It is sometimes hard to diagnose mild gas pains, and quite often only the suggestion of the term will make a patient complain. Nurses, also, will call anything gas pains that is associated with pains in the abdomen after operation. As long as there is no sign of even mild tympany, I have not felt myself justified to speak of gas pains but rather term them "cramps." These cramps are said to be relieved often by a rectal tube and therefore "must be gas pains," but I have found that they are also relieved by anything that will bring back a smooth downward peristalsis, as for instance by a glycerin suppository or an enema which does not bring flatus, and above all, by food. The empty bowel seems to contract violently in certain segments; and since the stimulus for a rhythmic downward motion, that is, food, is missing, peristalsis seems to churn back and forth in certain segments, producing "cramp-like" pains, which some of our patients likened to the sensation following the administration of compound cathartic pills. Estimating the number of patients with "cramps," I have, perhaps, been very liberal, as I may have called "cramps" what in reality was pain in the operative field, which by coincidence disappeared after the patient had partaken of solid food. Handling of the viscera undoubtedly predisposes to gas pains. In the second half of the cases observed all the patients that had gas pains had dense adhesions, which had to be broken up. They also had vaginal drainage, and I recall that as soon as the drain was removed, gas pains ceased at once in a few of the patients.

As far as the bowel at operation is concerned, none of us had any difficulty in packing off. I myself believe that the nonpurged bowel always appears normally pale and well collapsed, while the purged bowel has a slight distention and a dusky hue. I would emphasize particularly that the large bowel was sufficiently cleansed by the enema alone. No difficulty was encountered on that score. At the present time, preoperative purging has been stricken from the ward rules. Our patients seem to be better off, and much work and worry in treating gas pains has been eliminated.

GAS PAINS IN HERNIA

While the foregoing observations were being made, we found that the hernia patients were often bothered with severe gas pains. At my suggestion, Dr. John F. Cowan stopped the giving of castor oil to these patients before operation. A few were still given a mild laxative forty-eight hours before operation. With the permission of the members of the general surgical staff, I observed twenty-six laparotomies, including fifteen herniotomies and six appendectomies. Here again the results were very promising. Among the hernias there was not one case of severe gas pains. There was only one case of mild distention and practically no cramps, while in all the appendectomies the patients had been free from any abdominal distress. I have not counted in the patients who received the mild laxative, as they would spoil the argument. Dr. Emmet Rixford told me lately that he has made quite similar observations in his long practice, and that he also believes purging is not beneficial, and as far as technic is concerned, it can be dispensed with. He still feels that it is good practice to clear the bowel four or five days before operation with a mild laxative in order to remove any possible impacted fecal material.

It is not within the scope of this paper to discuss the treatment of gas pains. It may be said, though, that all treatment should tend to restore normal peristalsis. The best stimulus for this is food, and the earlier it is given, the better for the comfort of the patient. In many patients, especially those that have not been purged, the bowel will perform its function normally as soon as solid material finds its way into the bowel, and quite often even mild laxatives may be dispensed with.

The watchword of modern medicine is "prophylaxis." We have applied it to the operating room by developing asepsis to its highest degree. Do not let us stop there, but consider prophylaxis also for the postoperative condition of our patients. Let us eliminate untimely purging, one of the biggest factors in producing gas pains.

SUMMARY

1. Clinical and experimental observations strongly suggest that preoperative purging is a strong factor in the producing of gas pains.
2. At operation the strongly purged bowel is more difficult to handle than the unpurged bowel on account of congestion and distention.
3. An enema will clear the lower bowel sufficiently for any operative work.
4. Patients who have not been purged are comparatively free from gas pains.

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MOISTURE IN THE AIR SPACES OF THE LUNGS AND OXYGEN THERAPY *

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While on duty at a casualty clearing station in Flanders in the summer of 1917, I observed several respiratory phenomena associated with foam in the air passages which conferred an entirely new significance on the presence of moisture in the bronchial tree.

The patients who exhibited these symptoms were men who inhaled phosgen and chlorin. These patients had, essentially, a large amount of plasma poured into the air spaces of the lungs, which was not secondary to incompetence of the left ventricle. It was a severe primary edema of the lungs unassociated with myocardial incompetence.

One of the things that impressed me was the disproportion between cyanosis and air hunger. The evidence of cyanosis was very much more pronounced than was air hunger in most of the patients.

Another very striking experience was the manner in which patients were relieved of cyanosis by the inhalation of oxygen, and the fact that the relief of cyanosis was not attended with any change in the volume or rate of respiration; and subjectively the patients were not made more comfortable from the inhalation of oxygen.

If a gassed patient, whose bronchial tract is filled with foam and who has cyanosis, breathes pure oxygen, he is relieved of cyanosis, but the relief does not come promptly. It is unlike relief from cyanosis which has been caused by laryngeal obstruction. When a

* Read before the Section on Pharmacology and Therapeutics at the Sixty-Ninth Annual Session of the American Medical Association, Chicago, June, 1918.

patient suffering from cyanosis due to laryngeal obstruction has the obstruction relieved, cyanosis disappears within a few seconds and the patient is instantly made comfortable. The gassed patient was not relieved of cyanosis until after he breathed pure oxygen for from three to five minutes. The patients could not tell when they were breathing oxygen and when they were breathing atmospheric air. In fact they preferred to be relieved of having a mask over their faces, although this method gave them the greatest relief from anoxemia. Evidently the psychic effect of the obstructing mask contributed to their discomfort and they begged to be relieved of it. They preferred to have the stream of oxygen conducted into their respiratory tract by way of a soft rubber tube inserted in the nostril, although by this method I did not see a single case in which cyanosis was diminished. It is quite apparent that cyanosis and air hunger did not develop apace with each other and that anoxemia could be relieved without giving any relief for air hunger. The problem that requires an explanation is how anoxemia and air hunger are largely dissociated when the patient is breathing atmospheric air, and why only cyanosis is favorably affected when the patient breathes pure oxygen.

It has been suggested the dissociation is due to the film of moisture which intervenes between the ventilating air and respiratory membrane. Carbon dioxide is twenty-five times as soluble in water as oxygen, and consequently the escape of carbon dioxide from the blood is much better fulfilled than the absorption of oxygen. Carbon dioxide can escape from the blood, but oxygen is absorbed in much smaller amounts. This explanation obviously will not satisfy the clinical experience. If, according to the predicate on which this theory is advanced, the patient is suffering from anoxemia and has an adequate escape from carbon dioxide acidosis, then the abrogation of anoxemia should give the patient perfect comfort. There is another point about this theory which is not consistent with the facts. Plasma in the bronchial tree and respiratory units does not lie as a film of moisture but occupies the air space as foam. At the end of an expiration, the entire air space of the lung from the nares to the respiratory membrane is occupied by foam. The total volume of this foam must amount to at least 1,200 c.c. and during inspiration all the contained foam is aspirated into the air spaces and will be unequally distributed throughout the respiratory units. Some units will be filled with foam, and no doubt many units will contain no foam whatever. This foam is alveolar air foam and when it has acquired the same partial pressure of carbon dioxide as the affluent blood to the lung, all escape of carbon dioxide from the blood into the unit occupied by foam will be impossible. When a respiratory unit is full of alveolar air foam, the only area for escape of carbon dioxide will be the cross section of bronchi which supply any portion of the lungs thus affected.

The fact that the circulating blood in the lung of a normal person at rest is able to contribute 20 c.c. of carbon dioxide to the ventilating air during each respiratory cycle is due to the large area of the respiratory membrane.

The following experiment illustrates the significance of the area of the cross section of a communicating path between two fields of different partial pressures of a gas.

Two 3-liter bottles were furnished with a connecting tube which was 20 cm. long and had a diameter of 1.5 cm. One bottle contained 19.5 per cent. of carbon dioxide and the other bottle contained 0.1 per cent. carbon dioxide. Both were at barometric pressure. The communication between the two bottles was established and specimens of air were taken from each bottle by employing a device which enabled us to remove specimens of air from each bottle without disturbing the barometric pressure.

The results summarized in Table 1 give the comparative concentration of carbon dioxide in each bottle at a given time.

TABLE 1.—COMPARISON OF AIR SPECIMENS IN CARBON DIOXIDE CONTENT

Time	Percentage Carbon Dioxide in A	Percentage Carbon Dioxide in B
11.35	0.1	19.5
11.45	1.25	18.2
11.55	3.25	17.0
12.55	3.75	15.3
1.25	4.85	13.9
2.40	6.20	11.4

The same experiment was then repeated by using a connecting passage of the same length as in the first experiment but with a lumen of 5 cm. The results obtained are set forth in Table 2.

TABLE 2.—COMPARISON OF SAME SPECIMENS AS IN TABLE 1, WITH LUMEN OF 5 CM.

Time	Percentage Carbon Dioxide in A	Percentage Carbon Dioxide in B
11.30	13.7	1.2
11.40	7.3	7.0
12.00	6.4	6.4
12.20	6.4	6.3

This simple experiment is given merely to cite the fallacy in thinking of partial pressure as synonymous with barometric pressure. It also enables one to visualize the significance of communicating areas in establishing equilibrium of partial pressure between two gas fields of different concentrations.

VENTILATION OF PULMONARY BLOOD

If a respiratory unit is filled with alveolar air foam, then the area for the evolution of carbon dioxide is manifestly cut down from the large area of the respiratory membrane to the cross section of its bronchiole, and if at the end of an expiration the entire air space of the lung is filled with foam, the only area for the escape of carbon dioxide is the cross section of the trachea. It seems perfectly clear that any portion of the respiratory membrane which is covered by alveolar air foam will be deprived of its normal respiratory function, both for the escape of carbon dioxide and the absorption of oxygen. If at the end of a deep inspiration there is an unequal distribution of foam throughout the air spaces, different portions of the pulmonary blood will be ventilated in different degrees. If the patient takes a deep inspiration of 2,000 c.c., he will then have superventilation of some of the air spaces, and consequently, superventilation of the blood in these respiratory units, but the air spaces occupied by foam will be eliminated from the respiratory function. Consequently, we shall have admixtures of varying degrees of venous and arterial blood going to the left auricle. From the superventilated unit there will be contributed an effluent blood with saturated oxyhemoglobin and a partial pressure of carbon dioxide less than normal. From the foam units there will be contributed a blood with a high carbon dioxide content and a low oxygen content. The total effluent blood

from the lung under such circumstances will then contain an increase of carbon dioxid partial pressure and a low oxygen content. The superventilated units compensate in some measure for the want of ventilation in the foam units, so far as the partial pressure of carbon dioxid is concerned, but they cannot compensate for anoxemic blood which comes from the foam units and this I conceive to be the source of the disparity between anoxemia and air hunger in gas poisoning.

Now what will happen if the patient breathes pure oxygen? When pure oxygen is breathed, it will require several minutes before the alveolar air foam will have been changed to oxygen foam, and when the oxygen foam is once formed, what will have been accomplished for respiration? The foam units will have their entire respiratory membrane exposed to oxygen foam, and thus the area for the absorption of oxygen will have been many times increased. What has been accomplished for the ebullition of carbon dioxid? That will remain the same as when the patient is breathing atmospheric air and leaves the carbon dioxid content of effluent blood of the lung unchanged; but anoxemia will have been disposed of. This I conceive to be the explanation for the clinical fact that, after the inhalation of pure oxygen for a period of from three to five minutes, the patient regains a normal color but retains the same respiratory discomfort. This idea of the admixture of unrespired blood with the effluent stream has been ignored in clinical practice, yet we have an abundance of evidence that it occurs in congenital heart disease, occlusion of a single bronchus, lung consolidations, and moisture in the bronchial tree.

A child with an open ventricular septum and an open ductus botalli will be cyanotic, but has no air hunger when at rest. If he is given pure oxygen to breathe, the cyanosis remains unchanged. For under such circumstances the unrespired blood does not come in contact with the respiratory membrane. If the child exercises, cyanosis, as well as partial pressure of carbon dioxid in the aortic blood, will rapidly increase. Consequently with exercise, cyanosis and air hunger increase in equal paces.

If such a patient is instructed, while quietly sitting, to employ forced breathing for three minutes, cyanosis will diminish. But under these circumstances, ventilation of the lung and oxygenation of the blood and minute volume flow of blood through the lung will all be greatly increased. Thus when, in morbus caeruleus, ventilation is increased disproportionately to the increase in gaseous metabolism, anoxemia lessens and the carbon dioxid partial pressure in the blood will have greatly diminished. In morbus caeruleus we have an admixture of superventilated blood, in which there is a low partial pressure of carbon dioxid and a normal oxygen content, with another stream of blood which has a high carbon dioxid and a subnormal oxygen content. Consequently, there will be a disproportion between cyanosis and air hunger.

It is perfectly clear that in any disease in which unrespired blood is contributed to the aortic stream, the partial pressure of carbon dioxid and oxygen of the alveolar air cannot be accepted as the measure of the partial pressure of carbon dioxid and oxygen in the aortic blood. Gas analyses of alveolar air are applicable to the circulating blood only when every portion of the respiratory membrane exposed to ven-

tilating air has also a normal exposure to circulating blood. Furthermore, in every part of the lung from which blood is contributed to the aortic stream, there must also be a normal exposure of the respiratory membrane to ventilating air. If these two conditions are not fulfilled, then interpretations from gas analyses of respired air will be erroneous.

CYANOSIS IN LOBAR PNEUMONIA

In lobar pneumonia there is always a certain amount of cyanosis. It is a common thing to see a patient with lobar pneumonia who has pronounced cyanosis and although the patient has moderate hyperpnea, he will deny any sensation of air hunger when he is questioned on the point. If blood flow through the pulmonary vessels of the consolidated lung is maintained, then the effluent blood from the consolidated lung must be venous blood. The superventilation of the uninvolved portion of the lung will compensate for the increased carbon dioxid contributed to the aortic stream from the consolidated lobe, but superventilation of the uninvolved parts of the lung will not compensate for the anoxemia which is contributed from the hepatized lung. In this mechanism we see an explanation for cyanosis without air hunger in lobar pneumonia. The cyanosis is due to anoxemia and not to methemoglobin, for when the shed blood is exposed to atmospheric air, it immediately takes on the normal color of shed blood. The anoxemia is not in any way associated with the infection. It is due purely to the admixture of unventilated blood with the aortic stream. When pneumonia terminates in crisis, cyanosis persists, and finally disappears just as the exudate is absorbed and ventilation of the affected lung is resumed.

The alveolar air of a pneumonia patient cannot be taken as an expression of the gaseous content of the aortic blood. One need only to estimate the carbon dioxid and oxygen content of the alveolar air of a pneumonia patient to be convinced of the fallacy. The patient will be cyanotic but in spite of this the partial pressure of oxygen in the alveolar air will be higher than in a normal person and the carbon dioxid partial pressure will be lower than normal.

During the past winter I have had many opportunities to test out lobar pneumonias. All the patients had cyanosis and the cyanosis was proportionate to the area of the lung which was involved, and was not at all associated with cardiac incompetence.

For instance, if one lung is hepatized and the other lung contains no moisture, the patient will have pronounced cyanosis with superventilation, but he will not admit that he has air hunger. If such a patient is given pure oxygen to breathe, cyanosis is unaffected. But if the patient has one lobe hepatized and an abundance of moisture in the remaining portions of the lung, then the inhalation of pure oxygen will partly diminish the cyanosis, but the experience of air hunger is unmodified. I think this accounts for the different views on the subject of oxygen therapy in pneumonia. A patient who is cyanotic from both lung consolidation and moisture in the air spaces will benefit from the inhalation of oxygen so far as anoxemia derived from the edematous lung is concerned, but he will not benefit by anoxemia so far as the consolidated lung is concerned.

A further proof of the origin of cyanosis in pneumonia lies in the fact that occasionally we see a patient in whom cyanosis is very pronounced and then ceases

without change in the exudate. Thus I have in mind two patients, one of whom was seen by Dr. R. G. Pearce and one by myself. Each had one entire lung consolidated, with normal heart and normal blood flow, so far as could be determined by physical examination, and no signs of moisture in uninvolved portions of the lungs. Then within twenty-four hours cyanosis ceased, although there was not the slightest evidence of improvement in the infection or improvement in consolidation of the lung. The only explanation I have for such a phenomenon is that during the course of the pneumonia the blood flow through the pulmonary vessels of the consolidated lung was greatly diminished at the time when cyanosis ceased.

The patient seen by Dr. Pearce died the day after the cyanosis disappeared, and the other patient under my observation, at Lakeside Hospital, made a good recovery.

Thus far there has been presented no direct evidence on which we can estimate the blood flow through the pulmonary vessels of a hepatized lung. It seems very reasonable to believe the blood flow through a hepatized lung is diminished by the loss of respiratory extension and retraction even when the pulmonary veins and arteries are patent. The anoxemia of lobar pneumonia and the fact this anoxemia is uninfluenced by the patient's breathing pure oxygen is very satisfactory evidence of the fact that an unrespired blood flow through the consolidated lung is the source of anoxemia. And the fact that anoxemia may cease or grow less during a period when fever, hepatization and the cardiac output are unchanged seems to be converse proof of the correctness of the theory.

It is a common experience to find patients with pleurisy with effusion sufficiently large completely to eliminate the lung of the affected side from respiratory function, and yet the patient may not have the slightest trace of cyanosis. In the compressed lung there is not only a want of ventilation but there is also a great diminution in the minute volume of blood going through the pulmonary circulation of the compressed lung. Consequently, there is little or no unrespired blood contributed to the aortic stream. This view will explain the want of cyanosis in many chronic tubercular diseases of the lung, and will also explain the presence of cyanosis in acute miliary tuberculosis.

In the chronic tubercular lung the ischemic process and the progress of infiltration go hand in hand. Consequently as a portion of the lung is eliminated from the respiratory function, its pulmonary circulation is encroached on, therefore such an involved portion of the lung will not contribute unrespired blood to the aortic stream. But acute miliary tuberculosis is accompanied by an abundance of moisture in the air spaces which will contribute unrespired blood to the aortic stream.

TWO CASES OF CARDIAC INCOMPETENCE

In former years we conceived air hunger and cyanosis accompanying incompetence of the left heart to depend on the diminished minute volume of blood passing through the lung. But since the rôle of moisture in the air spaces has assumed greater importance in our estimates of air hunger and cyanosis, I have had the opportunity of testing two cases of cardiac incompetence associated with pulmonary edema.

A patient had chronic cardiovascular and renal disease with great dilatation of the heart and of the jugular veins, a large and sensitive liver, and general anasarca. The patient had intense air hunger and was coughing up great quantities of

bloody foam. He was given pure oxygen to breathe by means of a mask connected with a rebreathing device, so that he breathed pure oxygen through a bag of oxygen which was inserted into the circuit. The carbon dioxide was removed from the expired air by a canister of soda lime which was introduced into the circuit. Breathing through this closed system for about three minutes was sufficient to change the patient's color from pronounced cyanosis to a normal pink color, but the patient experienced the same air hunger and the volume and rate of respiration were unmodified.

Another patient was brought into Lakeside Hospital suffering from a head injury. The patient was in coma. He had a dilated heart and a great abundance of foam in his bronchial tree. This patient was made to breathe pure oxygen through a closed system and the same thing happened as in the patient above described. The pulmonary edema and the volume and rate of respiration were all unmodified. But after inhaling pure oxygen for about three minutes the patient regained a perfectly normal color, which he retained for a period of about four hours, during which time he breathed pure oxygen through the closed system. At the end of that time he died from cardiac failure, and although he had cardiac dilatation, a filiform pulse and extreme pulmonary edema, he had no cyanosis when he breathed pure oxygen.

We have here exactly the same mechanism as was cited in cases of gas poisoning. The alveolar air foam was changed to oxygen foam. This compensates for the anoxemia that existed when atmospheric air was breathed, but it did not compensate for the increase in the partial pressure of carbon dioxide in the aortic blood.

A THIRD CASE

Another experience illustrates the diagnostic service of this point of view.

A man, aged 19, was known to have stenosis and insufficiency of the aortic valve with hypertrophy and dilatation of the left ventricle. Four days before entering Lakeside Hospital he had an initial chill, followed by fever. Cough and bloody sputum developed on the following day. Coarse and fine moist râles were heard over all of both lung areas. On the day previous to his admission to the hospital so severe a cyanosis developed that the attending physician and the family were alarmed. The patient was deeply cyanotic, but he did not suffer from air hunger. Hyperpnea, however, was pronounced.

Chest examination revealed an abundance of moisture throughout all the air spaces of both lungs, but there was nowhere marked dullness or any areas of bronchial breathing. The right auricle was not dilated. The cardiac signs revealed a marked enlargement of the left ventricle and a moderate enlargement of the right ventricle, with auscultatory and pulse indications of stenosis and insufficiency of the aortic valve.

On microscopic examination the sputum proved to consist of blood and plasma. The number of white cells had about the same proportion to the red cells found in normal blood. The sputum, in fact, consisted of transudated plasma with a large admixture of red cells.

The patient breathed pure oxygen through a closed system for rebreathing and after about three minutes all cyanosis disappeared without any change in the rate or amplitude of respiration. The fluoroscope was then used and this showed no area of infiltration in either lung and confirmed the dimensions of the several heart chambers as determined previously by percussion and palpation.

Three days after admission to the hospital the temperature was normal, the râles and cyanosis disappeared and the patient's heart and pulse continued to show the same physical signs as on admission.

This case was regarded as an instance of the disease described by Woillez. Had there been any hepatization of the lung, anoxemia would not have completely ceased with the breathing of pure oxygen.

MOISTURE IN CYANOSIS

Moisture may play an important part in the development of cyanosis in patients who have bronchitis, emphysema, and bronchiolar spasm. In the past we have underestimated the rôle of moisture in the production of anoxemia.

A young man who had bronchitis, with an abundance of moisture in his air spaces, had pronounced cyanosis of the lips and fingers. There was only a moderate hyperpnea. It did not amount to air hunger. There were no demonstrable areas of consolidation in the lung. The volume of the lung was very little increased, not enough, indeed, so that the pleural sinuses were filled with the lung. The patient's heart and vascular system showed no evidences of any circulatory impairment.

He breathed oxygen through the closed system and after he had inhaled it for about three minutes, all traces of cyanosis disappeared. But he breathed with exactly the same rate and volume as when he was breathing atmospheric air, and he could not detect any difference between his subjective experience when he was breathing oxygen and his experience when he was breathing air. This I conceive to be very important in estimating the functional disability in patients suffering from cardiorespiratory disease.

I am sure that in the past we have ignored the rôle of moisture in the air spaces and we have quite ignored the contribution of unrespired blood to the aortic stream. Nor has there been any attempt to point out the mechanism of this contribution in diseases of the heart and of the lung.

If, for instance, a patient with bronchitis and emphysema has cyanosis, and it can be demonstrated by inhalation of pure oxygen that the patient's anoxemia has its origin in moisture in the air spaces, then the indication for a change to a dry climate is perfectly clear. If his cyanosis is unmodified by inhalation of pure oxygen, then I think it is quite clear that the presence of moisture in the air spaces does not contribute to cyanosis and a dry climate will not improve his cyanosis.

The very simplest way in which unrespired blood can be caused to flow into the aortic stream is to stop a bronchus.

CYANOSIS INTERPRETED BY AN EXPERIMENT

About two years ago Dr. Pearce and I made a very simple experiment on a dog. A tampon of lamb's wool with a string attached was pushed down through a tracheal opening into the bronchus leading to the right lobe of the lung. This of course eliminated the right lobe from air ventilation but did not arrest the flow of blood through the pulmonary vessels. So far as the appearance of the buccal mucosa and the tongue of the animal was concerned, we could see no change in color. But during the time the tampon was in the right bronchus, blood was drawn from the femoral artery and put in test tubes under analine oil so the blood was not exposed to air. It was apparent during the period when the bronchus was blocked that the blood had a much darker color than when the bronchus was free. This was done repeatedly by withdrawing the tampon and replacing it, but at each time when the tampon was in place the aortic blood took on a venous hue. Whether the entire right bronchus or only the bronchus leading to the lower right lobe was occluded in each one of these trials was not determined, but from observations on the respiratory movement of the

lobes, we believe the lower right lobe only was eliminated from ventilation in most of our trials.

In either event it is a very simple method of demonstrating how a certain proportion of unrespired blood going to the left auricle will produce cyanosis. In our clinical experience we usually see only the phenomena for which we have an interpretation. What we cannot interpret we fail to see. As our interpretation of cyanosis is improved and enlarged, cyanosis will be much more frequently recognized in clinical experiences.

In the study of respiratory phenomena of cardiorespiratory disease, the rôle of moisture must be carefully interpreted. If we employ analyses of alveolar air for interpreting the partial pressure of oxygen and carbon dioxid in the circulating blood, such interpretations must predicate a uniform tonometric function of the entire respiratory area.

If areas of the respiratory membrane are eliminated from the respiratory function and other portions are normally functioning then it is obvious that the respired air cannot be employed to interpret the partial pressure of carbon dioxid and oxygen in the blood. When the entire respiratory membrane is normally functioning, then such interpretations are justifiable. But the moment the rôle of moisture in air spaces comes into play, or the air spaces of the lung are filled with an exudate, without any change in the flow of blood through the pulmonary vessels to the affected part, there will be unrespired blood contributed to the aortic stream, and analysis of respired air will give wholly unreliable interpretations of blood gases.

SUMMARY

The significance of the foregoing observations lies in the recognition of dissociation between anoxemia and increased carbon dioxid content of the blood.

The carbon dioxid and the oxygen content of respired air are measures of transpiration between blood and alveolar air only when the entire minute volume of blood passing through the pulmonary circulation is exposed to a respiratory membrane which has a uniform exposure to the ventilating air of the lungs. Normal subjects and patients with stenosis of the trachea or larynx will conform to these premises. So may patients who have respiratory symptoms from disturbed responses or altered control of respiratory rate and rhythm from the central or peripheral nerve supply to the lungs. But the moment the process of ventilation or blood flow is modified so that the uniformity of transpiration in the entire lung between the circulating blood and ventilating air is disturbed, then analyses of respired air cannot be employed to interpret the carbon dioxid and oxygen content of the blood.

ABSTRACT OF DISCUSSION

DR. ARTHUR D. HIRSCHFELDER, Minneapolis: I would like to ask Dr. Hoover whether sodium bicarbonate would be of value in such cases, at least in some of the border-line cases?

DR. CHARLES F. HOOVER, Cleveland: It is the same with all irritant gases; all the acids produce exactly the same thing. What is your idea of the alkali?

DR. HIRSCHFELDER: You could take care of a certain amount of the carbon dioxid excreted from the kidneys by continuous intravenous infusion of sodium bicarbonate. It seems to me there might be some border-line cases in which that might turn the tide.

DR. CHARLES F. HOOVER, Cleveland: I doubt if you could do very much in the way of luring it off in the form of a carbon through the kidneys. There are not any border-line cases.