Jarisch led to the term, Bezold-Jarisch reflex, to describe an arc originating in cardiac sensory receptors located principally in the left ventricle, particularly along the inferoposterior wall, but also along the base of the right ventricle. Stimulation of these receptors by stretch or chemical agents triggers impulses along nonmyelinated vagal afferents. The consequent increased parasympathetic and decreased sympathetic activity induce bradycardia, vasodilation, and hypotension. Such stimulation has been described in inferoposterior myocardial ischemia and infarction, coronary catheterization, exertional syncope with aortic stenosis, and vasovagal syncope induced by tilt table testing. In acute pulmonary embolism, the hyperadrenergic state results in increased contractility of an underfilled left ventricle. This, in turn, may stimulate Bezold-Jarisch ventricular receptors and increase vagal impulses, causing sinus and atrioventricular nodal slowing, peripheral vasodilation, and syncope. Such a mechanism is consistent with the findings noted on ambulatory ECG monitoring in this patient.

The ischemic ECG changes, ie, ST-segment depression and T-wave inversion, were, in retrospect, consistent with those previously described in patients with acute pulmonary embolism. Early precordial T-wave inversion, which was present on the initial ECG, is a common clue to right heart strain and suggested the correct diagnosis. In several series of patients with acute pulmonary embolism, T-wave inversion and ST-segment depression were among the most common associated ECG abnormalities, which also included low QRS-complex voltage, an S1Q3T3 pattern (seen in this patient, Fig 1), right bundle branch block, and right axis deviation.

This is the first known case report of documented, high-grade atrioventricular block in an ambulatory outpatient with pulmonary embolism and syncope. The use of a Holter monitor in this individual enabled exquisite sequential recording of ST-segment depression and high-grade heart block exactly concurrent with his symptoms, supporting a Bezold-Jarisch vasodepressor reflex as a pathogenic mechanism.

**References**


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**Symptomatic Pericarditis After Influenza Vaccination**

**Report of Two Cases**

Antoine de Meester, MD; Raymond Lucaert, MD; and Jean-Marie Chaudron, MD

The authors report two cases of benign acute pericarditis after the patients received vaccinations against influenza virus. The diagnoses were confirmed by serologic changes and by the findings of 12-lead electrocardiogram and echocardiography. Symptoms and clinical status improved on aspirin therapy. The authors underline the possible mechanisms of this rare complication of influenza vaccination. (CHEST 2000; 117:1803–1805)

**Key words:** benign acute pericarditis; influenza virus; vaccination

The frequency of the administration of vaccination against influenza virus increases in patients at risk, as recommended by the Immunization Practices Advisory Committee in 1990. Vaccination is usually well-tolerated. Benign local or general reactions, like fever or myalgia, are frequently encountered. More rarely, systemic vasculitis or other immunologic diseases have been observed. We describe two cases of benign acute pericarditis occurring after influenza vaccination, which probably were caused by an immunologic systemic mechanism. Other such rare cases are reviewed.

**CASE REPORTS**

**Case 1**

A 75-year-old man was admitted for fever, shivering, arthralgia, and chest pain that increased with deep breathing. His medical history included chronic renal insufficiency after undergoing a left nephrectomy for a neoplasia 10 years ago, diabetes mellitus, and cigarette smoking. No problem of allergy was ever encountered. The symptoms developed only 6 days after he received vaccination for influenza (Vaxigrip; Pasteur Merieux MSD; Brussels, Belgium). Clinical examination was normal, except for a temperature of 38°C that was recorded on three occasions. No precordial friction rub was heard. The results of a 12-lead ECG were also normal. The laboratory investigations on admission showed the following results: WBC count, 16,220/mm3 with a differential of 92% neutrophils; C-reactive protein, 13.2 mg/dL; and a normal chemistry profile except for creatinine, 2.6 mg/dL. The results of rheumatoid factor test was negative. The results of serology tests for other viruses, including hepatitis A, hepatitis B, cytomegalovirus, mononucleosis, Coxsackievirus, and echovirus were negative. A chest roentgenogram showed enlargement of the cardiac silhouette. An echocardiogram revealed a small pericardial effusion, without any signs of tamponade (Fig 1). Pericardiocentesis was not performed at that time. The patient

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was treated with aspirin, 1 g tid, for 1 month. Clinical improve-
ment was observed after a few days. The patient was discharged
after receiving aspirin for 10 days. After 30 days, echocardiogra-
phy showed a complete disappearance of the pericardial effusion,
and aspirin was discontinued.

Case 2
A 40-year-old man was referred from another hospital for
coronary angiography. He was a current smoker and had hyper-
cholesterolemia. His medical history included a recent peptic
ulcer. Two days before admission, the patient suffered from
intermittent acute chest pain that was not pleuritic or positional
in nature. Pertinent physical examination revealed the following
results: BP, 140/80 mm Hg; pulse rate, 100 beats/min; and
temperature, 37.5°C. The heart sounds were distant. No cardiac
or pleural rubs were heard. Twelve-lead ECG showed normal
sinoatrial rhythm, PR segments that were clearly depressed, and ST
segments that were elevated (Fig 2). A diagnosis of acute
myocardial infarction was suspected. The patient was transferred
for coronary angiography and primary percutaneous transluminal
coronary angioplasty. The results of echocardiography and
chest roentgenogram were normal. Initial laboratory data re-
vealed a negative troponin T test, an abnormal WBC count of
12,500/mm³, and an erythrocyte sedimentation rate of 50 mm/h.
Urgent angiography was performed and showed healthy coronary
arteries. Left ventricular systolic function was also normal. The
next morning, a clear pericardial friction rub was heard. There-
after, the diagnosis of acute pericarditis was made. The patient
afterwards acknowledged having received a vaccination against
influenza 5 days earlier. On the second day of hospitalization,
repeat echocardiography was unchanged. The patient was dis-
charged on the third day after admission and was prescribed
aspirin to be taken orally.

Discussion
Influenza infection is a common viral illness. It appears
every year, infects the respiratory tract, and is responsible
for considerable morbidity and mortality, especially in
COPD patients, the elderly, or immunosuppressed pa-
tients. Common features of influenza virus include fever,
myalgia, cough, sneezing, sore throat, pneumonia, asthma,
and bronchitis. Neurologic complications reported are
convulsions neuritis, Guillain-Barré syndrome, encephali-
tis, and coma.²

Myocarditis is a rare cardiac complication, but it occa-
sionally progresses to congestive cardiac failure and
death.¹,³,⁴ Benign acute pericarditis is even more rare. The
viral origin is suggested by a history of ongoing infection,
typical chest pain, the audible friction rub, the course of
ECG changes, and possible pericardial effusion. The
diagnosis is supported by a significant rise of antibodies
against influenza virus.

Only a few cases of pericarditis after vaccination have
been published in the literature. In 1981, Streifler et al⁵
described the first case of recurrent pericarditis after
influenza vaccination. In 1997, Desson and colleagues⁶
described a similar case in a 40-year-old patient. At that
time, the Centre National de Pharmacovigilance in France
reported four cases of pericarditis after influenza vaccina-
tion. Other sporadic observations of pericarditis were also
described after vaccination against hepatitis B, yellow
fever, and smallpox.⁷⁻⁹ In 1977, Bloth and Lundman¹⁰
described a case of pleuroperimyocarditis caused by im-
munization with bacterial antiphlogos vaccine, with circu-
lating immune complexes in the patient’s serum. Such
bacterial vaccine etiology had not previously been de-
scribed. In any case, the reason that the vaccination is the
suspected cause relates to the chronology of the compli-
cation, as well of the resolution, and the fact that no other
viral causes were encountered. In the previous reports, the
vaccine antigens were not even found in the patient’s
serum. The hypothetical mechanisms of immunologic
systemic reactivity were not proven because of the rarity of
the disease. All cases were described after a first injection
of the influenza vaccine. A provocative test should prob-

Figure 1. Parasternal long axis view of the pericardial effusion on transthoracic echocardiography.

Figure 2. Twelve-lead ECG. PR segments, as well as ST segments, are displaced.
ably help us to find the real mechanisms for this rare illness. For ethical reasons, such a test has not been performed.

In conclusion, such cases of patients with pericarditis after influenza vaccination are rare, but the true incidences of the illness are probably underestimated. However, this complication does not outweigh the beneficial effects of the influenza vaccination in patients at risk.

References

Oxygen uptake (VO₂) at the alveolar-capillary membrane is a function of alveolar ventilation (VA), diffusion, and pulmonary capillary perfusion (Qc). The application of positive end-expiratory pressure (PEEP) in the treatment of ARDS is thought to recruit and splint alveoli at end-expiration and increase both the area and duration of alveolar gas exchange, thereby potentially improving VA/Qc matching. However, increased inspiratory airway pressures with PEEP may compromise pulmonary perfusion by limiting cardiac output and effective Qc. Titration of ventilatory variables to optimize pulmonary VO₂ may preserve both VA and pulmonary blood flow.

We describe the use of single-breath gas composition and expiratory flow data obtained with a computer-assisted multigas analysis and spirometry system to guide ventilator therapy in a patient with ARDS. Computer analysis and graphical display of superimposed gas flow rates and single-breath O₂ and CO₂ concentrations were

Single-Breath Measurements of Pulmonary Oxygen Uptake and Gas Flow Rates for Ventilator Management in ARDS*

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Monitoring data in critical care and anesthesiology should be displayed to present a rapid and easily comprehensible definition of the patient’s clinical status. A graphic computer display of the analog output of gas flow rates and the O₂ and CO₂ concentrations of respiratory gases profiles the expired breath for an estimation of pulmonary function and gas exchange. An estimate of pulmonary perfusion, cardiac output, and the general adequacy of cardiovascular circulation is obtained from the computer calculation of O₂ uptake and CO₂ elimination, dead space, and alveolar ventilation. Adjunctive data from the spirometric measurements of airway pressures, volumes, and compliance, supplemented by hemodynamic monitoring, aids in the diagnosis of physiologic changes. For > 10 years, we have used this system to monitor patients who are anesthetized, sedated, and receiving mechanical ventilation during anesthesia and surgery, and recently have extended the technique to intensive care areas. Our experience has shown good correlation of changes in the computer-assisted expired breath analysis with coinciding clinical events, including upper airway obstruction, bronchospasm, and alveolar volume/pulmonary capillary blood flow impairment. To demonstrate the use of this system, we describe the ventilator management for a patient with severe ARDS. In this patient, changes in ventilator management, including pressure control ventilation, improved pulmonary O₂ uptake (mean, 18.7 vs 8.5 mL/breath), CO₂ elimination (mean, 17 vs 13 mL/breath), and compliance (mean, 29.7 vs 19.0 mL/cm H₂O), were compared with intermittent mandatory ventilation.

(CHEST 2000; 117:1805–1809)

Abbreviations: ABC = arterial blood gas; Fio₂ = fraction of expired oxygen; Fto₂ = fraction of inspired oxygen; Fio₂ – Fto₂ = inverted oxygen concentration; IMV = intermittent mandatory ventilation; PCV = pressure-controlled ventilation; PEEP = positive end-expiratory pressure; PIP = peak inspiratory pressure; Qc = pulmonary capillary perfusion; VA = alveolar ventilation; VO₂ = carbon dioxide output; Vd = dead space; VO₂ = oxygen uptake; Vt = tidal volume.

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