intestinal excitation occurred 10 seconds later. At the same time, the flow through the loop was increased from 56 drops to 76 drops per minutes.

Discussion

1) Sakurazawa et al (6) observed, mainly, the increase in tone by the stimulation of sinus nerve, but in the present work, both increases in tone and movements were always observed by the stimulation of the sinus and depressor nerves.

2) Contrary to the works of Funakoshi (3) and Suenaga et al (9), intestinal excitation was observed, when marked fall in blood pressure was obtained by the stimulation of sinus nerve and depressor nerve, regardless of the severance of the splanchnic nerves (see Fig. 1). The same effects were also observed after the bilateral vagotomy and ligature of the suprarenal veins.

3) Intestinal excitation was obtained on the denervated loop which was connected to the body through the blood vessels only (see Fig. 2). This fact shows that the mechanism of intestinal excitation is a secondary phenomenon due to the body circulation. However, it seems to me, on the contrary to the thought of Bayliss (2), that the cause of intestinal excitation is not the fall of blood pressure nor vascular dilatation. It was already demonstrated by some workers (1, 4) that the rate of epinephrine liberation from the suprarenal glands reduced during the stimulation of the sinus nerve or depressor nerve. And so, the cause of intestinal excitation can be easily explained from the standpoint of the chemical mediator of the cholinergic nerves.

4) The intestinal excitation was also obtained on the perfused loop of which the nervous supplies were intact (see Fig. 3), therefore, the direct nervous

control to the small intestine also can not be denied. This is in accordance with the conclusion of Tournade & Malméjac (10).

Conclusion

In dogs under urethane, stimulation of the carotid sinus nerve and depressor nerve causes a definite increase in intestinal tone and motility. It is not abolished by the bilateral vagotomy, splanchnicotomy and ligature of suprarenal veins. This excitation was obtained on the denervated loop which maintained normal circulation, and it was also obtained on the perfused loop of which the nervous supplies were intact. Therefore, it can be concluded that this mechanism is due to, on the one hand, a secondary effect from the blood flow, and the other hand, to the direct neurogenic control to the loop.

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From the Dainippon-Zōki Institute for Medical Research

The Pulmonary Artery Reflex Test and its Clinical Applications

By

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The cessation of breathing after the deepest possible inspiration causes bradycardia and the fall of blood pressure. Our investigations made clear that this phenomenon is caused reflectively by

stimulating the receptor area in the pulmonary artery as determined by Takino¹⁾²⁾ in 1933. Therefore, I proposed that this reflex be called the pulmonary artery reflex.

According to our clinical and experimental investigations, this reflex is very useful not only for the explanation of the symptoms which occur in the pulmonary circulation, for instance pulmonary embolism, but also for judging the condition of the autonomic nervous system of human beings.

However, up to the present the pulmonary artery reflex test has generally been used very little as compared with the carotid sinus reflex and Aschner's oculo-cardiac reflex. This fact is perhaps due to an insufficient recognition or understanding of the histological and physiological experiments with the pulmonary artery in animals and human beings. This misunderstanding is especially due to the incorrect application of the results of animal investigations to the analysis of human beings. Therefore, I shall at this point describe the nature of the investigations about the pulmonary artery reflex that have been tried up to the present.

Many years ago it was already known that the expansion and contraction of the lungs in the course of respiration influences the action of the heart (Saafeld³), Hering⁴), Brodie and Russel⁵) etc.). The reason for this influence however, had not been made clear by those investigators, although various hypotheses or experiments had been offered as an explanation.

Furthermore, the experimental and clinical observations on the pulmonary embolism which have been reported by many authors caused us to assume the possibility of some neuro-reflex from the lungs.

In 1935 Schwegk⁶) discovered that bradycardia and the fall of blood pressure in the general circulation can be caused by raising the blood pressure of the pulmonary circulation in the isolated lunglobe of the dogs. This phenomenon disappears when the n. vagus is cut off. So he called this phenomenon "Lungenentlastungsreflex" (lung discharging reflex). This fact shows beyond doubt that the receptor area such as the carotid sinus and the aortic arc, is to be found somewhere in the lungs. His methods of experimenting with animals, however, were not decisive enough for determining the location of the receptor area in question. For in his experiments he raised the blood pressure in the entire area of pulmonary circulation viz., the pul-

monary artery and the vein and the capillary. So, in order to explain his results, he made use of the morphologic data which had been reported by many authors such as Schumacher, Perman, Tello⁷), Micheluzzi⁸) and Mueller⁹). Indeed, they found some nerve endings of sensory type in the wall of the pulmonary artery. However, their observations were fragmentary and are not sufficient, not only for explaining Schwegk's data, but also for determining the source of the reflex in question in the lungs. Therefore, he referred chiefly to my histological investigations on the receptor area of the pulmonary artery in 1933, when he published his interesting physiological investigation on the reflex.

According to my investigations, on human beings and animals (cows, pigs, rats, guinea-pigs, rabbits, dogs, cats and pigeons) the distributions of the said receptors in the pulmonary arteries are very different between human beings and these animals (Fig. 1). In dogs, cats, guinea-pigs, rats and rab-

Fig. 1. Receptor Area in the Aortic Arc and Pulmonary Artery
V: Right and left vagi,
D: Right and left depressor
The receptor areas are thin black colored.

bits, the receptor is distributed from the root of the Botall's duct (ligamentum arteriosum) in the trunk of the pulmonary artery to the vicinity of the pulmonary hilus. In human beings, cows, pigs and pigeons, however, the receptor is distributed from the root of the ligamentum arteriosum in the trunk of the pulmonary artery to a distant part of the artery where its diameter seldom exceeds 400

Foot note:

M. Takino and Y. Hayashi: According to our investigations, inflating the lungs of the cats by means of a syringe not only causes the fall of blood pressure, but reflectively also strengthens the peristalsis of the small intestine. This strengthening of the peristalsis disappears after cutting both the vagi.

micron. In short, the receptor area is by far the wider in human beings, pigs, cows and pigeons than in dogs, cats, rabbits, guinea-pigs and rats. This difference in the distribution of the receptor area is the most decisive element for determining the importance of the pulmonary artery reflex in human beings, pigs and cows.

It is interesting to compare the pulmonary artery with the carotid sinus and the aortic arc. As is well known, the distributions of the receptor in the carotid sinus and the aortic arc are much the same among all animals except for the ruminant. In the carotid sinus and the aortic arc the receptors crowd thickly into a narrow part. On the contrary, the distribution of the receptor in the pulmonary artery is very different among animals. In the pulmonary artery the receptors distribute widely and sparsely (Fig. 2). This histological knowledge

Fig. 2. Sensible Endings in the Pulmonary Artery of Human Embryo (Female, age 9 months) near the Pulmonary Hilus.
E: Circular Ending
R: Reticular Ending

is very important for right operation of the physiological examination of this reflex.

Frankly speaking, many physiologists did not have sufficient understanding of the above-mentioned histological characteristics in respect to the receptor area of the pulmonary artery. This is perhaps the reason why their results¹⁰⁾¹¹⁾ were not always the same.

In order to decide the physiological meaning of the receptor area in the pulmonary artery I examined at first rabbits in cooperation with S. Watan-

abe¹²⁾ and T. Hotta¹³⁾, and then cats with Y. Hayashi¹⁴⁾. Our investigations differ from those of Shwiegk in that we operated on the animals in order to hold physiological examination, especially keeping in consideration the difference between the distribution of the receptor among different animals. Our investigations are the first in which results of the physiological examination agreed completely with the histological examination relative to the subject of the pulmonary artery. As the details of this investigation¹²⁾¹³⁾ have been published in 1937 and 1943, I shall not repeat them here (Compare with other authors).

In 1947 Parin¹⁰⁾ verified this reflex, too. However, as he mainly followed Schwiegk's method, he could not determine what part of the lung is the source of this reflex. In order to explain his data, he chiefly made use of Schemetkin's morphologic data reported by Dogel¹⁵⁾ in 1898. However, they did not describe the pattern of the sensory endings or their area, so far as it concerns the pulmonary artery.

De Burgh Daly¹⁶⁾ assumed that this reflex originates in the sensory endings in the pulmonary veins which was first found by me in 1933, and later by Nonidez in 1937. Contrary to Daly's opinion Parin regards the venous part of the pulmonary vascular tree as a possible source or origin of reflexes not of a depressor but of a pressor nature such as Bainbridge's reflex.

In 1951 Schwiegk¹¹⁾ reported his further examination on pulmonary cardiac reflex again, but again without a decision about the source of this reflex. Wigger¹⁸⁾ added my investigations on pulmonary blood vessels to his handbook. However, he quoted them wrongly, because he described that this reflex in question originates in the pulmonary veins. In 1943 at the forty first Annual Meeting of Internal medicine at Tokyo the pulmonary veno-cardiac reflex was first published by me and my coworker in Japanese. Later, in 1948 at the forty sixth Annual Meeting of Internal Medicine at Tokyo, this reflex was reported by Okinaka¹⁹⁾ as appearing in dogs. At that time we discussed this problem together with S. Saito. Saito reported that the sensory receptors are greater in number in the back wall of the left atrium of human beings and ganglion cells of unipolar nature (sensible) which I²⁰⁾ have already found in the wall of the extrapulmonary lung vein of human beings in 1937. From the

above-mentioned it is certain that the important part of the receptor area of the pulmonary veno-cardiac reflex is not in the intrapulmonary veins, but in the extrapulmonary veins and their vicinity in the left atrium.

After the second world war the outline of our investigations on the pulmonary veno-cardiac reflex²¹⁾ have been published in English in 1951.

As has been already stated, the pulmonary artery reflex is caused by the cessation of breathing after the deepest inspiration. This test, however, stimulates not only the pulmonary artery wall, but also the bronchus, the bronchia, the visceral and parietal pleura at the same time. That is to say, the bronchus and the bronchia is stimulated by the increased pressure within them, while the visceral and parietal pleura are simultaneously stimulated by their expansion and the increased pressure in the pleural cavity. Therefore, the technique of this test needs further examination if it is to be used for the bringing forth of the said reflex. In other words, it is extremely necessary to decide whether bradycardia and the fall of blood pressure caused by this test are influenced by the stimulation of the said tissues other than the pulmonary artery. This stimulation is because of the many sensory endings in the bronchus and the parietal pleura.

Here I shall first explain the relation between the pulmonary artery reflex and other reflexes like the cough reflex, secondly, the clinical symptoms such as syncope and asthmatic attack, caused by parietal pleura reflex, thirdly the comparison of the pulmonary artery reflex test with Valsalva's test.

Method

As to the experimental methods by which we operated on the animals for the physiological or histo-physiological determination of the receptor area in the pulmonary artery I have already described them minutely in the article entitled "Lungenarterienstammreflex" in 1943. Therefore, they are omitted here.

Clinically the change of pulse rate may be roughly judged by the palpation of radial pulse, that of respiration by inspection. For an exact examination, in other words, one which avoids the subjectivity of the investigator, however, it is absolutely necessary to measure the pulse rate and respiration by some instrument.

For the purpose of measuring the former, sphy-

gmometer is used. However, in order to observe the relation between pulse rate and/or blood pressure and respiration, I devised the following simple apparatus with Dr. Nishimura:

For this purpose, I made use of glass U-tube with the proper quantity of mercury in it, a glass bottle with three openings and the india rubber band of sphygmomanometer (Riva Rocci's) and an air-pump (Fig. 3). One of the openings of the

Fig. 3. Apparatus
K: Kymograph
U: U-Glass tube
A: Air-pump

glass-bottle is connected with the one end of the U-glass tube and another opening with the india-rubber band by the india-rubber tube. One third is connected to the delivery side of an air-pump. After the upper arm is bound with an india-rubber band, the air is sent by the air-pump. Then the researcher is required to search for a point of pressure at which the surface of mercury in the U-tube can be pulsated most strongly.

A light lever is put on the surface of the mercury (in the vertical part of the U-tube not connected with the glass bottle).

In recording respiration, a rubber apparatus connected by a rubber tube with Marey's tambour as improved by Dr. H. Yamasaki is inserted in the nostril. If the nose is stuffy, a gasmask with two openings is used.

Results and Discussion

Electric stimulation on the inside wall of the trachea of non-narcotized or shallow narcotized rabbits caused often coughing (Fig. 4). In this case,

Fig. 4. The Relation of Cough Reflex to Electric Stimulation of the Trachea, the Bronchus and the Bifurcation part of the Shallow Narcotized (urethane) Rabbit (Female, Bodyweight 2.65kg).

D: Duration of Stimulation

T: Time in 5 Sec.

B P.: Carotid Blood Pressure

1. Stimulation of the trachea at the entrance to the chest (coil-distance 10cm-5cm)

2. Stimulation of trachea in the chest (coil-distance 10cm-5cm)

3. Stimulation of the trachea very near bifurcation part (coil-distance 16cm-5cm)

4. Stimulation of the right bronchus (coil-distance 5cm)

5. Stimulation of the left bronchus (coil-distance 10cm-5cm)

6. Stimulation of the left bronchus (coil-distance 10cm-5cm) after cutting the left depressor.

Fig. 5. Electric Stimulation of the Bifurcation-Part of the Trachea and the 1. Bronchus of a strongly Narcotized (Urethane) Rabbit (Female, Bodyweight 2.1kg)

B. P.: Carotid Blood pressure

D: Duration of Stimulation

1: Stimulation of the bifurcation part of the trachea (coil distance 5cm-0cm)

2: Stimulation of the 1. bronchus (coil-distance 5cm-0cm)

however, the changes of blood pressure and pulse rate were too slight to be ascribed to the (aortic) depressor reflex. Only when the electrode reached the inside wall, not of the right bronchus, but of the left bronchus, where the electrode got very near to the aortic arc and the root of Botallo's duct (ligamentum arteriosum), did bradycardia and the fall of blood pressure suddenly result.

This phenomenon is perhaps due to the passage of electric current to the aortic arc and the root of Botallo's duct (ligamentum arteriosum), namely the receptor area of the left (aortic) depressor nerve: for, when the left depressor nerve is cut off, the said phenomenon either becomes very slight or

disappears almost completely (Fig. 5). Needless to say, mechanical stimulation at the same spot does not cause any such change of the blood pressure and pulse rate. Furthermore, due to the absence of sensory nerve endings which are related to pain sense in the bronchus, it may be readily assumed that in human beings also, stimulating the inside wall of bronchus and bronchia does not cause such a reflex as Aschner's oculo-cardiac reflex: for generally only sensory nerve ending which is related to pain sense can have an influence upon the heart, the degree and manner of the influences varying with the individual.

On the other hand, even if the trachea and

Fig. 6. The Comparison of the Pulmonary Artery Reflex by the Inflating the Lung by means of a Syringe before and after Narcotizing the Bronchus and the Trachea (by cocain-application) of the well Urethane-Narcotised Rabbit (Female, Bodyweight 2.18kg)

D: Duration of inflating the lungs by means of a Syringe (3-7cm)

B. P.: Carotid Blood Pressure

L. P.: Pressure of inflating the lungs

The fall of blood pressure caused by inflating the lungs is weaker after cutting both the right and left vagi than before cutting them. This relation is also verified even when the trachea and the bronchus are anesthetized by cocain-application.

bronchus are anaesthetized by cocain-inhalation or cocain-application, inflating the lungs of the rabbits by means of a syringe (3-7 cc) does cause the pulmonary artery reflex (Takino and Hayashi). In this experiment, however, the pulmonary artery reflex is often more or less decreased, for cocain-inhalation (0.5%) or cocain-application (1%) either first increases and then decreases the blood pressure or from the beginning decreases it according to the quantity applied (Fig 6).

In the visceral pleura, there are only a few sensory endings. So it is a matter of course that any stimulation on this pleura exclusive of that of the pulmonary hilus, does not cause any change in the blood pressure and respiration (Hotta and Takino). However, although stimulation on the said pleura at the pulmonary hilus caused a slight fall of blood pressure, this should not be ascribed to visceral pleura reflex, because there are nervous bundles of vagi near the pulmonary hilus and they may be stimulated by the passage of electric current (Fig. 7).

However, on the contrary, in the case of the parietal pleura there are very many sensory endings of various types such as Vater Pacini corpuscles, Golgi-Mazzoni corpuscles and so on. As is well known, a stimulation on this part is felt as keenly as it would be in the dentis pulpa or the eyeball.

Fig. 7. Effect of Electric Stimulation of Visceral Pleura of Rabbit (Male, Bodyweight 2.0kg)

B. P.: Blood Pressure

R: Respiration

T: Time in 5 sec.

Stim. 1: Stimulation of the pleura at the edge of the left lung

Stim. 2: Stimulation of the pleura at the pulmonary hilus

It is very interesting that the sense of this pleura is the same as that of the tooth in that both have no sense of heat. Stimulating this pleura by some instrument or disease, dry pleurisy for instance, causes the cough reflex.

Electric stimulation on this pleura, however, in the narcotized rabbits causes the increase of amplitude of respiration, bradycardia and the fall of blood pressure (Fig. 8). We must recognize that in this pleura there are no nerve endings related to the regulation of the blood circulation. So the

Fig. 8. Effect of Electric Stimulation of Parietal Pleura of Rabbit (Male, Bodyweight 2.1kg.)
P: Pulse-rate
BP: Carotid Blood Pressure
R: Respiration
T: Time in 5 sec.
Stim: Stimulation

said phenomenon is perhaps to be ascribed to the stimulation on the sensory nerve of the pain sense. I wish to call your attention to the fact that the said reflective changes of blood circulation and respiration by stimulating the parietal pleura, is of great significance for the explanation of various clinical symptoms such as shock, syncope, asthmatic attack and so on, which occur in some cases of the therapeutical manipulation of this pleura. The reason for these phenomena follows:

In persons under pathologico-physiological or pathological condition, especially in those with innate abnormality of the heart-vagus and the lung-vagus, stimulating the sensory nerve endings which are related to the cough reflex causes bronchospasm reflectively, and stimulating the sensory nerve endings concerning the pain sense causes such a reflex as Aschner's oculo-cardiac reflex.

For instance, as was noticed by G. Kusunoki and myself, the asthmatic attack is caused by

pneumothorax in some cases of pulmonary tuberculosis. Furthermore, in some cases of pulmonary empyema with the vagotonia of the heart syncope was seen when the pleura cavity was washed.

From these facts it will be easily understood that in the course of this treatment the influence upon this reflex by stimulation on the parietal pleura should be carefully examined.

According to our investigations of patients with dry pleurisy or with pneumothorax, the result of the stimulation of this pleura by the technique of the pulmonary artery reflex generally extends only to the sensory endings of the cough reflex. For it is almost impossible to stimulate, by this technique, the sensory endings concerning the pain sense, even in such persons with these said diseases. So, we may safely state here that generally bradycardia and the fall of blood pressure caused by this technique is not influenced even by stimulating the bronchus and parietal pleura.

Here I shall describe Sylla's experience. According to his roentgenological observation of human beings, bradycardia appears by stopping the breathing after the deepest inspiration. From this fact we may say that Sylla²²⁾ is the first to apply the technique of the test of pulmonary artery reflex to the human bodies. He has attributed this phenomenon, however, to the traction of the sino-auricular node caused by a lowering of the diaphragm following this test. However, I have observed in my patients with lung emphysema or phrenic avulsion, that bradycardia has nothing to do with the location of the diaphragm (Fig. 9 A. and B). If the diaphragm is very high, even after stopping the breathing, bradycardia is weak, because this location of the diaphragm prevents an expansion of the lungs which would be adequate to cause a remarkable

Fig. 9. The Pulmonary Artery Reflex Test in the Case of right Pulmonary Tuberculosis (Female, Age 30)
A. Before the right Phrenico-exauresis
B. After the right Phrenico-exauresis

Fig. 10 The Pulmonary Artery Reflex in the Case of severe pulmonary emphysema (Male, Age 24)

Fig. 11 The Comparison of the Pulmonary Artery Reflex Test and Valsalva's Test (A. B. C.)

increase of blood pressure in the pulmonary artery, namely, the pulmonary artery reflex. On the contrary, when the diaphragm is lowered by lung emphysema, this lowering does not generally seem to strengthen the pulmonary artery reflex (Fig. 10).

Finally I shall compare the test of the pulmonary artery reflex with Valsalva's test. The difference between the tests is that in the latter the cessation of breathing after the deepest inspiration is followed by the posture of expiration with mental and muscular straining.

It goes without saying that in the Valsalva's test the increase of intraalveolar pressure is seen more strongly than in the test of the pulmonary artery reflex. This stronger increase of the intra-alveolar pressure strengthens the resistance of the alveolar capillaries and leads to the increase of blood pressure of the pulmonary artery.

Judging from this fact alone, Valsalva's test produces better conditions for the study of the pulmonary artery reflex. This presumption has been verified in the persons with strong vagotonia of the heart. In such persons the bradycardia and the fall of blood pressure appear often more strongly in the cases of Valsalva's test than in that of the pulmonary artery reflex test. (Fig. 11 A)

Valsalva's test, however, affects not only the pulmonary artery, but also other organs, especially the sympathetic of the heart. For taking the posture of expiration with straining after the deepest inspiration causes some degree of excitement of the sympathetic, especially in the persons with sympathicotonia. And it is probable that in this case, Bainbridge's reflex is caused by the increase of blood pressure in the right atrium more strongly than in the case of the pulmonary artery reflex test;

and, that the decrease of blood pressure (in the aortic arc and carotid sinus) is caused by the blocking of the flow of blood to the lung and right heart, this decrease of the blood pressure acts upon and excites the heart-sympathicus more strongly in the former than in the latter. Such changes of the heart-sympathicus, namely the decrease of the heart-vagus or the increase of the heart-sympathicus weakens more or less, the irradiation of the pulmonary artery reflex to the heart. This is why bradycardia in some cases is not as apparent in Valsalva's test as in the test of the pulmonary artery reflex. (Fig. 11. B). However, the fall of the blood pressure generally is not weakened by Valsalva's test, but even strengthened thereby, because of the blocking of the flow of blood to the lung and right heart is stronger in this test than in the pulmonary artery reflex test.

Attention should be called to the fact that in spite of the decrease of blood pressure in the carotid sinus and aortic arc and the increase of blood pressure in the right atrium, which inevitably accompanies these tests, the increase in the blood pressure and pulse rate is not noticeable in the first moments of each test. Of course, in these tests the receptor areas of cardiac reflexes are stimulated either almost simultaneously or after another. However, on the basis of the above-mentioned facts it may be reasonable supposed that the area which is stimulated first and most strongly from among the above mentioned areas, takes the lead in causing the phenomenon that follows.

As the examinee continues to hold his breath, in both the Valsalva's and the pulmonary artery reflex test, the bradycardia and the fall of blood pressure (which always occur at least in the first period of the tests, and reach a minimum) generally return approximately to the original blood pressure and pulse rate and in some cases the pulse rate even exceeds the original one (Fig. 11 B. C). This rise of the blood pressure and pulse rate are often more strongly evident and observed earlier in persons with sympathicotonia than with parasympathicotonia. The degree of the above changes vary with the day, the mental state of the examinee and so on.

On the basis of the above facts, it follows naturally that the comparison of the changes caused in blood circulation by these two tests can be of much use for the diagnosis of the autonomic nervous

system of human beings. However, as to the nature of the changes in the blood circulation of the cardiac diseases cases which may be caused by these tests, we must continue our investigations. As is already stated, the tension or excitability of the heart-vagus is often heightened in the case of cardiac diseases. In this condition bradycardia is often apparent in both these tests, even in people with sympathicotonia. Which of these two tests causes bradycardia and the fall of blood pressure more strongly depends upon the difference between the tensions or excitabilities of the sympathicus and those of the heart-vagus.

Recently Dr. Y. Takenaka²³⁾ conducted minute investigations relative to the changes of pulse and heart beat in the so-called breath holding spells which occurred in 30 cases of children (from four months to age 4).

According to his report (in Japanese) and his private letters to me, in these cases, not only syncope and convulsion, but also the changes of pulse, such as weak or small pulse or complete disappearance, and the changes of heart beat such as bradycardia and standstill of heart beat were observed. He explained the latter phenomenon by the pulmonary artery reflex, because he thought that the mechanism of breath holding spells of children accompanied by syncope and convulsion, is very similar to that of Valsalva's test and therefore, it causes a remarkable increase of blood pressure in the pulmonary artery.

However, breath holding spells do not appear in all children. Therefore, this phenomenon seems to be closely related to some factors of constitution or disposition. As I have already stated in my monograph, the tension of the heartvagus is very high especially in children with spastic constitution. This is the reason why the cramp of the skeletal muscles, above all that of laryngeal muscles and the above-mentioned change of heart beat occur either almost simultaneously or one immediately after the other.

Summary

1. The pulmonary artery reflex test, that is, the cessation of breathing after the deepest possible inhalation results reflectively in bradycardia and the fall of blood pressure. According to our investigation these changes are due to high blood pressure in the pulmonary artery caused by the resistance of alveolar capillaries, strengthened by

the expansion of the alveolar wall, and by the increase of the alveolar pressure that follows the deepest possible inhalation.

2. This test, however, stimulates not only the pulmonary artery wall, but also the bronchus, the bronchia and visceral and parietal pleura at the same time. However, we made clear that the bradycardia and the fall of blood pressure caused by this test is practically unaffected when the above said tissues other than the pulmonary artery, are stimulated.

3. The degree of the said changes of pulse rate and blood pressure caused by this test differs among persons. Generally the bradycardia and the fall of blood pressure caused thereby appear more strongly in the persons with vagotonia (parasympathicotonia) than in those with sympathicotonia. In the same persons they vary with the day, their mental state and so on.

4. This test is very useful not only for judging the condition of the autonomic nervous system of human beings, but also for the explanation of the various symptoms which occur in the pulmonary circulation, for instance pulmonary embolism, breath holding spells of children and so on.

5. The comparison of the pulmonary artery reflex test with Valsalva's test and their clinical applications have been discussed in this article.

6. The parietal pleura has many sensory endings of various types, although they are generally different from those in the pulmonary artery, the carotid sinus and the aortic arc.

Although relatively infrequent, asthmatic attacks do occur while treating patients suffering from tubercular disease, with pneumothorax. This phenomenon is due to the stimulation of the parietal pleura by pneumothorax and the resultant parietal pleura reflex, but perhaps it only occurs in patients with a easily excitable lung-vagus.

Furthermore, the shock and syncope rarely occur during the therapeutical manipulation of this pleura, for instance washing the pleura of the patients suffering from the empyema of chest, with the physiological saline solution. But when shock and syncope do occur in such situations, this phenomenon is also due to the parietal pleura reflex

being caused by the stimulation of the said pleura by the manipulation, but perhaps only in persons with easily excitable heartvagus.

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