PHYSIOLOGIC ADVANTAGES OF GRUNTING, GROANING, AND PURSED-LIP BREATHING: ADAPTIVE SYMPTOMS RELATED TO THE DEVELOPMENT OF CONTINUOUS POSITIVE PRESSURE BREATHING

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IN a study on the physiologic uses of pleasure, Cabanac reported that the enjoyment of sugar by human subjects in the fasting state was replaced by an unpleasant sensation when sweet substances were administered two hours after ingestion of 100 gm. of glucose; man's behavior in this instance manifested a specific physiologic aim.[1]

I was stimulated by Cabanac to give a historical account of another teleological design: how observation of the symptoms of respiratory disease led to the development of forms of pressure breathing which later confirmed the adaptive value of the patient-suggested remedy. The expiratory grunt in lobar pneumonia and pursed-lip breathing in chronic-obstructive lung disease seemed to have physiologic advantages to the patient which were later duplicated by mechanical means: i.e., continuous positive pressure breathing (CPPB) during the respiratory cycle and expiratory positive pressure breathing (EPPB), which provided positive pressure in expiration only.

The clinical use of pressure breathing then evolved as a practical procedure for the treatment of bronchial asthma, obstructive laryngeal dyspnea, and pulmonary edema.[1-4] Seegal, in a discussion of Nature's role in assisting the investigator to perceive new methods of management, declared that it would be appropriate for authors to list Nature in the bibliographies of these articles, even if the credit was limited to the status of et al.[5]

HISTORICAL REVIEW

The earliest use of pressure breathing consisted of forced inflation of the lungs with a passive expiration, the original example of which seems to be the biblical record of Elisha, who restored the life of the son of a Shunammite woman by putting his lips on the mouth of the apparently dead child (II Kings 4:34). Mouth-to-mouth insufflation was not formally described until 1847, when Tossach revived a man "seemingly dead in appearance."[6]

Mechanical expansion of the lungs developed gradually, with the use of devices which maintained artificial ventilation of the lungs, to be known as intermittent positive pressure breathing (IPPB). In 1896 Norton reported the first cure of acute edema of the lungs in a patient exposed to the fumes of carbolic acid; the forced respiration apparatus was used at Presbyterian Hospital, New York City.[7] Many varieties of IPPB are now employed in respiratory failure, including body respirators as well as volume- and pressure-controlled mask devices pressured by air and oxygen.

Man himself may be said to practice IPPB since a negative intrapleural pressure is induced in the chest by the bellows mechanism of the diaphragm and intercostal muscles, thus initiating a flow of atmospheric air into the lungs. The evolution of more overt patient-suggested therapy is the subject of this paper, since the expiratory phase of pressure breathing was duplicated by CPPB and EPPB.

The expiratory grunt, a cardinal sign of lobar pneumonia, is reported infrequently at present, since antibiotic treatment generally stops the progress of the disease before the onset of groaning and grunting respiration. However, in 1919, as an intern at Presbyterian Hospital in New York, I had an opportunity to note this phenomenon, for it often became evident on the fourth to sixth day of pneumococcus pneumonia.

On one such occasion, a 40-year-old man exhibited loud forceful expiratory grunts on the fifth day of disease; morphine was injected to alleviate his apparent suffering. Within 10 minutes the grunt disappeared and moist rales of fatal edema of the lungs were heard. Oxygen was administered by a funnel held an inch from the patient's face; it was bubbled through a water bottle from a cylinder which contained 20 cu. feet of oxygen at 400 psi.

An identical reaction to hypodermic injection of morphine was observed in 1928 in a man in his early 30s. This patient had massive consolidation in one lung, with a pneumococcus type III bacteriemia. Although his color had improved in the oxygen tent on the second day of the disease, his temperature continued high and the pulse rapid. On the fourth day of illness an expiratory grunt became manifest and, as the day wore on, groaning respiration became more prominent. The patient was in such distress that he pleaded for relief. The first response to the injection of morphine, 0.015, seemed favorable, as he rested quietly. However, in about 15 minutes loud gurgling sounds from his lungs and throat heralded the fatal pulmonary edema.

The response of these two patients with lobar pneumonia suggested that termination of their expiratory grunts released a flow of serum from the lung capillaries; the positive pressure reflected backward on the pulmonary circulation appeared to have the physiologic aim of preventing edema of the lungs.

In 1934 a man's use of pursed-lip breathing (PLB) revealed the adaptive purpose of this maneuver in an unmistakable manner. This 60-year-old patient with pulmonary emphysema had come to our clinic for treatment of severe breathlessness. He manifested an unusually marked degree of pursed-lip breathing; his lips almost closed in expiration as he expelled air forcibly, with prominent contractions of the abdominal muscles. I illustrated to him how one could breathe with the mouth open. When he tried to exhale with the lips parted, he seemed to suffocate until he suddenly constricted his lips and forcibly exhaled the pent-up air from his lungs.

PLB had been observed in subjects with chronic obstructive lung disease (COLD) and emphysema by Laennec[8] in 1830; in fact, PLB had been advocated as a physical exercise for these patients by Saenger[9] in 1910 and Hofbauer[10] (1925). Breathing against a resistance to expiration imposed by constriction of the mouth has also been employed during severe physical exertion by mountain climbers, stonecutters, and lumbermen.[11] The latter use of PLB will be seen to be different in physiologic aim than that of COLD.

Relation of Adaptive Use of Pursed-Lip Breathing to the Physiologic Effects of Continuous Positive Pressure Breathing

The emphysema patient revealed that the back pressure induced by PLB had maintained a patent bronchial lumen during expiration. The relief was immediate, too quick to be accounted for by a slow respiratory rate. The use of CPPB was reported in conjunction with the administration of helium-oxygen mixtures in the therapy of bronchial asthma, obstructive lesions of the respiratory tract and, with oxygen, in pulmonary edema (1935-1936).[2-4] The advantages of patients' adaptive PLB were confirmed by the physiologic findings induced by CPPB: the peripheral venous pressure was elevated. The circulation time was prolonged in cardiac insufficiency with a decrease of blood entering the right heart. The small bronchi were enlarged in expiration as lung volume increased.[2-4, 12, 13] Clearing of pulmonary edema and nocturnal paroxysmal dyspnea in cardiac subjects was induced by the use of a pulmonary-plus machine that also maintained a continuous pressure through the respiratory cycle.[14]

In recent studies of physiologic effects of PLB on patients with COLD, considerable emphasis is given to the physiologic advantages of the slow respiratory rate, with its resultant increase in tidal volume and lowering of the $PaCO_2$. Thus, Thoman et al. had patients breathe with slow respiratory rates with PLB and found that appreciable declines in $PaCO_2$ occurred in this group as well as in those who used PLB.[15] These investigators state: "Whether the slowing of respiration or the increase in intra-luminal pressure, or both, are responsible for the observed improvement is not settled." However, they conclude that the changes in rate are the most important.[15] Ingram and Schilder, who found that patients who benefitted from PLB had a greater decrease in nonelastic transpulmonary resistance than those who did not purse their lips, suggested that PLB may have provided symptomatic relief by preventing collapse of larger bronchi.[16] In addition to the relief from the discomfort of breathing due to the prevention of bronchial narrowing by PLB, they mention that the theory of Campbell and Howell may be applicable, in which dyspnea is considered as the result of an inappropriate relation between the volume of breathing and the pressure used by the respiratory musculature.[17]

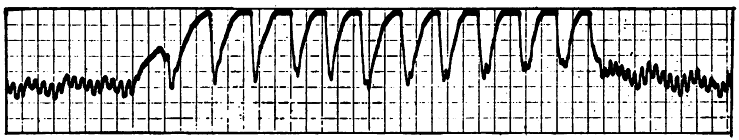
Mueller et al. reported that a lowered $PaCO_2$ was induced by PLB in resting subjects with COLD. They then state: "Our data suggest that symptom benefit from PLB is related to the magnitude of respiratory rate decrease and tidal volume increase it induces."[18] However, since these beneficial responses did not occur in exercising patients who often are markedly relieved by PLB, the blood-gas data seem an incidental finding. Further, the relief of dyspnea occurs at the *onset* of PLB, too soon for the improved blood-gas findings caused by the slow respiratory rate to be relevant to an explanation of the sensation of dyspnea. In fact, Mueller et al. also conclude: "One could also explain our findings and those of Ingram and Schilder by reasoning that PLB prevents airway collapse in those chronic airway obstruction patients who have this phenomenon."[18]

The circulatory response to PLB should also be considered in discussions of PLB since a marked increase in peripheral venous pressure may be induced by this maneuver. In patients in whom its adaptive use is to prevent excessive blood from entering the lungs, PLB is not necessarily accompanied by a slow respiratory rate. This group includes cases of overt or latent left-ventricular failure, including some with colds, as well as people engaged in severe physical exertions, chopping wood, shoveling snow, or climbing mountains. As seen in Figure 1, the venous pressure rise to 62 mm. H_2O during expiration illustrates the effect of increased intrapulmonary pressures induced by PLB.

I have observed patients with COLD who promptly adopt PLB when placed in the head-down position. The sudden inrush of blood into the heart prompts a defensive reaction in patients in whom latent or overt insufficiency of the left heart is present. This special circulatory function of PLB was observed in a patient who had a history of coronary thrombosis and recurrent paroxysmal pulmonary edema. In the absence of the EPPB device usually employed, a woman of 65 resorted to PLB on my instructions at a time when frothy fluid was already escaping at the mouth. In five minutes a full recovery took place, with short expirations utilizing forcible PLB.

In patients with COLD the mechanism of relief of dyspnea induced by PLB must include the instantaneous cessation of use of accessory muscles of respiration. As the patency of the bronchial passageway is restored, the obviously burdensome contraction of the girdle muscles of the neck and shoulder are eliminated, with immediate subjective relief; the diaphragm simultaneously takes over as the bellows responsible for ventilating the lungs. Blood-gas improvement is a later phenomenon and irrelevant as an explanation of relief of dyspnea (see accompanying figure).

The abdominal muscles, as a result of PLB, push the diaphragm to a higher position in the chest, from which it then contracts efficiently. This thesis is supported by the similar relief of dyspnea seen in COLD patients who lean forward or who lie head down; accessory muscle respiration is no longer utilized and the diaphragm resumes its function.



	Control	Pursed Lip Breathing	Control
Inspiratory	80.7	92.8	81.5
Expiratory	97.6	159.8	104.5
Mean	88.5	136.2	92.3

The mean rise of venous pressure during pursed-lip breathing was 48 mm. H_2O; during the expiratory cycle the rise for 62 mm. H_2O. Reproduced from Barach, A. L., Bickerman, H. A., and Beck, G. J.: Advances in the treatment of non-tuberculous pulmonary disease. *Bull. N.Y. Acad, Med. 28*:368, 1952.

Subjective relief and objective evidence of dyspnea are also terminated almost instantly.

Enlargement of the diameter of the bronchi has been demonstrated at pressure as low as 6 cm. H_2O,[12] lower in fact than those demonstrated with PLB.[19] Marked increase in bronchial size has been demonstrated in man at 20.0 cm. H_2O[20] and twofold at 40 mm. Hg in animals.[21]

Positive and expiratory pressure (PEEP) has gained acceptance in the treatment of acute respiratory failure (ARF); the advantages of this technique have been shown to be in large part associated with restoration of the patency of the bronchi with expiratory pressures of 5 to 10 cm. H_2O.[22] In fact, recognition of the beneficial consequences of PEEP has led to the present revival of CPPB in ARF;[23] as well as the use of new CPPB apparatus in patients with chronic obstructive lung disease.[24, 25]

The adaptive use of PLB and the similar physiologic effects of pressure were originally expressed as follows:

> The helpful effect of positive pressure in expiration as well as inspiration in patients with asthma and emphysema explains the clinical observation that some patients will purse their lips in such a way as to create an obstruction to expiration, as if they were blowing against a resistance. The purposeful nature of this at first sight extraordinary performance is confirmed by their own confession that they feel "easier in their breathing." The groan in pneumonia seems to me to have a purposeful significance also. It builds up a positive expiratory pressure against the pulmonary capillaries, tending to prevent exudation of serum

into the alveoli. Pulmonary edema is susceptible of prevention in animals and relief in patients by inhalation of air under positive pressure.[3]

Patients who claim disability, and those nervous individuals who may seek special attention from their families, often keep their mouths wide open as they exhale with loud manifest signs of obstructive, asthmatic dyspnea and the production of sonorous rales. The slow (prolonged) exhalation of these individuals *exacerbates obstructive dyspnea as the alveolarbronchial passageway verges on total collapse* at the end of expiration. These adverse effects are either eliminated or reduced by PLB, a consistent response that suggests that bronchial narrowing has been ameliorated.*

Summary

The physiologic advantages of the expiratory grunt and groaning respiration in lobar pneumonia appeared to be related to the production of positive expiratory pressures which challenged the entrance of blood into right heart and lungs. The adaptive aim of PLB thus included this specific effect on the circulation.

In COLD, the crucial physiologic gain seemed to be the preservation of the patency of the bronchial passageway. The resultant elimination of dyspnea is achieved by cessation of burdensome neck-and-shoulder girdle respiration as well as the initiation of diaphragmatic breathing.

The physiologic studies on continuous positive pressure breathing, expiratory positive pressure breathing, and positive end expiratory pressure have resulted in extensive confirmation of the clinical value of the expiratory phase of pressure breathing, including the adaptive responses to pursed-lip breathing.

*Expiratory sibilant and sonorous rales are not only eliminated by positive pressure breathing but also by inhalation of helium-oxygen. The high quavering voice produced by inhaling helium atmospheres lasts longer in patients with COLD than in normal individuals, which indicated a retarded emptying capacity of the lungs in these subjects; a quantitative test of the rate of clearance from the lungs included the measurement of successive samplings of expired air for helium by a densiometer.[3]

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