1. Tract through mural thrombus and right atrium (arrows) produced by polyethylene catheter.

eter, which remained in place throughout the course, was as great as 10 liters per 24 hours. The central venous pressure remained less than 6 cm H_2O during this period but the excursion of the column in response to the heart beat was greater than usual. The cardiac silhouette was normal two days postoperatively.

Early in the morning of the eighth postoperative day the patient became confused and the blood pressure dropped from 125/80 to 100/75 mm Hg. He subsequently became tachypneic and dyspneic but did not complain of chest pain. Shortly thereafter, the central pulse was 120 beats per minute with a weak femoral pulse of 70 beats per minute. Blood pressure was unobtainable and the central venous pressure was approximately 15 cm H_2O. The patient died shortly afterward. No intracardiac injections were attempted.

At necropsy the pericardial sac was distended by 600 cc of serosanguineous fluid. There was a small hole on the ventral epicardial surface of the right atrium. A friable mural thrombus, measuring 2 cm in diameter, was attached to the endocardial surface immediately beneath this hole. A well-defined oblique tract, with a caliber of 1 to 2 mm, coursed through the thrombus and atrial wall. The central venous catheter had been removed before the necropsy. Microscopic examination disclosed necrosis of the atrial myocardium around the tract. A small pulmonary vein contained a recent thrombo-embolus.

2. Oblique section of tract through right atrium with necrosis of myocardium and fibrin deposition. Epicardium shown at bottom (hematoxylin and eosin, ×200).

The head of the pancreas was replaced by adenocarcinoma, which invaded the duodenum, lymph nodes, and liver. Death was ascribed to hydropericardium.

Comment.—A polyethylene catheter used to monitor the central venous pressure can be inserted mistakenly into the right atrium. The accurate radiologic localization of a radiopaque catheter tip in the superior vena cava would avoid this complication. In this patient, the beveled catheter tip lodged in the right atrium and injured the myocardium, causing a mural thrombus. The beveled catheter tip was probably held close to the atrial wall by the thrombus, and ultimately perforated. The well-defined tract through the thrombus and myocardium, and the necrosis of the myocardium at the puncture site, support this view. The effect of continuous flow of liquid from the catheter against the atrial wall is not known. The abrupt onset of signs of cardiac tamponade on the eighth postoperative day heralded the atrial perforation. A patent catheter conducting liquid under pressure into the pericardial sac made hydropericardium unavoidable.

The presence of a radiopaque tip on polyethylene catheters used to monitor central venous pressure would facilitate their proper placement. Calibration of these catheters by etching them at appropriate intervals would also be of value. Prolonged (greater than 48 hours[2]) and indiscriminate use of central venous catheters must be avoided.

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1. Turner, D.D., and Sommers, S.D.: Accidental Passage of a Polyethylene Catheter From Cubital Vein to Right Atrium. *New Eng J Med* 251:744-745 (Oct 28) 1954.

2. Brown, C.A., and Kent, A.: Perforation of Right Ventricle by Polyethylene Catheter, *Southern Med J* 49:466-467 (May) 1956.

3. Johnson, C.E.: Perforation of Right Atrium by a Polyethylene Catheter, *JAMA* 186:584-586 (Feb 14) 1968.

4. Doering, R.E.; Stemmer, E.A.; and Connolly, J.E.: Complications of Indwelling Venous Catheters: With Particular Reference to Catheter Embolus, *Amer J Surg* 114:259-266 (Aug) 1967.

5. Druskin, M.S., and Siegel, P.D.: Bacterial Contamination of Indwelling Intravenous Polyethylene Catheters, *JAMA* 185:966-968 (Sept 21) 1963.

Asbestos Bodies And Their Bioeffects

To the Editor:—This letter outlines the history of the asbestos body from its discovery through a period of little clinical significance to the present day when it holds the position of being possibly one of the greatest industrial medical problems of the age.

Asbestos:—Asbestos is a general term that covers a number of minerals of which four are of commercial importance: crocidolite from the Republic of South Africa, amosite also mined chiefly in the Republic of South Africa, chrysolite which occurs in many countries of the world, and anthophyllite which is mined mainly in Europe.

Asbestosis:—This is a type of lung fibrosis in workers exposed to large amounts of asbestos dust. It may or may not be associated with recognizable clinical disease during life and is produced by all types of asbestos.

Asbestosis was recognized as an industrial disease at the turn of the century, but at the time, the bodies were not noticed. Gloyne cites Fahr and Feigel who, in 1914, discovered "strange crystals" in lung sections from a patient with asbestosis.[1] Later Cooke, in 1924, reported "curious bodies" from asbestosis cases, but because of their beaded structure, he thought they might be fungi.[2] Stewart and Haddow (1929) believed that the bodies were associated with the disease and coined "asbestosis bodies."[3] This term was used until it became noticed that large numbers of bodies could be found in the lungs of asbestos workers in the absence of much pathological change, with subsequent substitution of the term "asbestos body." Gloyne in 1932 demonstrated that each body consisted of an asbestos fiber as a core with some coating material around it, which might protect the tissues from the harmful effects of the mineral.[4]

Wagner in 1960 reported that the rare pleural mesothelioma in the

Republic of South Africa was associated with exposure to crocidolite in the mining areas.[5] The association of ordinary bronchial carcinomas with exposure to asbestos dust had long been recognized, but it has recently been shown that this is related to high dust dosages in asbestos workers and that careful environmental control has almost eliminated this tumor as a problem in factories. Wagner reported that many of his cases of mesothelioma occurred in patients who had never worked in the mines but had lived in mining areas perhaps only for a year or two as children. He also showed that the time lag between last exposure and tumor formation was always long, averaging 40 to 50 years.

Asbestosis and bronchial carcinoma obviously connected with severe dust exposure were problems solely for the asbestos industry. But, if a very small amount of dust could cause a mesothelioma, might not asbestos products be hazardous to the whole population?

It was logical to examine the lungs of normal members of the population for the presence of these bodies. Thompson in 1963 found 26% of autopsy cases in Capetown contained what appeared to be asbestos bodies.[6] Other studies include one by Cauna et al in 1965 which reported that 46% of autopsies in the Pittsburgh area contained bodies.[7] Although fewer than 600 cases of mesotheliomas have so far been reported in the medical literature, the increase in asbestos consumption in recent years might produce a great epidemic of these tumors in the future.

These reports have received great publicity and led to the suggestion that the use of asbestos should be banned altogether. One article (*Prevention Magazine [July] 1967*) suggested that it might be unsafe to visit Expo '67 because of the risk of asbestos in the air.[8]

This asbestos scare has ignored some of the facts on mesotheliomas, not all of which can be taken at face value. First, asbestos is not a single mineral. Wagner's mesothelioma cases were associated with the crocidolite areas and not with amosite or chrysolite. In other countries attempts to demonstrate that exposure to dusts other than crocidolite could produce mesotheliomas have so far been almost completely unsuccessful. The mesothelioma problem is, therefore, not one that involves the whole asbestos industry, but only users of crocidolite, and this mineral represents a relatively small proportion of total consumption.

In the South African mining areas from which Wagner obtained his cases, it is common practice to surface roads with the waste asbestos rock, the rest of which is deposited in dumps. Wagner states that some of his patients had played among these dumps as children and others no doubt drove along the roads with car windows open. Although they may have had a low exposure by industrial standards, these individuals probably inhaled far more dust than members of a normal urban population.

For many years asbestos bodies were believed to be formed only around asbestos fibers, but recent evidence, from an endeavor to discover how many materials will produce asbestos-like bodies and the chemical processes involved, has shown that the deposition of a similar coating can occur around a number of materials.[9-11]

Gloyne[4] had demonstrated that the body coating contained iron, and Beger[12] had shown that protein material was also involved. Asbestos bodies, in the electron microscope, show a coating composed of small dense granules about 60 Angstroms in diameter. Similar granules in a number of tissues had been assumed to represent either ferritin or hemosiderin. Since this tied in well with the iron-protein nature of the capsule, it was suggested that the asbestos body coating was made up of one of these chemicals, and occasionally of fine filaments about 60 A in diameter which we now believe to represent calcium deposits.

Experimentally produced bodies with nonasbestos materials appeared very similar to asbestos bodies. They were golden brown in unstained sections and were often segmented and the coating contained iron. For this reason Gross suggested the term "ferruginous" body, which should be used for all bodies found in human lungs at least until the mineral involved was positively identified.[10,11]

Whatever the foreign material that stimulates the production of a ferruginous body, the coating consists of the small dense granules described above.

The situation at present is then that we must not assume that ferruginous bodies seen in the lungs of normal persons are asbestos bodies until their mineral core has been definitely identified by electron diffraction, which involves isolating, concentrating, and manipulating the small bodies onto an electron microscope grid. In addition to our analytical investigation of these structures which we hope will reveal which human ferruginous bodies are caused by asbestos, other studies now being conducted are exploring their epidemiology. A report on such a study recently published by Enterline and Kendrick[13] was the basis of an editorial in THE JOURNAL (201:966, 1967). In the field as well as the laboratory, therefore, there is justification for awaiting further evidence which may be expected to provide a sound basis, in fact, for solving the genuine health problems related to asbestos.

Interested readers are referred to a paper, "Asbestos Bodies and Bioeffects, A Detective Story" given by J.M.G. Davis at the 32nd annual meeting of the Industrial Hygiene Foundation in October 1967.[14]

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1. Fahr and Feigel, cited by Gloyne, S.R., *Asbestos*, International Labor Office Occupational and Health Supplement, (Jan) 1968.

2. Cooke, W.E.: Fibrosis of Lungs Due to Inhalation of Asbestos Dust, *Brit Med J* 2:147 (July 26) 1924.

3. Haddow, A.C.: Clinical Aspects of Pulmonary Asbestosis, *Brit Med J* 2:580-581 (Sept 28) 1929.

4. Gloyne, S.R.: Abestosis Body, *Lancet* 1:1351-1355 (June 25) 1932.

5. Wagner, J.C.: *Proceedings of the Pneumoconiosis Conference, Johannesburg*, A.J. Orenstein (ed.), London: J. & A. Churchill Ltd., 1959.

6. Thompson, J.G., Kaschula, R.O.C.; and MacDonald, R.R.: Asbestos As a Modern Urban Hazard, *S Afr Med J* 37:77-81 (Jan 19) 1963.

7. Cauna, D., Trotten, R.S., and Gross, P.: Asbestos Bodies in Human Lungs at Autopsy, *JAMA* 192:371-373 (May 3) 1965.

8. Anjilvel, L., and Thurlbeck, W.M.: The Incidence of Asbestos Bodies in the Lungs at Random Necropsies in Montreal, *Canad Med Assoc J* 95:1179-1182 (Dec 3) 1966.

9. Gross, P.; Cralley, L.J.; and deTreville, R. T.P.: "Asbestos" Bodies: Their Nonspecificity, *Amer Industr Hyg Assoc J* 28:541-543 (Nov-Dec) 1967.

10. Gross, P., et al: "Pulmonary Ferruginous Bodies: I. Their Development in Response to Filamentous Dusts; II. A Method of Isolating and Concentrating Them, *Arch Path*, to be published.

11. Davis, J.M.C.: Electron-Microscope Studies of Asbestosis in Man and Animals, *Ann NY Acad Sci* 132:98-111 (Dec 31) 1965.

12. Beger in Virchow's *Archives* 290:280-353, 1933.

13. Enterline, P.E., and Kendrick, M.A.: Asbestos Dust Exposures at Various Levels and Mortality, *Arch Environ Health* 15:181-186 (Aug) 1967.

14. Davis, J.M.G., et al: "Asbestos Bodies and Bioeffects, A Detective Story," in *Transactions of Industrial Hygiene Foundation's 32nd Annual Meeting*, to be published.