and is labelled “SC” (systolic click) during inspiration. Their Figure 1B demonstrates similar findings, but with less change of the systolic click to later in systole with inspiration. The systolic click in both of their illustrations actually appears to become louder in early expiration. Such changes would be analogous to the systolic-click changes with mitral valvular prolapse. The early systolic murmur appears to be unrelated to tricuspid valvular prolapse and might be a functional early-systolic right-ventricular flow murmur.

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To the Editor:

Dr. Sassé’s observations on the auscultatory findings associated with the click-murmur syndrome in mitral valvular disease are certainly appropriate. At least one of these observations seems to support our view that in our cases the systolic click and murmur originated from the tricuspid valve. As Dr. Sassé points out, maneuvers which increase left ventricular volume result in the movement of the mitral systolic click later into systole. Inspiration, which augments right ventricular filling, results in a similar movement of the systolic click away from the first heart sound in our cases, reinforcing the notion that the clicks originate from the tricuspid apparatus.

Dr. Sassé correctly points out that in the classic situation the systolic murmur has its onset with the midsystolic click. A number of published phonocardiograms, however, indicate that the click may occur within the murmur. An example of this is Figure 1A in the report by Epstein and Coulshed cited by Doctor Sassé.

The point of our report was to indicate the probability that isolated tricuspid valvular disease could produce auscultatory findings similar to those described in mitral valvular prolapse. Dr. Sassé correctly points out that proof is lacking in our cases and that the diagnosis of isolated tricuspid disease is circumstantial. It will be difficult to accumulate a large series of cases of systolic clicks emanating from an isolated tricuspid lesion and to define all of its auscultatory features.

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IPPB: Yes or No?

To the Editor:

With reference to the interesting exchange of views in the April issue (Chest 67:469-472, 1975) between Dr. Martin Gold, who points out that the field of intermittent positive-pressure breathing (IPPB) therapy has grown into a gigantic business while the therapy itself remains a failure, and Dr. Theodore Noehren, who believes that despite its excesses, the field of IPPB therapy could not continue to thrive as it does were it not for some benefit to the patient, I should like to agree with both authors for the following reasons:

Therapy with IPPB can be of benefit in noncomatose patients with chronic obstructive lung disease (COLD) only in the presence of a specific indication and while observing specific modes of administration. The indication is the presence of potentially reversible airway obstruction due to mucus, edema, or spasm. The specifics of administration are the selection of a bronchodilator with a-adrenergic and β₂-adrenergic¹ properties, the adequate dilution (tenfold to 20-fold) of the bronchodilator with water, and the delivery of such an aerosol with machine inspiratory flow rates set to match those needed by the patient, which, in the case of airway obstruction, means low flow rates. Failure to comply with the specifics of administration just outlined usually results in failure of IPPB treatment, thus confirming Dr. Gold’s hypothesis.

The field of IPPB therapy continues to grow not only because it is a good moneymaker, but also because, if properly administered with the indications set forth above, IPPB gives most patients with COLD five advantages which cannot be as readily obtained by other means: (1) treatment and prevention of ventilatory failure and CO₂ retention, (2) more effective bronchodilation than with a hand bulb or pressurized aerosol, (3) improvement in intrapulmonary gas mixing, (4) reduction in airway resistance by adjusting inspiratory flow rate to the degree of patency of the patient’s airways, and (5) reduction in the work of breathing. It must be stressed emphatically that none of these advantages...
will obtain in patients whose airways have no reversible obstruction and in whom the specifics of administration outlined earlier are not observed.

In sum, it is the reversible component of COLD which requires prompt attention with bronchodilators, and it was pointed out long ago that IPPB is nothing more than an elegant way of administering bronchodilators. Miller and his co-workers3 established 20 years ago that IPPB offered no advantage which could be gained by power nebulization, except in patients with severe ventilatory failure. This work was confirmed by many others.5-6 Therapy with IPPB on the wards and in the home could be replaced entirely by power nebulization and Ventimask oxygen therapy, but while IPPB respirators are much more expensive, they may be simpler to use once they are set up.

We stressed in 19637 and again in 19728 that if IPPB is to be effective in COLD, the machine must deliver adjustable inspiratory flow rates at low cycling pressures and with sufficient dilution of the bronchodilator to minimize its undesirable side effects. These principles have been amply confirmed9-14 and, when applied in patients with reversible airway obstruction, have proved beneficial. This explains why Dr. Noehren's hypothesis is also warranted.

Without further long-term studies documenting in detail (a) the concentration and dosage of nebulized drugs, (b) the type of nebulizer and IPPB machine employed, including (c) pressure and flow settings, as well as (d) the proper inspiratory/expiratory time ratios so important in keeping mean intrathoracic pressure low, (e) the length and frequency of treatments, (f) the effect of additional therapy (drugs and exercises), (g) serial blood gas analyses, and, above all, (h) the extent of reversible obstruction present before initiation of treatment, it may well be difficult to confirm or invalidate the proposition that IPPB is beneficial to patients with COLD exhibiting reversible airway obstruction.

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To the Editor:

Dr. Sheldon, whose early rather analytic report1 on intermittent positive-pressure breathing (IPPB) therapy is well known to students of the subject, seems to say, in summary, “We cannot prove or disprove the efficacy of IPPB, but since (many or most) ill patients receiving it feel better, there is good reason for utilizing it. However, such victims of chronic obstructive lung disease (COLD) should receive IPPB according to strict and proper guidelines.” The specific guidelines delineated in his letter have not yet been established by accepted scientific methods, and my question remains unanswered: Where is the incontrovertible evidence in favor of IPPB therapy after 25 years of use?

Dr. Sheldon proposes that IPPB be used for patients with COLD only with specific indications and with specific modes of administration. There are a number of well-controlled investigations in patients who do exhibit “potentially reversible airway obstruction” and who have received IPPB therapy according to the manner and technique of administration which Dr. Sheldon carefully delineates.8-6 These studies conclude that IPPB does not improve measured variables significantly in patients with COLD. One of Dr. Sheldon’s requisites concerns the matching of machine flow rates to those needed by the patient; unless the therapist uses a pneumotachygraph or other similar device, this requirement may be difficult to obtain. The guidelines which are