forty minutes after intubation, he was responsive and able to follow commands. His breath sounds were much improved, although bilateral wheezes were present. The arterial blood gas levels were pH, 7.07; PCO_2, 93 mm Hg; and PO_2, 197 mm Hg (fractional concentration of oxygen in inspired gas [FIO_2], 0.50). Two hours after intubation, the arterial blood gas levels (FIO_2, 0.40) were: pH, 7.41; PCO_2, 36 mm Hg; and PO_2, 191 mm Hg. Although intermittent wheezing persisted, the patient was successfully extubated 15 hours later.

DISCUSSION

We did not rechallenge our patient with timolol in view of the severity of his bronchospasm; however, the temporal relationship between his first dose of timolol and the subsequent respiratory arrest strongly implicated timolol as the inciting agent. Timolol is known to be absorbed from the conjunctiva into the systemic circulation bypassing the liver where it is normally metabolized. The relative potency of timolol is approximately six times that of propranolol so that absorption of even the small doses of timolol used in ophthalmic solutions may cause systemic beta blockade. Patients with obstructive airway disease may be critically dependent upon beta-adrenergic stimulation for the maintenance of airway patency and thus be susceptible to severe and potentially fatal bronchospasm following the administration of timolol solution. In such patients, timolol ophthalmic solution should be considered only as a last resort when other methods of therapy have failed to control glaucoma. When timolol therapy is contemplated in a patient with obstructive airway disease, it may be helpful to perform spirometric evaluation before and after topical administration of timolol to determine if clinically apparent bronchospasm is provoked. We believe that patients with clinical evidence of increased airway reactivity should receive the initial dose of timolol ophthalmic solution under medical observation and that personnel and facilities should be available to perform resuscitation, if needed. Even if there is no apparent adverse reaction to the first dose, continued close medical follow-up is mandatory throughout the course of timolol therapy.

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