utmost capacity (about one hundred and seventy beds) corroborates this opinion; that similar institutions for incipient cases are needed in different parts of the State is also shown from the fact that the applications far exceed the capacity of the hospital, many of them being from western Massachusetts, the large majority, however, coming from the eastern portion, especially the vicinity of Boston. To increase the number of institutions moreover, rather than to greatly enlarge the capacity of the present hospital is much to be preferred. The largest sanitaria abroad with a corps of assistants have about 200 patients only. With a larger number we run serious danger of making the institution too bulky, and deterioration in the quality of work accomplished would almost inevitably follow.

Again I wish to emphasize what I believe to be one of the most important features of an institution of this nature; namely, its educational effect upon the community in general. Having positive proof of the beneficial effect of life in the fresh air, day and night, the patients preach this gospel far and wide to their families and friends with good results which I believe to be incalculable. Constant evidence of this is given in numerous letters received from former patients. During their stay they have had it impressed upon them that when they leave the institution they can act as missionaries, as it were, in the crusade against tuberculosis by teaching others the value of fresh air, proper diet, judicious exercise and cleanliness. That the lesson has not been lost upon them is one of the hopeful signs for the future.

To Massachusetts, then, belongs the credit of taking the first step in this direction in the United States. Indiana has lately followed her example, and if one may judge by the enthusiasm displayed at a recent meeting of the New York Academy of Medicine, New York will not be slow in following the example of her sister States. At that meeting a committee was formed to confer with the municipal and State authorities in consequence of a paper by Dr. Alfred Meyer, upon the Rutland Hospital. That many institutions of similar character will in another decade be established throughout the United States is more than probable.

One fact must, however, be recognized and guarded against. In the enthusiasm which marks the beginning of every new movement, we have to meet the inevitable errors of judgment and exaggerations of statement which, if not anticipated, will surely detract finally from the merits of any cause. Where popular interest is excited, as in this case, these dangers are more than doubled. Already we have been obliged to modify, if not deny, the exaggerated and often sensational statements of the lay press in regard to the Rutland Hospital. We, as physicians, are more or less prone to seize upon new ideas, and in the first glow of enthusiasm to paint with too glowing colors opinions which, under soberer judgment, have often to be modified, even when not completely abandoned. Progress seems to be always marked by the middle path between ultra-conservatism and blind enthusiasm.

In urging vigorous State action in the treatment of tuberculosis, we certainly cannot be accused justly of taking either extreme; we are simply building upon the idea which received its first strong impulse from Brehmer, in the mountains of Silesia, forty years ago; an idea which has steadily and surely grown, fostered by the patient and careful work of competent observers in Europe and America. Keeping in mind the fact that every method has its limitations and that as yet there is no known panacea, we can rejoice, nevertheless, in the public recognition of the necessity of using radical measures, as our State is now doing, believing them to be a great step forward, not only in the treatment but the prevention of tuberculosis. That the medical profession of Massachusetts, therefore, may strengthen by their personal influence the work just begun by the State is my earnest desire.

BRADYCARDIA, WITH REPORT OF A CASE.[1]

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BELIEVING that cases of persistent slow pulse are sufficiently rare and interesting to warrant reports, the following is submitted:

Mr. A., machinist, age seventy-five. His father and mother died of phthisis; four sisters and a brother died of heart disease; there is one brother now living, eighty years old. He has always used strong tea and coffee in liberal amounts, and for fifty years has consumed a ten-cent piece of tobacco daily, both smoking and chewing; of late this amount has lasted him a week. In alcoholic liquors he has indulged very moderately. There is no venereal history, and he has never received any injury of consequence. At the age of twenty-five he had rheumatic fever in several joints, of about three months' duration. From that time to seven years ago, he does not remember consulting a physician. Seven years ago he had an attack of bad breathing; said the doctor feared pneumonia, but was not confined to his room and fully recovered in a few days. At this time he gave up work and has done none since, although he has been well and active, frequently taking walks of several miles. For the past year he has been obliged to take occasional rests during the walks, on account of difficulty in breathing.

February 2, 1899, he consulted me on account of edema of both legs, scantiness of urine and general weakness, of about two weeks' duration. For past several months he has complained of a numbness of head, vertigo and faintness, but has never fainted; also of being short winded and always feeling cold.

Examination showed a man six feet in height, rather spare in flesh and active, of a calm nervous temperament. There was cyanosis of lips, nails and ears; the veins of hands and arms were very prominent; there was also some puffiness under the eyes and moderate edema of both legs and feet; no ascites. The pupils were contracted, but would react to light and accommodation. Knee-jerk and ankle clonus normal. The lungs and abdominal viscera were negative. There was no atheroma of radial, brachial or temporal arteries. Pulse in right radial while sitting was 30 per minute, strong, full and regular; the left radial the same, but less strong. A few minutes later while standing the pulse was 26 per minute.

Examination of heart. — Pulsation seen in epigastrium while sitting, not on standing. Percussion gave no enlargement; if any change, it seemed undersized. The apex beat was easily felt in the fifth interspace,

[1] Read in part before the Clinical Section of the Suffolk District Medical Society, November 15, 1899.

one inch within the nipple. At the apex the heart's sounds were heard, strong and labored; sometimes irregular, but usually regular; there was a soft, blowing, systolic murmur, transmitted to a little outside of the nipple and upwards for about two inches; not heard in the back. The pulmonic second sound was slightly accentuated. The sounds over other valvular areas were indistinct. The heart's systole and arterial pulsations were synchronous.

The appetite was fairly good, the bowels were regulated by a laxative. Acetate of potassium, 10 grains, three times daily, and tincture nux vomica, eight minims, three times daily, were prescribed. The patient at this time seemed very little inconvenienced by his affection.

February 22d. After ascending a flight of stairs the breathing was accelerated and the pulse was 32; after ten minutes, undergoing examination, pulse was 32; fifteen minutes later it was 30.

February 24th. Sitting, pulse 29. Edema diminished; less cyanosis; was passing more urine.

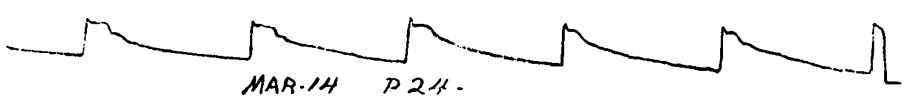
February 28th. Lying, pulse 29; twenty minutes later, 30. Edema still more dimininished; less trouble in breathing and feeling stronger.

March 2d. He was seen by Dr. Harold Williams at Boston Dispensary and shown to students. Immediately after going up two flights of stairs and being somewhat excited, the pulse was 34 and irregular.

March 5th. Edema nearly disappeared; no pitting. Pulse 24. Was seen by Dr. Twombly, who agreed in points of examination. During past several days had taken nux vomica irregularly.

March 10th. On account of the increased slowness of pulse, nux vomica was increased to 12 minims, three times daily, apparently without any effect. Examination of urine at this date was negative. Pulse 28.

March 14th. While taking a sphygmographic tracing, in which he was much interested, there was occasional irregularity of the pulse, which was 24.



March 17th. Pulse 27, standing; temperature 97°, respiration 26, following exercise.

March 19th. Pulse 27, standing; temperature 98°, respiration 21.

March 21st. Pulse 27, sitting; temperature 98°, respiration 20. Prescribed atropia, .01 grain, three times daily.

March 24th. Before going up two flights of stairs hurriedly, pulse 26, respiration 24. After, pulse 28, respiration 29. Some edema present; mouth very dry; pupils dilated. Omit atropia; take potassium acetate, 15 grains, three times daily.

March 30th. Pulse 29.

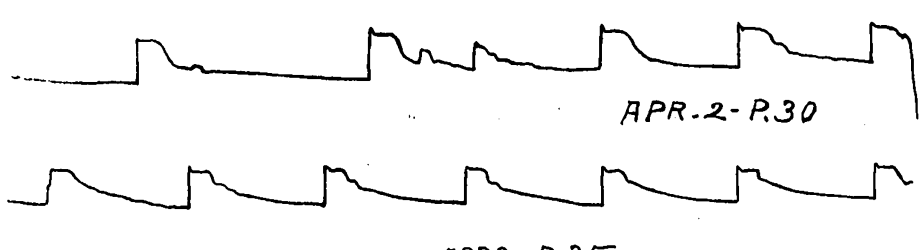
April 2d. Edema slightly diminished; slight pitting over tibiæ. Urine increased; gets up once at night, but has not formerly. On my arrival, pulse was 28, temperature 98.6°, respiration 22. While using the sphygmograph the pulse reached 36, the highest I had ever counted it. There was also an occasional irregularity, as noted on previous occasions, usually when patient was excited; the condition is shown by tracing of this date, the only time I succeeded in catching it with the sphygmograph. The high pulse rate and occasional irregularity I could not account for, as the patient was not excited; there was no apparent cause. This was the last time I saw him.

April 4th. He walked to his daughter's, a distance of three-quarters of a mile. On his arrival did not seem fatigued or out of breath; he sat down and talked a few minutes. Shortly his daughter, not receiving a reply to a question, looked up and saw that he was very pale and motionless — "looked as if he was dead." In a minute consciousness returned. He became purple, spoke a few words and vomited; afterwards he felt all right. Directly after a light dinner he had another attack; recovered in two minutes. He sat up that evening and talked as usual, retired at 8 P. M., slept well and arose next morning refreshed, but a little weak. During the morning of April 5th he had three attacks; could tell when they were coming on. That afternoon he went to bed, had one severe attack and died. A physician who was called said the pulse was as low as 25.

An autopsy could not be obtained.

The features of this case which seem of interest are, (1) the cause of the slow pulse; (2) the duration; (3) the influence of the excessive amounts of tea, coffee and especially tobacco; (4) the relation of the valvular lesion to the slow pulse; (5) the rise of pulse and temperature three days before the patient died.

It will be seen in the following list of cases that this case is assigned to a class in which the cause is rarely determined before death, and not always, by any means, at the autopsy. During his illness seven years ago, he said "the doctor took his pulse and remarked that he had some fever," and he inferred that the rate was high. From correspondence with the physician I found he did not remember the case; had there been a markedly slow pulse, it seems probable that he would have recalled it. The duration of the symptoms has been less than one year, and probably the slow pulse did not much precede them. Tea, coffee and tobacco



have been assigned as causes of bradycardia; that either was the cause in this case would be difficult to prove. Bradycardia, as we all know, is rarely met in valvular lesions, its presence from whatever cause might be expected to interfere with compensation, and in this case it seems not improbable that it may have done so. That the valvular lesion was the cause of the slow pulse there is no way of determining. In any similar case a spasmodically increased pulse rate without apparent cause would to me be an ominous sign.

Another case that recently came under my observation will serve as an example of another very different type of bradycardia, and may warrant a brief report.

Man, forty years old, health generally good. Examined him in May, 1899, with reference to his heart, which was found normal; pulse the same. Was called to him in October, 1899, on account of an attack of indigestion, and found the pulse 43 and 45 per minute. During the hour I was with him it did not exceed 45, and was synchronous with the heart's systole. The next morning the patient was feeling quite well and the pulse was 90; three days later it was 74 per minute. There were no symptoms referable to the slow heart's action; the duration probably did not exceed twenty-four to forty-eight hours. The cause was most probably a reflex one arising from the stomach.

The term "bradycardia" has been given by Grob to those cases of slow pulse which are synchronous with the heart's systole, the rate being less than 60 per minute. The term has, however, been incorrectly applied to cases in which the pulse and heart's systole were not synchronous, the pulse for some reason failing to indicate every ventricular contraction. Moreover, there has been a tendency with some writers to accept only a lower rate, as 50 or under, as constituting a bradycardia. Hence there is confusion not only regarding the rate but the frequency of occurrence of bradycardia. Grob, during a period of three years at the Zurich Medical Clinic, found 82 cases of slow pulse in 3,578 cases observed; all but four occurred in males. Riegel, in seven years' observation at the Giessen Medical Clinic, found 1,041 cases in 7,567 patients; 710 were in males, 331 in females. On the other hand, R. Gossett Brown in 1889 said: "In many thousands of cases both in hospital and in private practice, I have twice seen cases of slow pulse." The most obvious reason for this discrepancy in observations would seem to be a difference in pulse rate referred to, that of Brown's probably being much lower than that given by Grob. Accepting Grob's rate, cases of bradycardia may be considered of fairly common occurrence.

It must, however, be borne in mind that the figures of Grob and Riegel have reference to hospital cases only, a class in which slow pulse is more commonly met, being secondary to some illness, but it occurs in the healthy as well as in the sick. Statistics collected from all sources, though of larger figures, would show a much smaller per cent. of bradycardia than the above. Prentiss, who in 1889 must have made a diligent search for individually reported cases, collected 94. To this number I have been able to add but nine cases reported since 1889, making a total of 103 cases. This collection differs from that of Grob's and Riegel's in embracing a far larger proportion of the unusual types. Of these 103 cases, excluding those due to fatal injury and others in which the age is not given, we have 77 cases, of which the average age is found to be fifty-three years. Adding 84 of these cases in which the sex is recorded to Grob's and Riegel's collections we have a total of 1,207 cases, which is a larger number and offers a greater representation of all classes than any statistics previously presented. Of these 1,207 cases we find over 70 per cent. occurred in males. The fact that bradycardia is more common in males than in females has given occasion to Morison for an article on this feature, in which he concludes: "The belief is justfiable in the meantime until statistics prove the contrary that the constitution of the male more frequently results in bradycardia than do the like conditions in the female, although large statistics show that heart disease is more common in women than in men."

The conditions in which bradycardia occurs are of interest, and are well given by Riegel in his classification, which, by the way, is the one most generally accepted. It is as follows:

(1) Physiological bradycardia.

(2) Pathological bradycardia.

Physiological bradycardia includes those cases due to physiological conditions only. Probably Napoleon's case is the best-known example of this type. The condition is reported to have been observed in entire families. It occurs in the puerperal state at full term, abortions and premature labors; also in fasting and other conditions. In any case of slow pulse an extended observation may be necessary to determine its class.

Pathological bradycardia embraces all cases not physiological. It occurs in the following conditions:

(1) Convalescence from acute fevers, such as pneumonia, typhoid fever, erysipelas, scarlatina, diphtheria, acute rheumatism, and the like. It is most seen in young persons in whom the fever has run a normal course. Hibbard concludes that its presence in young children with diphtheria is a sign of serious heart trouble. According to Atkinson and others, it is met with in the beginning of acute rheumatism as well as in convalescence.

(2) Diseases of the digestive system: Acute, chronic and nervous dyspepsia, ulcer and cancer of stomach, and esophageal affections.

(3) Respiratory diseases: Emphysema, bronchitis, pleuritis and laryngeal affections.

(4) Circulatory disturbances: Myocarditis, fatty degeneration of heart, pericarditis and arteriosclerosis; not common in valvular diseases.

(5) Diseases of the urinary organs: Nephritis and cystitis.

(6) Toxic agents: Uremia, lead, alcohol, tea, coffee, tobacco and certain drugs.

(7) Constitutional disorders: Anemia, chlorosis and diabetes.

(8) Diseases of the nervous system: Epilepsy, apoplexy, cerebral tumors, medulla affections, injury to cervical cord, meningitis, mania, melancholia, and the like.

(9) Various other affections, as skin diseases, affections of the sexual organs, sunstroke, etc.

After giving these divisions and subdivisions, Riegel feels that there are still cases which may not properly

come under this grouping, and mentions as an example those cases in old persons in whom some affection of the heart or blood-vessels, most probably due to old age, is the cause of the slow pulse. Again, of the 94 cases by Prentiss, in 35 no cause is assigned for the slow pulse. This is of particular significance for the reason that every writer has probably done his utmost to ascertain the cause.

For the above reasons, but more particularly from the observation of one of these cases and the study of many reported cases, I am led to believe there is a class of case which thus far has not received its due amount of consideration. In confirmation of this belief I present a series of cases which, both in number and in similarity to one another, seem sufficient to establish a class of their own. In number they constitute about one-third of all the individually reported cases, and undoubtedly many more might be added to this list; were they not of particular interest they probably would not have been reported.

Of 33 cases in which the age is given, the average is found to be over sixty years, and but one case occurred under forty years. Of 34 cases in which the sex is recorded, 30 were in males. In more than half of the cases the duration has been years; in any case it was probably longer than was recorded, as the condition is rarely discovered in the very beginning. The pulse rate recorded was the lowest observed, and although it may not have remained at that point it usually continued low to the end. From the fact that in these cases the slow pulse was persistent, whereas in other cases of a different type it is a temporary condition, Truffet has divided all cases into (1) *Transitory* and (2) *Permanent* bradycardia. This division, like all others, has been more or less criticised. The temperature in the few cases recorded was subnormal, except perhaps near the end.

The bradycardial symptoms referred to consist of attacks of syncope, vertigo, dyspnœa and sensitiveness to cold, all of which may be due to disturbances of the circulation, resulting from the slow pulse. These symptoms were present in all but one case. It is of interest to note that in a large number of these cases no accompanying disease has been apparent, and even in those cases attended by some affection it would often seem a question of how much influence it may have had on the bradycardia. From the indiscriminate use of the terms "epileptic," "epileptiform" and "apoplectic" in the reports of these cases, it is difficult at times to determine if a true epilepsy or apoplexy were present. Perhaps attacks of syncope may have been mistaken for true "epileptic fits."

In the cases where a fatal termination has been reported, many times it seems to have been caused by the bradycardia, the person dying in, or directly following, the attacks of syncope. Fatty degeneration of the heart and atheroma were most frequently found in the ten autopsies; in some, however, there were no pathological conditions which might be assigned as the cause of the slow pulse. This is the type of case we see reported under the headings, "A remarkable case of slow pulse," "An unusual case of persistent slow

BRIEF REPORTS OF THIRTY-FIVE CASES OF SLOW PULSE, DESIGNATED BY THE AUTHOR AS CASES OF IDIOPATHIC BRADYCARDIA.

No.	Sex.	Age.	Duration.	Bradycardial Symptoms.	Lowest Pulse.	Temperature.	Respiration.	Accompanying Disease.	Termination.	Autopsy.
1	M.	54	Years.	Present.	12+	94.5-98°		None mentioned.	Died in attack.	Thorough but negative.
2	M	83	"	"	36	96°	20	" "	Living.	
3	F.	75	"	"	34			" "	Living.	
4	M.	54	Days.	"	9			" "	Died.	Four ounces fluid in ventricles of brain.
6	"	46	Years.	"	14			Epilepsy.	Living.	
8	"	70	Weeks.	"	16			None mentioned.	Died in fit.	Ossification of aorta.
11	"	67	Years.	"	32			None mentioned.	Living.	
12	"	70		"	38			Symptoms of brain.	Living.	
18	"	40	Years.	"	40			None mentioned.	Died in fit.	Fatty degeneration of heart.
19	"	70	Years.	"	26			" "	Living.	
20	"	60		"	17			" "	Living.	
38	"	45	Years.	"	28			" "	Died in fit.	Hydropericardium. Heart muscle softened.
44	"	58	Weeks.	"	22			" "	Living.	
48	"	53	Years.	"	28			Valvular.	Sudden death.	
49	"	57	Years.	"	18			None.	Died in fit.	
58	"	62	Months.	"	33	95.6-96.8°		None.	Living.	
59			Months.	"	23			Epilepsy. Syncope?	"	
63	M.	96		"	36			Slight bronchitis.	"	
68	F.	69	Years.	"	30			None mentioned.	"	
69	M.	71	Years.	"	28			" "	"	
82	"	52		"	22			" "	Died suddenly.	
83	"	79	Months.	"	4			" "	Died with fits.	
85	"	70	Years.	"	25			" "	Died in fit.	
92	"	38	"	"	19	97.4-98°		" "	Living.	
16	"	62	"	"	30			" "	Living.	
23	"			"	26			" "	Died.	Heart dilated.
52	F.	78		"	18			" "	Living.	
55	M.	66			21			" "	"	
61	"	61	Months	Present.	36	96°		" "	"	
Mayer.	"	65	Months.	"	18			" "	Died in attack.	Fatty degeneration of heart.
Claybaugh.	F.	55		"	36			La grippe.	Died as result.	Ossification of coronaries and aorta.
Mengy.	M.	75		"	27		27-30	Epilepsy?	Died.	
Hanot.	"	55	Weeks.	"	28			None mentioned.	"	Atheroma and hydronephrosis.
Hanot.	"	72	Months.	"	36			Bronchitis.	"	General atheroma.
Author's.	"	75	Weeks.	"	24	97°	20	Valvular.	Died with attacks.	

Numbered cases are taken from Prentiss's collection.

pulse," and the like. Unlike the cases due to acute fevers, digestive disturbances, etc., this type is rare; from the usual fatal termination it becomes of especial interest and warrants consideration. Should it not then be assigned to a class of its own? Two classifications have already been referred to, Riegel's and Truffet's.

Riegel's classification is based on the physiological and pathological conditions supposed to exist, for certainly our knowledge of the physiology and pathology of bradycardia at the present time warrants but little more than a supposition in any case. Besides, under one heading he groups those temporary cases usually terminating favorably, together with the persistent cases generally ending fatally. Surely a distinction should be made between these two classes.

Truffet, as I have said, bases his classification on the duration, and places under one division those persistent cases generally believed to be due to physiological conditions, and not affecting the health, together with the persistent cases ending fatally. Here again a distinction should be made, if possible.

Flint has divided all cases into "functional" and "organic." But this division, like Riegel's, calls for a knowledge of the pathological conditions, which we lack.

Without entering further into a discussion of this feature, let us understand (and it is hoped it has been made clear) that there are three types of bradycardia, which differ from one another either in cause, duration, symptoms, or termination. Then let us assign each type to a class of its own. Such a division, one consisting of three classes, has been made by Grob, and he has used the terms "physiologic," "idiopathic" and "symptomatic," which are perhaps as significant as any, and to avoid the introduction of new terms it seems best to use these, but with a somewhat different assignment of cases. Thus we have (1) *Physiological* bradycardia, (2) *Idiopathic* bradycardia, (3) *Symptomatic* bradycardia.

Physiological bradycardia includes those cases due to physiological conditions only. This class is fairly well agreed to by all writers on the subject.

Idiopathic bradycardia may embrace those cases of persistent slow pulse usually occurring in advanced life and more commonly in the male sex, accompanied by the usual bradycardial symptoms, but rarely attended by any discoverable disease which is the cause; the termination is usually fatal, though cases have been known to last for years without seriously affecting the health.

Symptomatic bradycardia should include all cases embraced by Riegel's pathological division, excepting the above classes. To this class belong those cases in which the pulse is symptomatic of some usually apparent condition, ordinarily of temporary duration, but it may include cases terminating fatally from injury, sunstroke, poisoning and certain other serious conditions. As has been said, all classifications are subject to criticisms, but it is hoped the one now offered may in a degree serve its intended purpose.

Cause. — The immediate cause of bradycardia may be due to (*a*) a condition of the nerve centres producing either irritation of the pneumogastric or paralysis of the sympathetic (accelerator) nerves of the heart; (*b*) a condition of the pneumogastric increasing its irritability; (*c*) a condition of the sympathetic nerves paralyzing them; (*d*) or to some condition of the cardiac ganglia in which the influence of the pneumogastric preponderates; (*e*) or a condition of the heart muscle whereby it fails to respond to the normal stimulus; (*f*) or to poisons acting either on the nerve centres or endings.

Pathology. — Our knowledge of the physiology of bradycardia is limited, and the same is true of the pathology; at the same time, regarding the pathological causes of bradycardia we are not lacking in theories. The following 31 autopsy reports give some idea of the pathological conditions thus far observed.

Disease of Brain. — Hydrocephalus, Cases IV, XXIV and XLV; softening, Case XXXIX; adhesions, Case XXV; abscess of medulla, Case XXVIII. Total, six cases.
Disease or Injury of Cord. — Fracture of cervical portion, Cases XXIX, XXX, XXXI, XXXII, XXXIII, XXXIV, XXXVII; induration, Case XXXV. Total, eight cases.
Disease of Heart. — Fatty degeneration, Mayer's, Cases XXXVIII, XVIII; ossification of aortic valves, Case X; hypertrophy, Case XXII; dilated, Case XIII; heart flabby and mitral lesion, Case L; mitral lesion, Case LXXVIII; aneurism of ventricular septum, Case XLVII; gumma of ventricular wall, Coggeshall's. Total, ten cases.
Disease of Circulatory System. — Ossification of coronaries, Claybaugh's, Case XXI; arteriosclerosis, Hanot's, two cases; ossification of aorta, Case VIII. Total, five cases.
Negative. — Cases I, LXXXVI. Total, two cases.
Numbered cases are taken from Prentiss's collection.

Prognosis. — From what has already been said it is believed the prognosis of the various types of bradycardia is fairly evident.

Treatment. — As to treatment it is also evident that many cases require none, the removal of the cause effecting a cure. In those cases where the patient suffers from the result of a slow pulse, and in which life may be threatened, rest and certain drugs seem indicated. Strychnia, atropia and nitroglycerin are most commonly used, but in the majority of cases without any beneficial effect.

A complete review of this subject has not been attempted. If the reader's attention has been directed to the following points the object of this paper is attained.

(1) A pulse rate under 60, which is synchronous with the heart's systole, constitutes a bradycardia, accord to Grob.

(2) The condition, all classes considered, is of common occurrence.

(3) It is much more common in males than in females.

(4) There are three types of bradycardia, as classified according to their clinical aspects.

(5) The class here termed idiopathic bradycardia, on account of its usual fatal termination, must not in any case be passed by as a mere curiosity.

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A BRIEF NOTE ON SOME OF THOSE GRAVE ABDOMINAL LESIONS WHICH OFTEN DEFY DIAGNOSIS.

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IT is a fundamental principle in our profession and in the science of the healing art, that the first step in the management of a case is to study, interpret, and elucidate symptoms and signs; in other words, make a diagnosis. And, generally speaking, the art of diagnosis has of late years spread far in advance of the power to arrest or cure disease.

Abdominal lesions, traumatic or pathologic. — The above holds good in the lesions of any organ in the body; however, there yet remain very many pathologic conditions within the abdominal cavity wherein definite diagnosis is not possible, except through the breach made by the surgeon's scalpel, the exploratory incision, and exploratory manipulation, something which under many circumstances greatly adds to the gravity of many cases.

Hemorrhage, perforation, obstruction, or enteric paresis. — Very often, after a grave abdominal contusion, who can say with certainty which one of the above conditions prevails? The patient is in shock, with a distended belly; in pain; is vomiting, and depressed in spirits. He is anxious to live and ready to take the chances of an operation. But let it not be forgotten that injudicious operations kill. Our responsibility here is terrible when we properly view the case, and assume for the moment that the patient is a member of our own family.

Without operation. — But we endeavor to quiet our conscience by affirming that the patient "must die in any event." This is a vicious conclusion, as we all know that the abdominal viscera possess remarkable power of safely adjusting themselves to many pathologic conditions. Intra-abdominal hemorrhage is rarely mortal *per se*. Stimson, some years ago, showed by statistics that of the penetrating gunshot wounds of the abdomen, the mortality was larger after laparotomy than those cases treated on conservative lines. Reclus, of Paris, has demonstrated the same condition of affairs. The latest surgical reports from the campaign in South Africa by Sir William MacCormac[1] point strongly in the same direction.

The late Hispano-American imbroglio again showed that there was no place for "abdominal surgery on the battlefield," as the few who had the abdomen opened for bullet wounds succumbed, while all those who were left alone did well.

If in doubt, don't interfere. — Unless our patient is in fairly good form, or unless the symptoms point unequivocally to the seat of a lesion which *we know* to be definite, leave the patient alone, or rather follow closely on conservative lines. Unhappily, in our days, there is altogether too much surgery of the abdomen done simply for the purpose of "operating."

When death follows an acute abdominal lesion, before reacting inflammation sets in, it is invariably attributed to *hemorrhage* or *shock*. The rupture of a hollow viscus is always followed by great shock. To operate on one in this state deprives him of his only hope of recovery. Rather wait till reaction sets in, though we have then to deal with peritonitis. But let us not open our eyes in amazement at the mention of "peritonitis," for this follows every so-called "aseptic" operation performed in the peritoneal cavity, even when very good recoveries are secured. The French use the refined term "*peritonisme*" for this post-operative condition.

Perforation or mechanical obstruction. — Will not some of those of a fervid imagination and who have an explanation for everything be kind enough to point out the fundamental distinction between the symptoms of perforation and those of obstruction? Absence of hepatic dulness is a fairly certain guide in some instances, but it may be wanting. In both the onset is sudden; the patient is seized with furious colic, just such as is noticed in acute hepatic, renal, or appendicular stenosis. Manipulation of the abdomen, with the history of the case, will not always assist us in ferreting out the seat of trouble.

But, with some grave cases, there is no antecedent history of any moment; the abdomen may be clothed by a six-inch layer of abdominal fat, or it may be as hard as a plank from muscular spasm.

Traumatic and pathologic conditions. — In traumatic conditions leading us to suspect grave intra-abdominal lesion, the time to operate, if to operate at all, is indeed a serious problem; but in pathologic states, with but few exceptions, indubitable signs or symptoms of perforation or obstruction call for prompt surgical interference as the only means of saving life.

Crushes of the abdomen with grave symptoms. — When laparotomy is performed after violent compressive or concussive force has been sustained the mortality is very great.

To do a laparotomy for a moderate intraperitoneal hemorrhage is an inexcusable blunder, as it places life in needless jeopardy for a condition comparatively harmless in itself. When the extent of visceral disorganization is large, with few exceptions, the only effect of a laparotomy is to shorten life. In violent abdominal contusions after evisceration, we are frequently unable to immediately determine just what extent of gangrene may later follow.

In this class we should hesitate, deliberate, and weigh our cases well before we commit them to the last extremity of major surgery. I can recall about a dozen such cases in my own practice. Not a single one survived that was laparotomized. As the full course of anesthesia was approached their eyes closed, never to open again, for but few of them ever rallied from the shock. Three of such cases have come under my care within a year.

CASE I. A vigorous young man riding a "wheel" collided with a heavy watering-cart, was thrown down, both wheels passing over his body, just above the umbilicus. Brought to the hospital in desperate shock. Six hours later, rallied somewhat. Belly ballooned, hypersensitive, lower extremities drawn up, vomiting, but no blood, hands cold, pulse thready, bedewed in a cold perspiration, suffered agonizing pain till morphine was freely given. Hepatic dulness absent, abdomen tympanitic to percussion, rather of the character of *false* meteorism than true or *enteric*.

Operation. — Everything in readiness for complications or accident, etherization was begun and continued

[1] Lancet, January 20th.