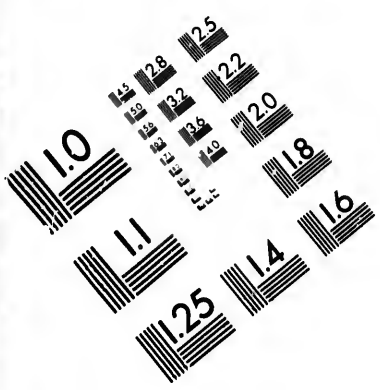

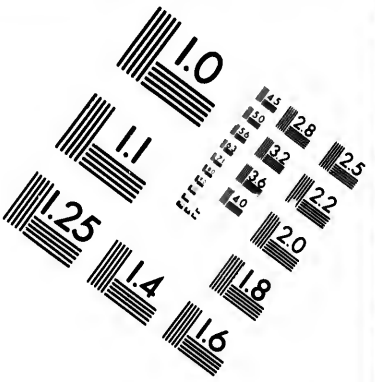

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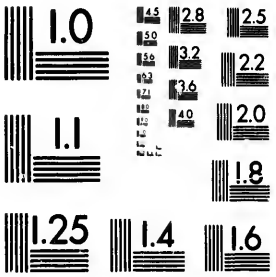

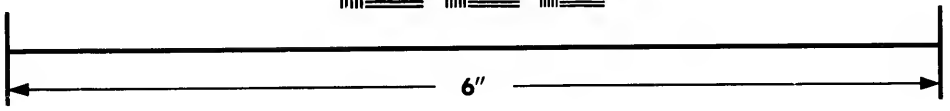

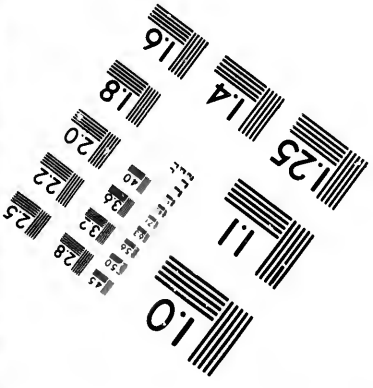

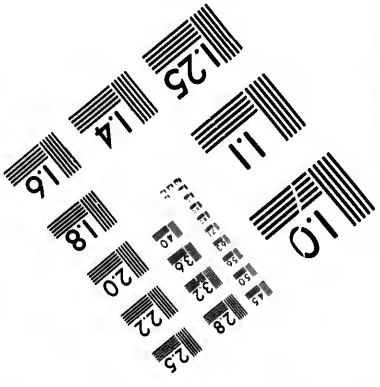

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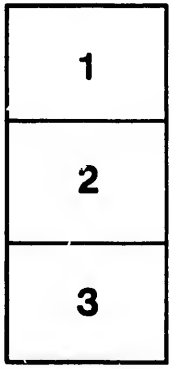

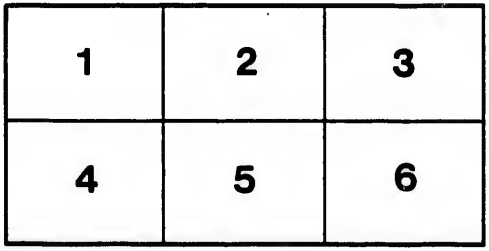

(*Reprinted from the* MONTREAL MEDICAL JOURNAL, *September*, 1890.)

CLINICAL LECTURE

UPON A CASE OF MITRAL VALVE DISEASE.

DELIVERED AT THE GENERAL HOSPITAL, FEB. 26TH, 1890.

BY R. L. MACDONNELL, M.D.,
Professor of Clinical Medicine in McGill University; Physician to Montreal General Hospital.

GENTLEMEN,—At our visit the day before yesterday we examined together a new arrival into the clinical wards, Jean F., whose history is instructive and interesting. You had an opportunity of hearing him tell his own story, and we examined together the physical signs presented. To-day I propose to resume the study of the case. F—— is an old country Frenchman, aged 50. He is intelligent, and gives a well-connected personal history. He has led a roving life, having been a soldier in the French army, subsequently a miner and western adventurer, and now he describes himself as a teacher of languages, having in his travels learnt Spanish, German and English. So far as we can learn he has never been intemperate, nor has he ever had syphilis. In childhood he had scarlet fever, and he has suffered from three attacks of rheumatism—the first at the age of 20, while a soldier; again in 1871 he was laid up for three months, and lastly in 1886 while he was working at the silver mines in New Mexico he was exposed on one occasion to great cold, and immediately afterwards was attacked with pain in nearly all the joints. This illness lasted nine months, and he understood from his medical attendant that it was of the nature

of acute rheumatism. During the course of the attack, after he had been ill some three or four months, he became suddenly paralysed in the right side of the body. He was aware, previously to this attack, that the heart had been affected by the rheumatism.

You know that in the course of acute rheumatism, in a large proportion of cases, probably in as many as fifty per cent., the morbid process attacks the valves of the heart, being particular in nearly every instance to select the mitral valve. Rheumatism is the usual cause of endocarditis, and in that disease certain changes take place in the valve. The smooth, glistening surface becomes vascular and roughened by an exudation leading to a deposit of fibrin. The process may be likened to the freezing of a river; the ice tends to form upon rough projections, shallow bays, rocks, in fact anything that presents an obstacle to the rapid current, while in deep places, up against smooth embankments and wharves ice does not so readily form. The fibrin of the blood is not only always ready to deposit itself upon a rough surface, but in acute rheumatism it is put into a condition in which coagulation is especially prone to occur. A deposit of fibrin, then, having taken place, a piece of it becomes detached, enters the circulation, and is carried from the big arteries to the little arteries, until it finally sticks in one of them, usually a cerebral artery. This is by no means an uncommon occurrence. Disease of the valves of the left side of the heart is most frequently the starting-point of this embolism. Here paralysis of the opposite side was the result. The vessel in which the clot is arrested is almost always one of the Sylvian arteries. It was formerly commonly taught that the left artery was more liable to plugging than that on the right, but this, it appears, is not the case, or at least it has not been confirmed by Fagge's observations in the post-mortem records of Guy's Hospital. Among twenty-one cases of embolism limited to the Sylvian artery of one side, he found that there were eleven in which the left one was affected and ten in which the clot entered the right artery.

The immediate result of plugging of the Sylvian artery, if that, indeed, was what occurred, was to shut off the blood from those parts of the brain in which the motor fibres of the right side of

the body take their origin. There was a sudden cessation of nutrition in the affected area, equivalent to a traumatic irritation, and paralysis was the result. Recovery was slow, but at the end of nine months he found himself upon his feet again.

Possibly the attack of rheumatism in 1886 may not be alone to blame for the endocarditis and the embolism. He was 46 in 1886, and rheumatism does not often, in fact you may say it never affects persons at this age unless they have had previous attacks. Nor is at all likely that the attack in question gave rise to the endocarditis. The tendency to endocarditis diminishes rapidly with age, especially after the thirtieth year, and more particularly in men, since women are more subject to the cardiac complications of rheumatism than are men. It is probable that the original endocarditis dates from one of his earlier attacks of acute rheumatism, and that in 1886 it took on fresh action, and this is rendered likely from the observed fact that embolism is specially apt to occur when valves previously the seat of disease undergo a fresh attack of acute endocarditis.

For the next three years he seems to have been in fairly good health, but notice that his occupation was no longer active, but was sedentary, and he became a teacher of languages. He never felt as strong since that attack of rheumatism. On the 22nd of December last, after partaking of an unusually good dinner, he suffered his first attack of palpitation and pain in the precordial region. The abdomen and the feet became swollen at night, and it was with great difficulty that he could attend to his daily duties. On the 14th February last, twelve days ago, he was obliged to take to bed. So far he has complained of the three symptoms of mitral regurgitation—(1) Pain. (2) Palpitation. (3) Dyspnœa on exertion.

State on admission.—It is evident at a glance that some serious disturbance of the circulation exists, for his general appearance and decubitus is that of a man who is suffering from dyspnœa. He sits up in bed, and in his conversation with me you observe his breathlessness. The face is pale, and there is a slight degree of œdema in the lids. The superficial arteries, the temporal and the carotid, do not pulsate visibly, but the veins

in the neck are in a state of active pulsation. The conjunctivæ are subicteroid. The abdomen is evidently enlarged and the feet are œdematous. He complains of uneasy sensations and pain in the precordial region, especially over the right hypochondrium. The temperature is normal. The pulse small and weak, but not irregular. Respirations are 28 to the minute. Over the precordial region we notice a general diffuse impulse, but the exact position of the apex cannot be made out. No thrill is perceptible. The area of superficial dulness is increased. It begins at the third costal cartilage above, extends to a point about an inch beyond the nipple and an inch below it, and on the right side it corresponds with a line one inch beyond the right edge of the sternum. The outline is made out with difficulty. The heart is evidently enlarged, and particularly on its right side. The absence of perceptible apex-beat is probably the result of right heart dilatation, which would push the apex away from the chest-wall. The pulsation in the veins of the neck shows there is leakage through the tricuspid orifice. This is a common result of disease beginning in the left side of the heart, not because the tricuspid valves become diseased, for it is rarely that they are ever attacked by endocarditis, but because the walls of the right heart become involved in the general deterioration of the muscular structure of the whole organ. The worst feature in the case is the presence of this tricuspid regurgitation, which shows that the endocarditis has gradually affected the heart structures, travelling backwards in the course of the circulation from the mitral to the tricuspid valve. At the apex-beat the sounds are regular but weak. The first sound over mitral area is replaced by a murmur, which can be heard around the chest as far as the boundary of the axilla, but it is not heard in any other than the mitral area. It is distinct, bnt not loud.

State of the lungs.—In diseases of the heart, the state of the lungs is of more or at least of equal importance to the state of the heart, because valve affections most commonly kill by interference with the lungs. The degree, therefore, to which the lungs are affected measures to a certain extent the dangers of the case. The respirations are quick and shallow. There is slight

cough. Both pulmonary bases are dull for a limited extent, and from the spines of the scapulæ downwards fine crepitating râles can be heard. There is therefore a moderate degree of pulmonary œdema. The lung changes are not as advanced as you might expect when you take into consideration the generally deranged condition of the circulation. The reason for this lies in the fact that the occurrence of tricuspid regurgitation tends to relieve pulmonary congestion, inasmuch as it weakens the force with which the right ventricle contracts. Consequently less blood is pumped into the lungs, because we see the veins in the neck receiving at each beat of the right ventricle some of the blood which ought to go to the lungs.

The appetite is poor; the bowels fairly regular. He complains of pain in the epigastrium and right hypochondrium. There is no vomiting, and he has never brought up blood. The abdomen is distended and measures at its widest part 37 inches. Fluctuation is present. There is dulness on percussion in both flanks, which is moveable on the right side, but not so on the left, owing probably to enlargement of the spleen. There is no fluctuation. Note the rapid onset of this abdominal dropsy, which is the immediate result of the regurgitation through the tricuspid. The abdomen suffers first, or nearly first, when this takes place because of the absence of valves in its veins, and this damming up in time acts on the portal circulation, producing symptoms due to the inability of organs to clear themselves of their blood. Congestions ensue, function is interfered with, and symptoms result. The pain and distress at the stomach of which this poor man complains is probably the result of the damming back of the blood in the gastric veins. The liver is large. Hepatic dulness in right mammary line extends fully three inches below the margin of ribs. The smooth surface of the organ can be readily felt separated from the abdominal wall by a thin layer of fluid. The spleen is large, possibly as the result of the old standing malarial disease, possibly as the result of the congestion from which it is evident all the viscera are suffering.

The quantity of urine passed is less than the normal. Since

his admission but some twenty or thirty ounces are voided daily. The fluid is high-coloured and cloudy; contains a whitish deposit which microscopical examination shows to consist of phosphates. The reaction is alkaline; sp. gr. 1025; no sugar; no albumen. Considerable bile pigment. After he had been in hospital a few days the bile pigment almost entirely disappeared. He is the subject of a right inguinal hernia and there is a discharging sore upon his right tibia.

Such is an outline of the history of the case before us. The sequence of events seems very plain.

1—Rheumatism.

2—Endocarditis.

3—Embolism and hemiplegia; recovery.

4—Enfeebled health.

5—Compensation and restoration to comparatively good health.

6—Failure of compensation, shown by precordial pain, palpitation, dyspnœa, cough, symptoms resulting from congestion of abdominal organs, general anasarca.

General effects of valvular disease.—Regard the circulatory and the respiratory system as one machine. Interference with that machine at any point produces a disarrangement of the whole, more especially is this the case when the part disarranged, as in the case of the heart, is the one which is so situated as to be a central point to all the machinery by which the body works.

The general effect of all valvular lesions, no matter where they may be situated, is to enrich the venous and impoverish the arterial systems.

In front of the lesion there is too little blood and behind it there is too much; *e.g.*, mitral valve lesions cause—

1—Increased pressure in left auricle.

2—Diminished pressure in left ventricle.

Therefore less pressure in aorta.

3—Back pressure is increased and extends to the pulmonary veins.

Therefore the first effect of mitral lesions is to impede the pulmonary circulation.

Compensation.—In the progress of a case of valvular lesion

there is a double process at work, a process of injury to the system and a process by which this injury is minimized, and to this latter we apply the term "compensation," which, it may be stated, generally consists in hypertrophy of the cavity immediately behind this lesion.

What occurred in this case after the mitral valve became incompetent?

1. Dilatation of left auricle.
2. Hypertrophy of the same.
3. Circulation in lung impeded.
4. Increased tension in pulmonary artery.
5. Dilatation of the right ventricle.
6. Tricuspid regurgitation.

When the compensation begins to fail, the cavities which are behind the valves begin to dilate. This may be checked and the power of the heart restored for a time. Changes then occur in the nutrition of the cardiac muscle, in the vessels, and in the general nutrition, bringing on *failure of compensation*. In this state the cavity has no longer power to expel its contents fully into the vessels, so it becomes increasingly distended and structurally deteriorated.

Symptoms of failing compensation.—1. Attacks of palpitation from slight causes, or even during sleep. In this case an over-distended stomach was the exciting cause. Remember that a deranged stomach can cause a derangement of the heart in two ways. By its bulk it can press upon the heart and hamper its action, and through its nerves it can derange those of the heart. You remember that the vagus goes to both.

2. Irregularity of pulse, due to abortive contractions which do not reach the wrist, or to contractions unequal in force or in the quantity of blood expelled. The pulse becomes small, unequal, irregular and compressible.

3. Cardiac oppression and anginal attacks from distension of the cavities of the heart.

4. Faintness and giddiness from cerebral anæmia.

5. Visceral complications. *Lungs*—Remembering that valve lesions act backwards, you can understand that in mitral incom-

petence the lung vessels are in a chronic state of engorgement which gives rise to a hypersecretion of mucus—*i.e.*, a state of chronic catarrh. This over-pressure acts upon the walls of the vessels, which become dilated and varicose ; hence there arise œdema and hæmorrhages. This is specially the case in mitral stenosis. Repeated attacks of this kind bring about a condition of lung known as brown induration. The varicose condition of the vessels in the alveoli interferes with oxidation and aids in the deterioration of blood which the other visceral congestions favour.

The state of the Liver.—As there is *general* venous stasis, the blood which passes from the liver to the inferior vena cava is impeded in its flow and a passive congestion of the liver is the result. The organ assumes that condition to which the term ' nutmeg' is commonly applied. It is at first large, but eventually shrinks in very chronic cases to half its size, and it may, like true cirrhosis, set up an ascites. As a result of this passive congestion of the liver catarrh of the tubes is set up and jaundice is thereby produced. There was a subicteroid conjunctiva when the patient was admitted and bile pigment was found in the urine. Similarly, hæmorhoids and epistaxis are prone to occur. Further results of the chronic congestion of the liver show themselves in other branches of the portal vein, and as a consequence, congestion and chronic catarrh of the *stomach* and *intestines*, which impede digestion and assimilation. The spleen is always ready to undergo enlargement, and when the liver becomes passively congested the return of blood by the splenic vein is impeded and splenic enlargement is the result.

Kidneys.—When the mechanical effects of valvular disease extend to the general circulation, the function of the kidneys is more or less disordered. Valve diseases fill up the veins at the expense of the arteries. Lessened arterial tension is the first stage, and as a result it makes itself felt in the Malpighian tufts and scanty, dense, high-colored urine is the result. When the venous stasis becomes general a further change takes place in the urine. The arterial anæmia still keeps it scanty, but the venous stasis leads to the transudation of serum, and albuminuria is the result. Long-continued congestion leads to structural

changes—hyperplasia of connective tissue and degenerative changes in the tubules. In advanced cases uræmia is added to the patient's troubles.

Brain changes.—Coarse lesions are not found in the brain unless a detached clot plugs a vessel and gives rise to symptoms, but the brain substance generally is œdematous and the membranes are thickened. Delirium amounting to mania is an occasional symptom in the course of heart disease, and when present is of very evil import. We have had recently in our wards several cases in which maniacal symptoms appeared in the course of valvular lesions. The blood-vessels of the general circulation are frequently affected with atheroma when the left ventricle is hypertrophied, and hence the likelihood of cerebral hæmorrhages.

Treatment.—In the complication of evils present one would suppose little relief could be afforded, but fortunately we have at hand the means of alleviating a great deal of suffering. The nurse has provided him with a bed-rest, by means of which he can rest fairly comfortably. We began with free purgation with calomel. Two grains every two hours were given until the bowels acted freely. In this way good was done in various directions. The intestines were unloaded, the congestion of the gastro-intestinal mucous membrane was relieved, and as calomel is a diuretic under certain conditions, it probably reduced the dropsy as well. There was certainly much less fluid in the peritoneum after the action of the calomel. Having cleared the way, I prescribed fifteen minims of tincture of digitalis to be taken every four hours. The night before last there was very urgent dyspnœa, and Dr. Campbell succeeded in affording relief by very freely dry-cupping the back.

Postscript.—The course of the case was at first satisfactory, though the stomach was very irritable and it became expedient to suspend the administration of the digitalis. At the end of three weeks there was considerable improvement—the dyspnœa was not so urgent, the anasarca was less, more urine was passed, and he was able to sit up and move about the ward. But this happy state of affairs did not continue, for on the 15th of April, after he had been 57 days in hospital, dyspnœa set in (respira-

tions 40), and cough became troublesome. Expectoration was very copious. The temperature rose. Signs of fluid were detected at the left base, and a pint of pus was withdrawn by the aspirator. Three days later a second aspiration was performed, but now his strength seemed unequal to this new strain and he became rapidly weaker, and died upon the 21st April.

In the main the autopsy verified the diagnosis. There was chronic valvular endocarditis with hypertrophy and dilatation as had been supposed, but there existed in addition chronic adhesive pericarditis. The liver and spleen were not so large as would be expected from the physical signs. The former was of a decidedly nutmeg character. There was a localized peritonitis in the hepatic region, which probably accounted for the pain in the right hypochondrium the patient complained of when he was admitted. The left pleura was the seat of an acute fibrino-purulent pleurisy.