Verification of Peritoneo-Pleural Fluid Communication and Mediastinal Lymph Nodes by Intraperitoneal Injection of Technetium-99m Albumin Colloid

TO THE EDITOR: Ascitic fluid draining into the pleural cavity, resulting in simultaneous presence of ascites and pleural effusion, has been documented in patients with cirrhosis of the liver (*1–4*). Existence of diaphragmatic defect was demonstrated through a diagnostic pneumoperitoneum, resulting in the development of a pneumothorax, by a thoracoscopy in cirrhosis patients with ascites complicated by pleural effusion, and/or at autopsy (*1,2,4*). Ascites draining to the mediastinal lymph nodes has also been demonstrated by nonradionuclide technique (*5*). We report the use of scintigrams taken after technetium-99m (^{99m}Tc) albumin colloid injection into the intraperitoneal cavity for demonstrating peritoneo-pleural fluid communication and visualized mediastinal lymph nodes.

A 61-yr-old man with a 20-yr history of alcohol abuse was admitted because of known cirrhosis of the liver with ascites and left pleural effusion. Physical examination revealed a malnourished man with yellowish discoloration of the sclera and skin. The abdomen was distended and showed evidence of ascites. Abnormal laboratory tests included a total bilirubin of 7.4 mg% (N = 0.15–1.00), albumin of 2.08 g% (N = 3.5–5.0) with total protein of 6.6m5% (N = 6.0–8.0), alkaline phosphatase of 148 μ/ml (N = 30 to 110), and SGOT of 80 μ/ml (N = 10–40). Prothrombin test was 18.2 min (control 12.3); PTT was 44 min (control 26.6). The chest radiograph showed left pleural effusion. Ultrasonography showed moderate ascites. To verify suspected communication between the peritoneal and the pleural cavities, the patient underwent the following radionuclide procedure: After intraperitoneal injection of 5 mCi [^{99m}Tc]albumin colloid i.p., the patient was placed in Trendelenberg's position, then sequential images of chest and upper abdomen were obtained at 5, 10, 15, 25, and 30 min, and 5, 22, and 27 hr. Small foci of radiotracer accumulation were first seen in the mediastinum at 15 min; they persisted throughout the study. Radioactivity in the left chest was first seen in the 30-min image and persisted through all subsequent images (Fig. 1). The ascitic fluid was confirmed as transudate. Technetium-99m albumin colloid liver-spleen scintigraph performed 2 wk later was compatible with the diagnosis of cirrhosis of the liver. The patient underwent supportive treatment with some improvement.

Radiopharmaceuticals have previously been used to demonstrate pleuroperitoneal communications, including [^{99m}Tc] sulfur colloid (*6–8*), [^{99m}Tc]albumin colloid (*9*), and ^{99m}Tc tin colloid (*10*). In our study, scintigraphic demonstration of lymphatic drainage to the mediastinum from the peritoneal cavity may be explained by drainage either directly into the mediastinum or indirectly to the mediastinum through the pleural cavity. Lymphatic drainage of the peritoneal cavity into mediastinal lymph nodes has been reported in the rat: After intraperitoneal injection, chick erythrocytes enter lymph node lacunae in the diaphragm within 30 min and can be detected in the mediastinal lymph nodes in 60 to 120 min (*5*). Lymphatics are capable of removing intraperitoneally injected protein-rich fluid fairly rapidly (*5*); therefore, ascitic fluid is not stagnant.

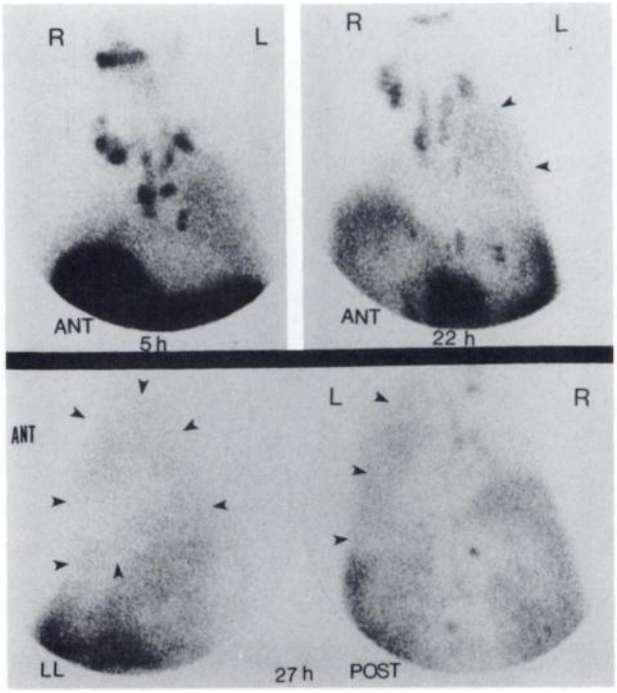

FIGURE 1
Chest and upper abdominal images at 5 hr, 22 hr, and 27 hr post i.p. injection of [^{99m}Tc]albumin colloid showing "hot spots" in the mediastinal region in the lower neck. Diffuse radioactivity in the left chest—in the pleural cavity is seen (arrowheads).

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Biventricular Forward and Regurgitant Flows by Radioangiography

TO THE EDITOR: In the paper published in the Journal (*1*) Nusynowitz et al. presented their methodology to assess central circulation kinetics from the first-pass data. More recently the same group reported on clinical aspects in applying their methods (*2*). I wish to comment on several aspects of their work (*1*) that I believe are in error.

In order to calculate biventricular flows they used the Stewart-Hamilton principle (SH) which, applied to the first-pass radioindicator ventricular kinetics, reads (*3,4*):

$$\text{Flow} = \frac{\text{equilibrium count rate} \times \text{blood volume}}{\text{area under the first-pass curve}} \quad (1)$$

In applying Eq. (1) sequentially to the right ventricle (RV), the lungs and the left ventricle (LV), they observe progressive diminution of the flows. They ascribe these downstream flow declines to bolus smearing (in nonregurgitant patients) and to smearing and regurgitation (in regurgitant patients). They further assume that, in absence of right-sided regurgitation, the lung flow equals forward LV flow. Thus they conclude that LV regurgitant flow is the difference in the apparent LV flow, calculated from Eq. (1) using LV area, and the "corrected" LV flow, calculated from Eq. (1) using pulmonary radiohistogram.

The described methodology is ill-posed: the flows obtained utilizing SH principle via Eq. (1) are forward, functional flows, that are invariant to both regurgitation and bolus smearing. I will briefly reestablish these known facts. SH principle relates the indicator concentration at the system output c(t) with indicator output F·c(t). Here F is recognized as the system effective output that carries convectively the indicator particles across the output boundary. Only then the total indicator input (I) is recovered by summing the sequential outputs F c(t):

$$I = F \cdot \int_0^\infty c\,(t)\,dt, \quad (2)$$

which is the formulation of the SH principle. That F in Eq. (2) is the forward flow is widely recognized (*4–7*) and practically utilized (*5–7*) feature. In order that homeostasis is preserved the biventricular forward outputs must be the same and equal to the lung flow, unless there are atrial or ductal shunts. In the absence of shunts unilateral regurgitations cause the difference in the ventricular total: forward+regurgitant flows, the feature widely explored for radionuclide quantitations (*8–11*). The flows calculated from Eq. (1) also do not depend on the tempo of indicator input, that is maybe more obvious by observing that the denominator in Eq. (1) is the product of the total indicator input and the mean residual time of the indicator particles in the ventricular cavity (*12*).

Further, there are two obstacles in applying Eq. (1) to the lung area, as done by Nusynowitz et al. First, in developing Eq. (1) from SH principle one assumes that the radiohistogram generated over the whole system is proportional to the indicator concentration curve at the system output. This may be closely fulfilled for the ventricles, but lungs are hardly close to the perfect mixing model. Second, only part of the lungs is in the background free area, available for the curve generation, whatever projection be used. This further introduces uncertainties in calculating pulmonary flow via Eq. (1).

Finally, Nusynowitz et al. utilized the following relations (*1:Eqs.3,4*):

$$\text{end-diastolic volume} = \text{stroke volume/ejection fraction} \quad (3)$$

$$\text{end-systolic volume} = \text{end-diastolic volume- stroke volume} \quad (4)$$

Equation (3) is correct if both stroke volume and ejection fraction are measured consistently. Nusynowitz et al. calculated the stroke volume from the forward flow F obtained via Eq. (1), while they analysed the first-pass curve oscillations, that gives the total ejection fraction. Thus they underestimated the end-diastolic volumes of their valvular patients. Equations (3 and 4) give:

$$\text{end-systolic volume} = \text{end-diastolic volume} \times (1\text{-ejection fraction}). \quad (5)$$

It follows from Eq. (5) that underestimation of diastolic volume implicates underestimation of systolic volume.

The interaction of the two errors in the paper of Nusynowitz et al.—estimation of LV output using pulmonary radiohistogram and underestimation of LV volumes in valvular patients— may explain for apparent success in correlating radionuclide with catheterization data (*1*) (*Figs. 4, 5, and 6*).

Currently valvular regurgitations can be evaluated by radionuclides using several approaches: biventricular difference in stroke counts obtained from equilibrium (*8,9,10*) or the first-pass study (*11*); comparing the total ventricular flow obtained volumetrically with forward output obtained via SH principle (*5–7*); using pulmonary input deconvolution of radioventriculogram for calculation of forward ejection fraction (*13*), or analysis of the delayed transit time components (*14*).