limitation, Simon⁴ has suggested using an absolute measurement of the transverse heart diameter, and normative data are available.⁴ These data, taken from 100 normal chest radiographs, puts the upper limit of normal at 15.5 cm in the male and 14.00 cm in the female (with median values of 12.5 and 11.5 cm respectively). In the 100 chest radiographs reviewed by Simon and myself, the correlation coefficient for thoracic width and transverse heart diameter was 0.59 (p < 0.001) which perhaps justifies the use of the CTR in population studies. In individual cases, however, the ratio does not take account of such variables as the senile chest,⁴ where shrinkage and change in shape occur with age, and an absolute measurement may therefore be more appropriate in clinical practice.

Adrian J. Williams, M.B.
Co-Director, Pulmonary Function Laboratory,
VA Wadsworth Medical Center;
Assistant Professor of Medicine,
University of California, Los Angeles

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5 Burch GE. Of the heart-to-chest ratio. Am Heart J 1976; 92:284-65

To the Editor:

We have reviewed Dr. Williams’ references and agree with him that in absolute terms the measured transverse diameter of the heart may be more accurate in assessing cardiac size than the CT ratio.

In our article, we stated that the heart was evaluated in a subjective manner for chamber enlargement and pulmonary vasculature status. Fraser and Paré agree that it is preferable to evaluate cardiac status subjectively on the basis of experience. We would also suggest that the configuration of the chest be evaluated at the same time for variations in contour which might affect the overall impression of cardiac size. In our investigation, in order to quantitate heart size, we used the cardiac thoracic ratio because it is the most widely known and accepted standard for evaluation of heart size. In retrospect we should have reported the transverse cardiac diameter as well.

It is interesting to note that Benge and colleagues,¹ also use the cardiac thoracic ratio in measuring heart size. This emphasizes the fact that this measurement is still the most widely accepted standard for cardiac size. In the future we would advocate that investigators in this field should report both measurements.

Donovan B. Reinke, M.D.
Chief, Diagnostic Radiology Service;
Rex B. Shafer, M.D.
Chief, Nuclear Medicine Service,
Veterans Administration Medical Center,
Minneapolis

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1 Benge W, Litchfield RL, Marcus ML. Exercise capacity in patients with severe left ventricular dysfunction. Circulation 1960; 61:955-59

Risk of Cardiac Complications in Surgical Patients with Bifascicular Block

To the Editor:

I wish to comment on an article by Bellocci and coworkers in Chest (1980; 77:343-48).

Atrial pacing, as a provocative test, can be useful in assessing the His-Purkinje conduction system in patients with chronic bifascicular block during sinus rhythm. A latent conduction disorder of the third functioning fascicle is implied if rapid atrial pacing (100-200 beats/min) provokes block distal to the His bundle.¹ Pacemaker insertion is recommended if provocative pacing induces intermittent trifascicular block with intact AV-nodal conduction.

Temporary pacing in patients undergoing surgery may be indicated in patients with unstable angina or in those with a recent myocardial infarction. AV block is more apt to occur during episodes of acute ischemia.

Altschuler and co-workers² recommend pacemaker insertion in patients with bifascicular block, unexplained dizziness or syncope, and prolonged H-V intervals. No sudden deaths were observed in those patients prophylactically paced.

Donald Forester, M.D.
Director of Emergency Services, Queens Hospital Center, Jamaica, New York

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2 Altschuler H, Fisher JD, Furman S. Significance of isolated H-V interval prolongation in symptomatic patients without documented heart block. Am Heart J 1979; 97: 19-25

To the Editor:

We thank Dr. Forester for his comments and the interest he has shown in our article. In our study, we have demonstrated that patients with bifascicular block undergoing anesthesia and surgery, even in presence of prolonged H-V interval, do not require prophylactic pacing since the risk of complete heart block (CHB) is low and therefore they do not require pre-operative electrophysiologic studies. We have not considered longterm follow-up of our patients in order to identify those at high risk of CHB. On the other hand, the question of the value of the H-V interval in predicting the chances of CHB, even in symptomatic patients, is still controversial. In a recent editorial, Surawicz³ has stated that “the cumulative evidence from several studies suggests that the His bundle electrogams play a very small role in therapeutic decisions in patients with chronic bifascicular block and that the prognosis of these patients can be more accurately assessed by a thorough clinical evaluation than by the measurements of the H-Q interval.”

We agree with Dr. Forester that pacing-induced block distal to H with intact A-V nodal conduction implies disease in the His-Purkinje system and it would be an indication for pacemaker implantation; however, as demonstrated by Dhingra et al.,³ this pathologic response is an infrequent finding occurring in approximately 3 percent of electrophysiologic studies in patients with bifascicular
block and thus the diagnostic yield is too low to use this test in asymptomatic patients. These data further confirm our conclusion that preoperative electrophysiologic studies should not be recommended in preoperative patients with bifascicular block. Finally, we agree that preoperative patients with unstable angina or recent myocardial infarction are at high risk of infarction, reinfarction and, perhaps, CHB; however, since in our series no patient had unstable angina or recent myocardial infarction (in the preceding six months), actually we have no available data to indicate a more precise management of these patients.

Fulvio Bellocci, M.D., Pietro Santarelli, M.D., Department of Cardiology, Catholic University of Sacred Heart, Rome, Italy

Reprint requests: Dr. Bellocci, Servizio Cardiologia, Universita Cattolica S. Cuore, Larga A. Gemelli 8, Rome, Italy 00168

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1 Surawicz B. Prognosis of patients with bifascicular block. Circulation 1979; 60:40-42

Effects of Inhaled Atropine in Chronic Bronchitis

To the Editor:

We read with interest the report by Marini and Lakshminarayan (Chest 1980; 77:591-596) which describes the effects of inhaled atropine in chronic bronchitis. In a recent case report,1 we described a patient who exhibited their standards of improved pulmonary function (15 percent or greater increase in FEV1) following atropine, but not with a sympathomimetic.

In our patient, it was of particular interest that inhaled atropine not only relieved airways obstruction, but also suppressed severe paroxysmal coughing that had been associated with an episode of respiratory arrest. The mechanism for coughing in asthma and chronic bronchitis has been related to an increased sensitivity of subepithelial receptors which provoke cough by vagal responses2,3 or by local effects.4 We suggest that in addition to bronchospasm, the presence of cough may be an important clinical clue to the possible effectiveness of atropine or its derivatives in patients with chronic bronchitis.

Theodore J. Witek, Jr., B.S., RRT; Norman L. Dean, M.D., F.C.C.P. Department of Respiratory Services Griffin Hospital, Derby, Conn; E. Neil Schachter, M.D., F.C.C.P. Pulmonary Section, Yale University School of Medicine, New Haven

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1 Witek TJ, Dean NL, Schachter EN. Aerosolized atropine and relief of bronchoconstriction: a case report. Respir Care 1980; 25:570
2 Corrao WM, Braman SS, Irwin RS. Chronic cough as the sole presenting manifestation of bronchial asthma. N Engl J Med 1979; 300:633

To the Editor:

The stimulus for coughing associated with bronchospasm is not known with certainty. Stimulation of irritant receptors by bronchial deformation or by the chemical mediators of constriction1 is an attractive, but unproven hypothesis.

Although irritant receptor stimulation may initiate either bronchospasm or coughing, it does so by different pathways. The afferent limbs of both reflex arcs are carried by the intrathoracic vagus nerve. However, the efferent to bronchial smooth muscle return via the vagus, while the efferents of the cough reflex are located in phrenic, glossopharyngeal, and spinal motor nerves.2 Since inhaled atropine antagonizes the efferent vagus at the cholinergic receptor but leaves vagal afferents unaffected, it would be surprising if atropine proved a more effective antitussive in patients with bronchospasm than another non-anticholinergic drug which relieved constriction to a similar degree.

In this regard, it is worthwhile to note that in Simonsson's study (referenced above) atropine relieved bronchospasm but not cough, and that clinically, atropine has not proven useful for coughing unassociated with bronchospasm.2 Furthermore, in Corrao's study of coughing patients with positive methacholine responses (referenced above) theophylline and terbutaline were uniformly successful as cough suppressants. Therefore, the disappearance of cough following atropine in patients with reactive airways seems more likely to be related to the lysis of bronchospasm than to any specific antitussive action of the drug itself.

Rather than providing an indication per se for atropine, unusually severe coughing in a patient with bronchospasm may suggest an important contribution of irritant vagal reflexes to bronchoconstriction and therefore the utility of an anticholinergic for the purpose of bronchodilatation.

John J. Marini, M.D., and S. Lakshminarayan, M.D., F.C.C.P. University of Washington, Seattle

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3 Ziment I. Respiratory pharmacology and therapeutics. Philadelphia: WB Saunders, 1975; p 290

Gas Exchange Monitoring of ARDS

To the Editor:

In the article entitled "Evaluation of the Progress and Prognosis of Adult Respiratory Distress Syndrome" (Chest, 1979; 76:180-86), Shimada et al report their experience in following evolution of ARDS by measurements of pulmo-