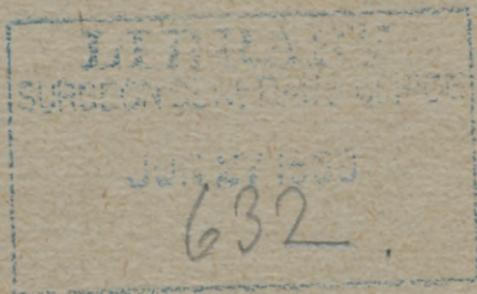


VAN SANTVOORD (R.)

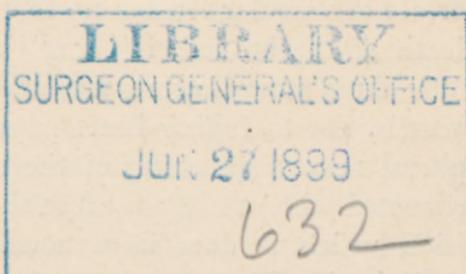
THE STATE OF THE VASOMOTORS
IN ACUTE LOBAR PNEUMONIA,
AND ITS BEARING ON TREAT-
MENT.

BY
R. VAN SANTVOORD, M.D.

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THE STATE OF THE VASOMOTORS
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BY R. VAN SANTVOORD, M. D.

IN the *Berliner klinische Wochenschrift* of 1895, Nos. 51 and 52, Romberg published a series of investigations undertaken to determine what parts respectively the state of the heart and that of the vasomotors take in the condition commonly regarded as weak heart in acute infectious diseases. He experimented upon rabbits with the *Bacillus pyocyaneus* and with the pneumococcus of Fränkel. In a second paper read before the Fourteenth German Congress for Internal Medicine, Päsler and Romberg presented a further communication on the same subject, the Loeffler bacillus being on this occasion the organism experimented with.

It was found that the effect of infection by any one of these agents was to increase markedly the amplitude of the pulse tracing and to lower arterial tension. The effect of abdominal massage, which mechanically emp-

* Read before the New York Academy of Medicine, April 21, 1898.

ties the abdominal vessels, was always to at once cause a marked increase of vascular tension up to a short time before the death of the animal, it being thus demonstrated that the circulatory failure was due to dilatation of the vessels, not to failing heart power. Irritation of peripheral nerves (pinching of the nose of the animal experimented with) failed to cause the increased vascular tension which it does in a normal subject. As this reflex irritation has been demonstrated to act through the medullary vasomotor centres, this failure was regarded as proving that these centres were paralyzed. Asphyxia, which acts upon both the spinal and medullary centres, caused a slight elevation of pressure, the relative intactness of the former being thus shown. Chloride of barium, which has been proved to act on the peripheral vasomotors, caused a marked elevation of pressure. The final conclusion arrived at from these experiments was that in cases of infection from the pneumococcus, *Bacillus pyocyaneus*, and the Loeffler bacillus the circulatory embarrassment heretofore attributed to cardiac weakness produced by toxæmia and malnutrition is due mainly to vasomotor paralysis. This conclusion did not apply to the late circulatory disturbances in diphtheria, which have been demonstrated to be due to cardiac lesions, the circulation in the acute stage of the disease only being considered.

During the past winter I have been studying cases of pneumonia as they presented themselves in hospital and private practice with these conclusions in mind, in order to determine if possible what applicability they have to pneumococcus infection in the human being. Sphygmographic tracings were taken in eighteen cases of the disease. As these records confirm the results of obser-

vations made by many other observers in like cases, they may safely be accepted as the basis for the following remarks.

The tracings were almost invariably of great amplitude, the primary wave being generally sharply pointed, without any indication or only a faint indication of the tidal wave. The dicrotic wave was often very pronounced, sometimes not very strongly indicated. These characteristics were strongly developed in all cases in which constitutional symptoms were very marked until a few hours before death in fatal cases, when the pulse would become too feeble to give a good tracing, or convalescence. They were also pronounced in some cases in which the disease ran an only moderately severe course, judged by the general condition of the patient. In a few mild cases the deviation from normal was not very great. The pulse, as a rule, was large, often bounding, but usually of less than average tension, as judged by the finger, in the severe cases very much below the average. These latter observations were confirmed in several instances by the use of the sphygmomanometer of von Basch. This instrument measures the amount of pressure necessary to compress the pulse so that it can not be felt beyond the point of pressure. It is a measure, therefore, of the tension that exists at the period corresponding to the apex of the pulse tracing. The tracings demonstrate the fact that the arterial pressure falls with great rapidity after this primary wave, so that we have proof that the mean arterial tension is often low. It is the commonly accepted explanation of this form of tracing that it is due to dilatation of the peripheral vessels. We have the fact demonstrated, therefore, that in pneumonia, as it exists in the human subject, we have

the same condition of low arterial tension due to relaxed peripheral vessels as was found to exist experimentally in animals infected by the same coccus.

The one constant factor in the circulation of the blood is gravity. When the general arterial tension is lessened, either as a result of cardiac weakness or of excessive dilatation of the peripheral vessels, this factor increases in its relative importance and the blood tends to accumulate in dependent portions of the body at the expense of the more elevated. Owing to the greater ease experienced by dyspnoic individuals in a position with the head and shoulders raised, this is the attitude which a patient gravely ill with pneumonia assumes, and this position is obviously directly provocative of cerebral anæmia. Here we have a mechanical factor which is probably of considerable importance in causing the nervous manifestations of the disease and in determining its final outcome.

The condition of the peripheral vessels which makes the least demand upon the heart in order to secure an adequate capillary pressure throughout the body is one of medium contraction. When the peripheral arteries are strongly contracted, as in cases of renal cirrhosis, the ventricle is forced to hypertrophy in order to overcome the obstruction, and toward the last we have the striking combination of ringing heart sounds and tense arteries with the dyspnoea and swollen feet indicative of inadequate capillary circulation. On the other hand, when the peripheral vessels are greatly relaxed, the capacity of the arterial system is largely increased. If the heart continues to inject into the aorta only the amount of blood in a unit of time that was adequate to maintain the tension at a safe level, under normal conditions, the

result must be slowing of the current, lessened friction, hence lessened tension, which decrease may be sufficient to dangerously impair the circulation in the organs at a higher level than the heart. This danger may be avoided by the injection by the heart into the aorta of a larger quantity of blood in a unit of time—*i. e.*, by increased rapidity of contraction, or by the discharge of a larger amount of blood with each systole. When, finally, extreme dilatation takes place, this compensation becomes impossible. The heart is imperfectly supplied with blood, it falters and stops.

This sequence of events Romberg declares takes place as a matter of observation in his infected rabbits. At first, although the increased oscillation of the tracing showed a general relaxation of peripheral vessels (his measurements were taken in the carotid), the average tension did not fall. Later, when this did fall, the heart still responded when abdominal massage was employed, showing that the decline was due, not to failing heart power, but to increased relaxation of the vessels. Finally this response ceased and the animal died.

It is often stated in the books (Wagner's *Pathology*, for instance) that the characteristic pulse of pneumonia is one of high tension. In the limited number of cases that I examined with this particular question in view this was certainly not the case, though the variation was very considerable in different cases, those of very low tension corresponding to great rapidity of heart action and grave constitutional conditions. The bounding character of the pulse due to the wide variations in tension during each cardiac cycle and its fullness give it a deceptive feeling of vigor which may readily be mistaken for high tension. The number of cases exam-

ined by me for the purpose was not sufficient to establish the rule, if there is a rule. There is, unfortunately, no exact method of determining clinically the exact tension of the arteries. From the foregoing, however, it is obvious that if in a given case of pneumonia the tension approaches or exceeds the normal, it can be only by reason of increased exertion on the part of the heart. In extreme vascular relaxation, therefore, we have a mechanical factor which renders necessary increased exertion on the part of the heart, in order to adequately supply all the organs with blood, and the ability of the heart to respond to this demand may determine the outcome of

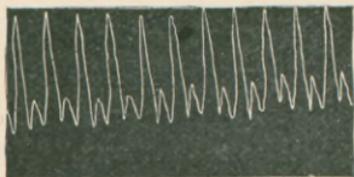


FIG. 1.—Alcoholic, aged fifty-one. Pulse 153. Two hours before death.

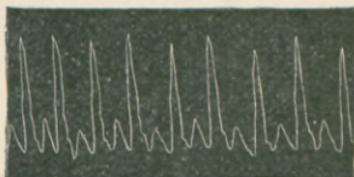


FIG. 2 —Boy, aged sixteen. Never very ill.

the given case. I present here two sphygmograms which are almost identical—one taken about twenty-four hours before death from a case of pneumonia with tremor and delirium and very low arterial tension in a middle-aged alcoholic, the other from a boy of sixteen, who not only recovered but never appeared to be gravely ill. The difference in the outcome of the two cases was probably largely influenced by the difference in the ability of the hearts to respond to the increased demands upon them.

The weak point in the transfer of the results of experiments upon rabbits with the pneumococcus to the human being is that in the former the infection produces only a septicæmia, without the pulmonary lesions which

form the prominent feature in the latter. Nevertheless, the animal experiments shed light, I think, not only on those cases in which the toxæmia is the prominent symptom, but also on the frequent cases in which the pulmonary infiltration plays a prominent part, of which the following is a type: A previously healthy and vigorous man was admitted into the Harlem Hospital with an infiltration, which finally involved the whole right lung. Dyspnoea was marked, and became gradually worse, until in three days he developed œdema of the left lung and died. Nervous symptoms were not marked. The sclera were yellow. The second sound over the pulmonary valves was louder than over the aortic. The tracing of his pulse, taken the day before his death, is here shown.

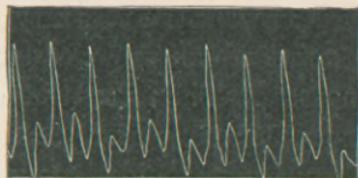


FIG. 3.—Extensive pulmonary infiltration. Marked dyspnoea.

The autopsy disclosed the following conditions so often seen in death from pneumonia: The cavities of the right heart contained much yellow clot and fluid blood and were dilated. The left ventricle was relaxed, but contained little clot or fluid blood. The right lung was in a condition of red hepatization throughout; the left congested, œdematous, and coated externally with fibrinous exudate. The liver was congested. What causes this obvious blocking of the right heart? Direct observation shows that when a living tissue is irritated the effect is to cause, first, a dilatation of the vessels, with sometimes a temporary acceleration of the blood stream. Then occurs a slowing of the latter amounting, when the irritation is intense, to complete stasis. To trans-

late the words of Cohnheim, in speaking of this phenomenon: "As the driving force and the blood itself have undergone no change, the local retardation of the blood current can result only from local resistance; the lumen of the vessels, however, does not offer this in any locality; on the contrary, arteries, veins, and capillaries are universally relaxed—they are even dilated above the normal. Therefore there remains, so far as I can see, no other possibility than that the increased resistance is to be sought in the changed relations of friction and adhesion between the blood and the walls of the vessels." This sort of resistance, therefore, doubtless is offered to the circulation of the blood in the hepatized lung, as is generally recognized; the pulmonary exudate, by interfering with the normal dilatability of the vessels, being an additional factor. How are we to explain the œdema of the unhepatized lung? The mere narrowing of the blood current by the more or less complete shutting out of the vessels of the solidified lung does not explain it, because we frequently see patients with one lung solidified who nevertheless exhibit no marked signs of respiratory or circulatory distress. Welch long ago demonstrated that the only way in which œdema of the lungs could be produced mechanically in the laboratory was by causing relative insufficiency of the left heart, so that the sufficient right heart keeps on pumping blood into the lungs which the deficient left is not able wholly to receive and forward, a mechanism which is exquisitely exemplified in many cases of chronic interstitial nephritis. Extensive ligation of pulmonary arteries never produced œdema in the portion of lung supplied by the unobstructed branches.

The conditions here are exactly the reverse. We

have the left ventricle forcing blood through the abnormally relaxed greater circulation and the right ventricle forcing the blood through an abnormally contracted lesser circulation, so that in this instance the strain is upon the right ventricle, which, if it proves insufficient, is not able to dispose of the blood brought to it by way of the greater circulation, with the result that congestive phenomena in the abdominal organs, most markedly in the liver, as in the above case, are to be noted. In the lungs we would look for nothing except possibly some hypostatic congestion in the portions not inflamed, as the narrow point past which the right heart must exert itself to force the blood is the pulmonary arterial system, not the wider capillaries and veins. Yet this increased strain upon the right heart is in part due to the greater amount of work thrown upon the organ as a whole by the necessity of maintaining the equilibrium of the circulation which is threatened by the paralysis of the vasomotors. Congestion, other than hypostatic, and œdema of the lungs occurring under these circumstances can not be due to any mechanical cause. It can only be inflammatory, and the inflammatory œdema of previously not involved portions of lung with distention of the right heart, which so often accompanies the end of the disease, is to be regarded as an extension of the pulmonary inflammation, and the final giving out of the right heart is owing to this added obstacle. That this is the explanation of the particular case cited above by way of illustration is rendered still more probable by the existence of considerable fibrinous exudation on the surface of the congested and œdematous left lung.

We turn now to the bearing of the above-enumerated

considerations on the treatment of the disease. It is hardly necessary to state that so conspicuous a phenomenon as the relaxed condition of the peripheral vessels has not escaped observation and consideration from a practical point of view on the part of other authors. Romberg's especial point is that this factor is the dominant one in certain acute infectious diseases in causing embarrassment of the circulation, and I have endeavored to present the reasons for accepting his conclusions from a clinical standpoint, so far as the grave symptoms of the disease under discussion depend upon general toxæmia and not upon local lesions.

The most obvious method of combating extreme dilatation of the peripheral vessels is to administer drugs which cause their contraction. With this object in view, I have employed the chloride of barium in doses of four grains every four hours. In one case, after two days, the previously very much relaxed arteries contracted materially, the pulse slowed, and the marked delirium and prostration of the patient disappeared, although the temperature and pulmonary lesion remained unchanged. In others, no apparent result followed the same treatment. Fluid extract of ergot in doses of half a drachm every three hours had apparently some good effect in a few cases. The number of cases on which these drugs were used was, however, entirely too small to warrant any conclusion as to their utility, and I mention them only by way of suggestion.

It is, I think, significant that the sulphate of strychnine, which is now so widely used, and upon which I have mainly relied of late years, not only has a tonic effect upon the heart but also causes contraction of the peripheral vessels, through its action upon the spinal

vasomotor centres, which, according to Romberg, are not affected by the pneumococcus.

Although in Romberg's experiments the terminal cardiac failure was apparently secondary to the extreme dilatation of the peripheral vessels, yet it has been demonstrated that before the final failure occurred increased work was demanded of the heart. The outcome of any given case may depend upon the ability of the heart to respond to this demand. I have previously shown the almost identical tracing presented by a boy of sixteen, who recovered, and a middle-aged alcoholic, who died, the difference in result depending probably on the different ability of the two hearts to meet the extra demands upon them.

Owing to the number of cases in which antecedent cardiac lesions exist and to the difficulties imposed upon the right heart by the pulmonary lesion, the necessity of directly stimulating the heart is more often present in the disease as it occurs in man than as it is artificially produced in rabbits.

The remedy which theoretically and in the opinion of many practitioners practically best meets the requirements of the situation is digitalis, because it contracts the peripheral vessels as well as stimulates the heart. The use of digitalis in pneumonia is an old and much-discussed question. Its most energetic supporter to-day is Professor Petresco, of Bucharest.* He stated, in 1891, that he and his pupils had treated 1,641 cases of pneumonia with a mortality of only 2.06 per cent. He gives the drug in an infusion of four grammes of the leaves to two hundred cubic centimetres of water,

* *Therapeutische Monatshefte*, February, 1891.

to which are added forty cubic centimetres of syrup of orange peel, in doses of a tablespoonful every half hour to begin with, and in quantities representing four, six, eight, or twelve grammes of the leaves daily, according to the indications of the individual case, and continues these doses for from three to four days. He claims to have found it well borne by the digestive organs, and never to have seen a case of poisoning, although the pulse will sometimes drop to 24 to the minute. So far as I am acquainted with the literature of the subject, there are few who have the courage to imitate his example; yet it may well be that in the vasomotor paralysis which we have been discussing we have both the explanation of the tolerance of such enormous doses and the indication for their employment. In delirium tremens it is acknowledged that the tolerance for digitalis is enormously increased. It is quite possible that in pneumonia a similar tolerance exists and that most of us have been too conservative in the use of this drug because we have based our expectations of the effect to be looked for on results obtained in non-febrile maladies, and that the condemnation of the use of the drug in large doses in pneumonia in particular by high authority has been influenced by experience derived under conditions of the circulation which are vastly different from those obtaining in this disease. It is not to be forgotten that the preparation used by Petresco—viz., the infusion—does not represent the full potency of the leaves, as one of the most powerful alkaloids, digitoxine, is not soluble in water. The U. S. P. infusion contains alcohol, and is therefore a more powerful preparation than Petresco's.

With regard to the treatment of those cases in

which right-heart embarrassment occurs, I desire to call attention especially to nitroglycerin, the use of which is

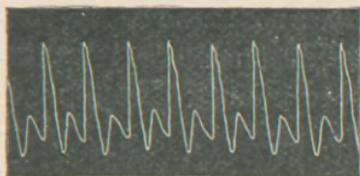


FIG. 4.—Pneumonia.

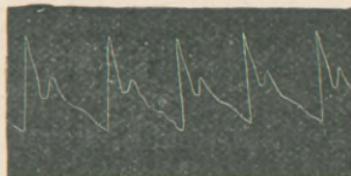


FIG. 5.—Convalescence.

advocated by Dr. A. H. Smith. I here show the tracing taken from a patient during an attack of pneumonia of considerable severity, one taken during convalescence, and one a few moments after the latter, when the circulation was under the influence of a very large dose of nitroglycerin. The similarity of the first and the last is apparent.

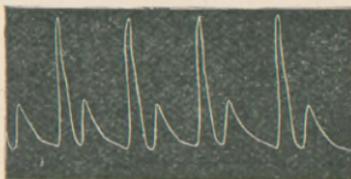


FIG. 6.—Nitroglycerin.

It seems a little like carrying coals to Newcastle to administer nitroglycerin in such a state of the peripheral vessels; yet, nevertheless, a still greater relaxation of the vascular system than that already existing may be a source of relief to the embarrassed right heart by lessening temporarily the amount of blood which it is called upon to force through the obstructed lungs. Its action is exactly analogous to bleeding. Its use should, however, be reserved for this specific indication. In the cases in which the vasomotor paralysis is a source of danger, this danger would be obviously increased by its employment.

As we all are only too painfully conscious, the treat-

ment of acute lobar pneumonia is not as satisfactory as we could wish. I offer these suggestions in the belief that the conditions of the vasomotors is a factor in the pathology of the disease which has not received the consideration which its importance demands, and that intelligent effort on the lines above indicated would save lives which would otherwise be sacrificed by trying to stimulate the heart under the idea that the circulatory failure present is due solely to insufficiency, from whatever cause, of the heart muscle, and neglecting to lessen its labors by toning up the peripheral circulation.

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