For instance, a man is shot through the head—and I have seen more than one person shot through the head fall down and get up and walk off—and there is some shock, and the man gets well after all this. I saw a man in whom I am sure the cells were very much torn; he had a buzz saw cut his skull open from the occiput to a point between the eyes tearing out the sinus—and not a small buzz saw but a large saw—and that man got well to my positive knowledge and lived ten or twelve years afterward; and I made a postmortem—after his death, of course (laughter). The point I want to get at is this: There was a very marked disturbance of the cells of the brain, and yet that man did not have any marked shock, he did not have any delirium. I know another case that was hurt on a railroad in an accident near Loudenville, O., the whole side of the head was crushed in; that boy got up and in less than two minutes he walked two blocks from the engine leaving a part of his brain lying on the platform, and dripping over him, and he did not become delirious. I begin to think that the theory which the doctor did not qualify, is untenable ground, and we should be careful in taking such a position, first, because it is not tenable, and second, because we have "our friends, the enemies," ready to say that is the cause of all the spinal calamities we go into court with. I do not think it is right to take that ground which is dangerous and which has a foundation not absolutely tenable. I simply bring these cases up as evidence that it is not always, at least, shaking up the nervous system that produces delirium.

Dr. BRYANT—I am not a pathologist, but I have a friend who is a histologist and pathologist, and he tells me that all cells, nerve cells as well as others, eat, grow, reproduce and have motion; and as a pathologist he says shock is due first to lack of eating—nutrition; he tells me the loss of blood will cause shock, interfering with nutrition; anything, like joy, grief or fright, which will interfere with digestion will also cause shock—it interferes with the nutrition of the nerve cells—and that is as far as I can go in this matter; but I think that is correct, and perhaps time will prove it.

Dr. HARNDEN—I had thought perhaps I would elicit some information on the cause of the peculiar condition. I find I have failed in my object. I am satisfied we will not be able to find the cause, the peculiar condition. I do not think I have expressly stated in any part of my brief paper anything which explains the condition to my mind or to yours; I thought Dr. Reed was going to give an explanation, and he got the buzz saw started half way through the man's head, but I found he stopped the demonstration, and I stop too.

RELATIVE INSUFFICIENCY OF THE PULMONARY VALVES.

Read before the Chicago Pathological Society, Feb. 8, 1897.

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I shall make no apologies for presenting to the Society the report of a case and a brief paper upon so rare a condition as insufficiency of the pulmonary valves and the rarer condition of relative insufficiency. If we would make accurate anatomic diagnoses of the commoner valvular affections, we must have some familiarity with the signs of the rare ones. I am confident that you will all agree that we should not content ourselves with the diagnosis, "heart disease," but should endeavor in all cases to make as detailed a diagnosis as possible.

Relative insufficiencies, that is, those due to enlargement of the orifices rather than to contraction of the cardiac valves, are now admitted by almost all clinicians. Those upon the two auriculo-ventricular orifices and the aortic orifices are well known and the recorded cases are very numerous.

The relative insufficiency of the pulmonary valves has received very little attention as yet, and the reported cases can be counted almost on the fingers of one hand. I believe, however, that when once the attention is directed to the subject, the cases will rapidly multiply, for many of the conditions which may in time cause a relative pulmonary insufficiency are common.

The literature of this subject may be dismissed very briefly. Cases have been reported by Stokes,[1] Kolisko,[2] Bristowe,[3] Coupland,[4] Litten,[5] Chauffard,[6] Gouget,[7] each one and supported by autopsy. Pawinski[8] in 1894 reported four cases, but only one went to autopsy some months after the diagnosis of relative insufficiency had been made, and no insufficiency of the pulmonary valves was found. Grawitz[9] reports one case unsupported by autopsy, so that his case is not above question. Chauffard's case seemed well established clinically, but at the autopsy the pulmonary valves were competent to the water test. Barie[10] in 1891 collected 58 cases of pulmonary insufficiency with 43 autopsies, and included in his list the cases of Stokes, Kolisko, Bristowe and Coupland. He evidently did not regard them as relative, for he questions the possibility of relative pulmonary insufficiency, although in a later article, 1895, he admits the possibility. The literature thus contains but six cases supported by autopsy.

Because of this very brief list we must infer much of the etiology and symptomatology by comparison with aortic and organic pulmonary insufficiency.

Because of the anatomic and functional similarity between the aortic and pulmonary valves, we are justified in the inference that conditions similar to those causing relative aortic insufficiency might cause relative pulmonary insufficiency if brought to bear upon the lesser circulation. The possible causes of relative aortic insufficiency are numerous, but they may be conveniently classified in three groups: 1, those affecting the aorta, altering its structure so that it is dilated more easily, as in acute aortitis, arterio-sclerosis; 2, those increasing the peripheral resistance, as in nephritis; 3, those dilating the left ventricle, as myocarditis.

By transferring this grouping to the pulmonary valve, we have:

1. Arterio-sclerosis of the pulmonary artery is uncommon and rarely reaches a high degree. There is no reason why it might not cause relative insufficiency of the valves.

2. This group of causes, namely, those increasing the resistance to the circulation of the blood through the pulmonary artery, is most important and includes many different diseases. Gourand[11] in 1865 drew attention to the possibility of relative pulmonary insufficiency resulting from emphysema, chronic bronchitis and indurative pulmonary processes. Coupland's case was secondary to emphysema, Bristowe's to emphysema and obliterative pleurisy. Stokes does not specify in his report as to the primary condition, but states in relation to the question that he has seen marked dilatation of the pulmonary artery in cases of emphysema.

Gouget's and Chauffard's cases were secondary to a combined mitral lesion. It is well known that these lesions cause an increase in the resistance to the pulmonary circulation, and there is no reason why the increase may not so dilate the pulmonary artery as to render the valves incompetent. The four cases reported

by Pawinski, and the one by Grawitz, were cases of mitral stenosis. From the clinical report we can not but agree with Pawinski's diagnosis, but the autopsies are wanting.

Litten's case was very different. The pulmonary artery was plugged by an embolus, an echinococcus cyst. The vessel was dilated to such a degree that the valves became incompetent.

Other diseases which might belong in this group are obliterative pleurisy, obliterative pericarditis, kyphoscoliosis, mediastinitis, aortic aneurysms and other mediastinal tumors, and all conditions which sometimes give rise to pulmonary embolism.

To these we must add the cause found in the case to be reported. When for any reason the left ventricle fails to do its work, the blood may dam back into the lungs and reach such a tension as to cause relative insufficiency of the pulmonary valves. This adds a large number of diseases, all those which can cause an increase in the resistance in the systemic vessels.

3. The third and last group, those affecting the myocardium, could as well cause relative pulmonary insufficiency as it does other relative insufficiencies.

The physical signs of relative pulmonary insufficiency do not differ essentially from those of an organic insufficiency. There is a varying degree of cyanosis, but often far less than one might expect. Sometimes there is a visible systolic pulsation in the second left intercostal space, and this may be so marked as to suggest a thoracic aneurysm. Over this same area there is often a palpable thrill, systolic or diastolic, or both. On deep palpation below the ensiform we can sometimes make out a diastolic thrill in the right ventricle.

Percussion shows that the cardiac dulness is increased to a varying degree to the right. The left borders of the heart are normal or nearly so, unless changed by some complicating condition. The dilated pulmonary artery often gives a retrosternal dulness extending to the left second intercostal space.

Auscultation shows a diastolic murmur loudest in the second left intercostal space, propagated down the sternum and outward toward the left clavicle and usually not audible over the auscultation point of the aorta. According to Bernhardt, the murmur is loudest during expiration. The murmur is quite similar to that heard in cases of aortic insufficiency, but is not propagated into the vessels of the neck. Its point of maximum intensity is not peculiar to it, for the murmur of aortic insufficiency is frequently loudest in the second left interspace. It is often accompanied by a systolic murmur, just as the diastolic murmur of aortic insufficiency is.

Friedreich in 1867 stated that there could be no doubt but that the same vascular phenomena occurred in the pulmonary circulation with insufficiency of the pulmonary semilunars, that occur in the systemic vessels in aortic insufficiency. These vessels, although removed from direct observation, still give rise to certain signs described by Gerhardt.[12] Gerhardt[13] found that a manometer connected with the nose gave a tracing of the pulsation of the pulmonary artery. The tracing taken from a case of pulmonary insufficiency differed very greatly from those taken in the normal individual and in cases of aortic insufficiency, being higher and steeper and resembling the pulse tracing of aortic insufficiency.

Although we can not apply the stethoscope directly to the pulmonary artery, we still find traces of auscultatory phenomena similar to those of the peripheral vessels in aortic insufficiency. Gerhardt found in five cases of pulmonary insufficiency two dull tones audible over the entire chest and similar to those heard over the brachial artery in cases of aortic insufficiency. Similar tones are heard over the normal chest close to the heart, but not at a point so remote as the outer border of the right scapula. Wide diffusion of these tones is not, however, peculiar to pulmonary insufficiency, for they are found also in well-marked cases of mitral insufficiency, where they arise in the pulmonary vein, and in cases of aortic insufficiency, where they arise on the aorta.

Pulsation of the pulmonary capillaries occurs just as it does in the peripheral capillaries in aortic insufficiency. Systolic intensification of the vesicular murmur during slow and deep inspiration in the portion of the chest near the heart has been known for a long time, but this phenomenon, called by Gerhardt an "audible capillary pulse" over remote portions of the chest, occurs only with the pulmonary insufficiency when the capillary pulse is derived from the pulmonary artery, with cases of mitral insufficiency when the capillary pulse is due to a retrograde wave of blood through the pulmonary vein, and in cases of aortic insufficiency when it is exactly comparable to the capillary pulse in the systemic vessels.

The second pulmonary tone may be present or absent.

These, briefly, are the physical signs in cases of relative pulmonary insufficiency. We will find in addition the signs of the primary condition.

The following is a brief clinical and postmortem report of a case, seen in Dr. Herrick's ward of the County Hospital:

German laborer, 42 years old, single and with negative family and personal history, except for an attack of gonorrhea seven years ago. Never had rheumatism or any other illness. About two years ago ran four miles without special inconvenience, but the next day spat blood, and had palpitation of the heart for several days. About one year ago had an attack of weakness and shortness of breath. Present illness began some days ago with dyspnea, headache, vomiting, cough, precordial pain and edema of feet and legs.

Examination showed a well-nourished man, face flushed and slightly cyanotic, moderate dyspnea, edema; veins of the neck prominent, showing a negative pulsation, and at no time did they show the positive pulse characteristic of tricuspid insufficiency. Temperature normal. Pulse slow until just before death. Small amount of fluid in chest, later increasing to such an amount that the chest was aspirated, drawing off a pint and a half of serous fluid of specific gravity of 1009, and containing few red blood corpuscles. Chest and lungs otherwise negative.

Heart: Apex beat in sixth interspace in the anterior axillary line. Deep cardiac dulness to second rib above, and one and one-half inches to right of sternum and to the apex beat. Palpation showed over the apex a systolic and presystolic thrill. The pulse had the characteristics of the pulse of aortic insufficiency well marked; capillary pulse distinct. Auscultation at the apex showed systolic, diastolic and presystolic murmurs. At the base two diastolic murmurs resembling the murmur of aortic insufficiency, that is, soft, long drawn and blowing. One of these murmurs was transmitted downward from the aortic area toward the apex; the other downward along the sternum. The second pulmonary tone was wanting, as was also the second aortic.

The liver was enlarged from passive congestion.

The urine scanty, of high specific gravity, albumin with granular and hyaline casts.

The interpretation of the cardiac findings was not entirely simple. The shape and size of the cardiac dulness indicated an enlargement of both ventricles. The character of the pulse and the diastolic murmur at the base satisfactorily demonstrated an aortic insuf-

ficiency, which accounted for the enlarged left ventricle.

The enlarged right ventricle was referred to a mitral lesion, either double or single. Relying mainly upon the presystolic thrill at the apex, the lesion was thought to be a combined mitral insufficiency and stenosis. Had there been only the presystolic murmur at the apex, it would have been interpreted as the presystolic murmur described by Flint in cases of aortic insufficiency. I have since learned that Schwalbe has found a presystolic thrill at the apex in a number of cases of aortic insufficiency without any accompanying mitral stenosis.

We had yet to account for the second diastolic murmur at the base, transmitted down the sternum, and for the absence of the second pulmonary tone, which, if there was a mitral lesion, should have been accentuated. Both of these could be easily explained by the supposition of a pulmonary insufficiency. This supposition was strengthened and confirmed when, after a few days' treatment, this murmur disappeared and the second pulmonary tone reappeared. The improvement was but temporary and the pulmonary tone was again replaced by a murmur.

The clinical diagnosis was aortic insufficiency, mitral stenosis and insufficiency, relative pulmonary insufficiency, parenchymatous nephritis, hydrothorax and passive congestion of liver.

After being in the ward a few weeks the patient died, and an autopsy was held four days after death.

The heart unopened weighed 1040 grams. Walls of both ventricles thickened, left ventricle 2 centimeters, right ventricle 1 centimeter. Aortic semilunars thickened, not more than two-thirds the normal size and incompetent to the water test. Pulmonary valves unchanged in structure, but are incompetent to the water test. Mitral valves thickened at the base, many vegetations. Orifice admits six fingers. Myocardium soft and friable, with scar in the interventricular septum. Tricuspid negative. Aorta shows atheromatous plaques. Liver of passive congestion. Kidney of parenchymatous nephritis.

Postmortem diagnosis: Hypertrophy and dilatation of the heart; aortic, pulmonary and mitral insufficiency, fatty and congested liver, parenchymatous nephritis.

The autopsy showed first that there was no mitral stenosis, so that our inference of mitral stenosis from the presence of the presystolic thrill at the apex was not warranted.

It is much to be regretted that the pulmonary orifice was not measured. The normal, according to Rauhing, being 67 millimeters in the male, measured in fifteen cases, and 63 millimeters in the female, measured in sixteen cases. Peacock's figures vary from these, being 90 mm. in the male, measured in sixteen cases, and 88 mm. in the female, measured in thirteen cases.

The water test as applied to the pulmonary valves is much more reliable than when applied to the aortic valves, for there are no coronary arteries through which the water may escape, and the difference between the blood pressure under which the valves close during life and the pressure of the water test is not so great as with the aortic valves.

From this case and the others so far reported, we must conclude that relative insufficiency of the pulmonary semilunar valves is possible, and inasmuch as the primary diseases which may cause this lesion are common, we are justified in believing that this condition is not so rare as so far supposed.

1 Stokes: Diseases of the Heart and Aorta, Case xx.
2 Kolisko: Zeitschrift d. K. K. Gesell. d. Aerzte zu Wien, 1859, xv.
3 Bristowe: Trans. Path. Soc. London.
4 Coupland: Trans. Path. Soc. London, 1875, 26.
5 Litten: Charité Annalen, 1878, ii, 182.
6 Chauffard: Rev. gen. d. clin. et d. Therap., 1895, 2, 321.
7 Gouget: Rev. d. med., 1895, xv, 768.
8 Pawinski: Arch. klin. Med., 1894, 52.
9 Grawitz: Zeitschrift klin. Med, 1893, 23.
10 Barié: Arch. gen. d. med., 1891, 27 and 28.
11 Gourand: Paris Thesis, 1865.
12 Gerhardt: Congr. Intern. Med., xi.
13 Gerhardt: Charité Annalen, 1892, 17, 255.
14 Schwalbe: Virchow's Arch., 1890, 119: Deut. Arch. klin. Med., Volumes xliv and xlv.

ON THE PRODUCTION OF HYDROCHLORIC ACID IN THE GLANDS OF THE STOMACH.

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PRELIMINARY COMMUNICATION.

In *Medicine* of November, 1895, I showed that hydrochloric acid is secreted by the mucous membrane of the stomach and not formed by chemic action on the food products or fermentation. In these experiments I found a nearly constant relationship in the percentage of acid produced after each stimulation. Since then I have performed the same experiments upon three cases of chronic gastritis. In these cases free hydrochloric acid could never be detected with Boa's reagent, but upon quantitative determination of total chlorin before and after evaporation of stomach contents a difference of chlorin was found which gave about the same per cent. of volatile chlorid as was found in the cases of normal stomach, where free hydrochloric was always present.

This decomposition of chlorid was not syntonin, as the experiments were performed upon empty stomachs. This led me to believe that I was dealing with a very unstable organic chlorid, which when evaporated to dryness volatilized the same as free hydrochloric acid, but would not give Boa's test. On the strength of this observation I performed the following experiment:

1. Pure nucleinic acid was obtained from three dozen thymus glands of sheep.

2. A syntonin solution was prepared; this contained .2 per cent. of egg albumin and .1 per cent. of hydrochloric acid; the acid was all taken up by the albumin.

Then before proceeding, the nucleinic acid solution and syntonin were tested for free hydrochloric acid with Boa's reagent, to be absolutely certain that it was absent. To 5 c.c. of syntonin a saturated aqueous solution of nucleinic acid was added as long as a precipitate was formed. (The precipitate is a nuclein proteid.) This precipitate was filtered out and the filtrate tested for free hydrochloric acid. Boa's reagent gave a positive reaction. The experiment was repeated several times, the results always the same.

On making a quantitative estimation of the chlorin, in the syntonin and after precipitation with nucleinic acid, it was found that all the combined chlorin had been discharged as free hydrochloric acid.

About this time I had the good fortune of seeing a case at the laboratory which I must report as it will be the keynote of my theory, which I will state in the conclusion. The patient was a man who has suffered from chronic gastritis for several years. The first examination of the stomach contents gave the following: Reaction alkalin, free hydrochloric acid absent,