manifestation. A central collection of epithelioid cells is surrounded by a rim of lymphocytes (hematoxylin-eosin, original magnification X200).

the left with the Weber test (512 Hz) lateralizing to the left.

A complete blood cell count, differential white blood cell count, sickling test, routine chemistry, arterial blood gases, urinalysis, and ECG were normal. The VDRL was nonreactive. Gamma globulins were elevated at 22 g/L (7 to 15) as was angiotensin- converting enzyme at 115 U/L (<75). A chest radiograph showed bilateral hilar enlargement. Paranasal sinus radiographs showed mild left maxillary antrum mucosal thickening. A radiograph and bone scan of the right knee revealed lateral femoral condylar subarticular lucency and increased activity respectively.

There were foci of increased activity on gallium scan in the mediastinum, hila, left supraclavicular region, and groins. Results of pulmonary function tests were normal. Borderline mitral valve prolapse was seen on echocardiogram and cardiac color Doppler study.

An audiogram disclosed mild conductive hearing loss on the left. Brain-stem auditory evoked responses were normal. A computed tomographic scan of the head demonstrated a soft-tissue density in the left middle ear deep to the tympanum and without bony erosion. The left mastoid air cells were opacified (Fig 1).

At left tympanotomy, a pale, pink granular mass could be seen filling the posterior portion of the tympanum and extending into the aditus and toward the sinus tympani. Portions were removed. Histopathologic examination showed noncaseating granuloma with collections of epithelioid cells surrounded by a rim of lymphocytes. There were occasional multinucleate giant cells and no acid-fast bacilli (Fig 2).

A left nasal septal polypectomy 1 month later also showed noncaseating granulomas. After several months on prednisone therapy, 40 mg daily, the hearing improved while the tinnitus remained. The postoperative audiogram showed slight conductive hearing loss on the left.

**DISCUSSION**

Sarcoidosis of the nose, nasopharynx, paranasal sinuses, larynx, pharynx, salivary glands, and cervical lymph nodes has been described. Middle ear involvement in sarcoidosis has hitherto been indirect, due to otitis media secondary to nasopharyngeal disease. Hearing loss has been of sensorineural type with various mechanisms proposed. Eighth nerve sarcoid involvement is commonly associated with other cranial neuropathies or uveitis. Auditory system sarcoidosis has also included the external ear. The initial presentation of sarcoidosis may be a head and neck manifestation.

In our case, systemic sarcoidosis was established by physical examination, blood tests (e.g., angiotensin-converting enzyme), and imaging. Pathologic confirmation came via the left middle ear mass and the nasal polyp.

We are unaware of an explanation for the rarity of middle ear involvement by sarcoidosis. The mucosa, blood supply, and oxygen tension are not significantly different from other regions of the upper respiratory tract. Oscillating vibration and a relatively small surface area to occupy are unsubstantiated, speculative possibilities.

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**Cough-induced Nonsustained Ventricular Tachycardia**

LeonardoLetsis, M.D.; Yosef Blaer, M.Sc.; Jamal Jafari, M.D.; and Mordechai Manoach, Ph.D.

We present herein a case of cough-induced nonsustained ventricular tachycardia for which we found no definitive explanation. It could be an anomaly, but this so far unreported clinical fact is worth noting. Notwithstanding this interesting case, we believe that cough can be recommended as a safe and efficacious technique for overcoming hypotensive cardiac arrhythmias.

**NSVT=nonsustained ventricular tachycardia; VT=ventricular tachycardia**

Coughing has been recommended as a safe and efficacious technique for overcoming ventricular tachycardia (VT). A deep forceful cough can be used as an effective resuscitative technique during hypotensive VT and hypotension. It has also been shown that it helps in altering the severe bradycardia and hypotension that may occur after intracoronary injections of contrast medium.

In an exceptional case, a 66-year-old woman was found to have episodes of nonsustained ventricular tachycardia (NSVT), following coughing attacks. We present herein a case report in which cough caused NSVT.

**CASE REPORT**

A 66-year-old woman presented at the Intensive Care Cardiac Unit with NSVT during coughing attacks (Fig 1).

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Physical examination revealed the following: blood pressure 120/78 mm Hg; pulse, 72 and regular; heart, normal; and mild wheezing in the lung. Results from ECG, chest radiograph, blood gases, and routine blood tests were within normal limits. Doppler echocardiography showed a normal heart. Ergometry induced NSVT in the second minute of the Bruce protocol.

Cardiac angiography revealed normal coronary arteries and normal left ventricular function. Electrophysiologic study was planned but the patient refused further investigation.

**DISCUSSION**

The use of coughing as a tool for conversion of VT has been known for many years. It has been suggested that coughing could help as a form of "cardiac massage," due to the intrathoracic pressure it induces.

Wei et al\(^5\) suggest that there are several advantages to cough-induced cardiac compression over external massage: the procedure is simple and can be self-induced; it does not lead to traumatic complications or possible damage to ribs or sternum; and it can be carried out anywhere and in any position.

Sakai and Mori\(^5\) reported a case of "schlucktachycardia" in 1926. Omari et al\(^5\) were the first to report a case of cough-induced tachyarrhythmia. To the best of our knowledge, this is the first report of NSVT precipitated by cough.

While coughing, the intrathoracic pressure becomes elevated and can increase to 450 mm Hg. The linear air velocity is 50 to 120 m/s and can rise as high as 280 m/s, approaching the speed of sound. The amount of air expired during a cough ranges normally from 1 to 3 L. From these values, one can estimate the amount of kinetic energy generated as approximately 1 to 25 J,\(^2\) thus, strictly on the basis of energy considerations, it may be feasible for cough to produce sufficient mechanical energy to cause cardiac depolarization.\(^3\) We found no common pathophysiologic mechanism to convincingly explain the precipitation of NSVT during cough and exercise. However, it is possible that increased air linear velocity in both exercise and coughing could stimulate the larynx, causing reflex NSVT.

**REFERENCES**


**Pediatric Lung Transplantation for Graft-Versus-Host Disease Following Bone Marrow Transplantation**

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Nine years after receiving a bone marrow transplant for aplastic anemia, a 14-year-old girl with severe pulmonary disease associated with graft-versus-host disease received a double lung transplant. Subsequent to lung transplant, her lung function improved dramatically (FEV\(_1\) increasing from 20 to 73 percent predicted normal, residual volume decreasing from 316 to 130 percent predicted normal values). The patient is currently well 15 months after transplant, while receiving immunosuppression consisting of FK506 and azathioprine. Double lung transplantation may offer a therapeutic option for the treatment of graft-versus-host pulmonary disease in selected patients.

(Chest 1994; 105:1584-86)

BMT=bone marrow transplantation; GVHD=graft-versus-host disease

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