that a different distribution could have been seen with broadening of the palette of PiM subtypes with M₁ and M₆.

Independent of this, it would have been advisable to acquaint ourselves more closely with the biochemical properties of electrophoretic variants. The functional changes of the primary structure alone can hardly be the sole factor of pathologic action. A change in the tertiary structure can also be influenced by an inductive effect, for instance, the binding energy of the neuramine acid and therefore the splitting of the ester bond, and thus lead to a different biological action. An indication of this can be the split change of the Pi.

Presently we are informed concerning the structure of the pathogenic Pi variant Z¹⁰ and S¹⁰ as well as some of the problems of microheterogeneity.¹¹,¹² The question of the connection between structure and biological activity is still open.

In the phenotypes V, S, T or Y, Z the Pi values are not larger than M₁, M₃, M₄; however, Z and S have been assumed to be pathologic.

K. Bencze, Ph.D.; and G. Fruhmann, M.D., F.C.C.P.,
Institut and Poliklinik Arbeitsmedizin, University of Munich, Munich, Germany

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Preoperative Evaluation of Pulmonary Function

To the Editor:

With the advent of noninvasive radionuclide methods of determining regional perfusion and ventilation, there has been increasing interest in evaluating preoperatively the patient who requires pulmonary resection. I have followed recent publications, including the one by Ali et al. (Ches 1980, 77:337-342) who have discovered that following pneumonectomy, pulmonary regional and overall pulmonary functions are relatively stable, and that following subtotal pulmonary resection, there is a severe decrease in pulmonary function which gradually improve with time. They state that "the quantitative evaluation of the late functional recovery has not been documented before."

I have¹,² shown that following pulmonary resection of seven segments or less, patients suffered restrictive changes which were almost identical, but postpneumonectomy patients, especially those who developed contralateral hyperinflation, do not demonstrate this effect.¹,² Almost all patients recovered from restrictive effects in approximately six weeks, in a very predictable manner.

I also note that Boysen’s editorial (Ches 1980, 77:8-7), still places reliance on ventilatory tests in screening patients. We have found that ventilatory tests are of no greater help than clinical impressions in prognostication. Our continued experience supports our original findings³ which indicate that mean pulmonary artery pressures correlate best with prognosis.

Thus far, scintiscanning appears to offer little more than bronchospirometry, being preferred because it is noninvasive. The data of Ali et al, indicate that the technique is not accurate enough to rely upon where subtotal resection is planned.

David V. Pecora, M.D., F.C.C.P.,
Chief, Surgical Service, Veterans Administration Medical and Regional Office Center, Wilmington, Delaware

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To the Editor:

Dr. Pecora has commented about our discovery that follow- ing pneumonectomy, pulmonary regional and overall pulmonary functions are relatively stable. I refer Dr. Pecora to Table 3 of our paper published in Ches (1975; 68:392), which shows that we were the first group to present and publish data emphasizing the stability of regional pulmonary function following pneumonectomy.

Dr. Pecora’s quotation of our statement is incomplete. The sentence in our paper reads “Although this short term discrepency between the physiologic and anatomic loss following partial pulmonary resection has been noticed by other investigators,¹³ to our knowledge, the quantitative evaluation of the late functional recovery has not been documented.

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Self-terminating Torsades de Pointes or Holter Conversion?

To the Editor:

Undoubtedly, the case of “Holter auto-thumping” for termination of presumed quinidine-induced ventricular tachycardia was read with some interest and great amusement (Chest 1980; 78:674). There is, however, a more plausible explanation for the patient’s cardiovascular event based upon an analysis of the case history and ECG rhythm strips. It is clear from such analysis that the patient has a prolonged QT interval at the time of his normal sinus rhythm, undoubtedly related to quinidine therapy, followed by the degeneration to an arrhythmia characterized by an alternating pattern of electrical polarity where the QRS complexes appear to spiral around the isoelectric line. These features assure that the patient, in fact, experienced a classic episode of torsades de pointes, (TDP) as described by Dessertenne et al1 in 1966 and which was recently reviewed by Smith and Gallagher.2

The importance of this distinction is that TDP is usually self-terminating after a couple seconds to minutes, and this is the probable reason for apparent cardiovascular without the aid of standard resuscitation in this patient. In addition, since TDP is rarely converted successfully by standard methods of cardioversion,3 it is hardly plausible that the “Holter auto-thump” was responsible.

Also, we must await further investigations and case reports to assess the role of the Holter monitor as a therapeutic modality.

Frederick T. Zagibe, Jr. M.D.
University of Rochester Schools of Medicine and Dentistry, Rochester, New York

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Hepatoma Presenting as a Single Cavitary Lung Mass and Leukemoid Reaction

To the Editor:

I read with interest the article by Cooper and Hayes published in Chest, February, 1980. I recently had the opportunity to see a similar case referred in consultation because of a leukemoid reaction white blood cell count of 40,000 with a platelet count of 1,200,000 and normal hemoglobin of 14 gm.

The patient complained of evening fever up to 39°C associated with night sweats and pleuritic chest pains. On examination, decreased breath sounds were heard in the right lower hemithorax. A chest x-ray film showed a 2.5 x 3 cm lobulated cavitary lesion in the right lung base. Her alkaline phosphatase level was greater than 500 (normal up to 250), LDH, 557; SCOT, 88; and bilirubin 2.2. Isotope liver scan and CAT scans showed two large defects in the right and left lobe of the liver.

A needle biopsy of the liver demonstrated multiple foci of active granulomatous inflammation compatible with mycobacterial infection or fungal infection. The patient received a course of antituberculosis therapy with INH, ethambutol and rifampin. The patient’s condition continued to deteriorate and she became semicomatose. Her white blood cell count increased to 70,000 and the platelet count to 1,500,000. Her leukocyte alkaline phosphatase was very high (215) compatible with a leukemoid reaction.

At this time repeat sections of the original liver biopsy were made which revealed a moderately differentiated hepatocellular carcinoma (hepatoma). The patient expired two weeks after her admission and, unfortunately, an autopsy was not obtained. This case is interesting since it represents the second reported case of a hepatoma presenting as a single cavitary lung mass. Also, the very severe leukemoid reaction and the granulomatous lesions in the liver misled us originally to the presumptive diagnosis of tuberculosis.

Hepatomas, as do many other neoplasms, often produce a leukemoid reaction2 and hepatocellular carcinomas have produced secondary polycythemia in a number of reported cases,4,5 due in some instances to ectopic erythropoietin production.6

As in the case reported by Cooper and Hayes,1 our patient had a very high alkaline phosphatase level which probably is the most commonly elevated laboratory test in hepatoma.

We wish to emphasize that these interesting tumors could masquerade as polycythemia or even, as in our case, as cavitary pulmonary tuberculosis with liver involvement and severe leukemoid reaction.

Julio F. Ochoa, M.D.
St. Petersburg, Florida

Reprint requests: Dr. Ochoa, 1210 16th Street North, St. Petersburg 33705

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