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T A C H Y C A R D I A.

T H E S I S F O R T H E M.D. D E G R E E

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I have met with two cases of Tachycardia, one in a patient who also suffered from Raynaud's Disease, the other in a man who had symptoms of Locomotor Ataxia.

I have gathered together other reported cases of Tachycardia, the main facts of them I have tabulated, and along with them I now submit a brief account of the two cases in my own experience.

The condition is one of the greater interest because of the slight literature on the subject, and of the conflicting hypotheses as to its causation.

The following is the plan of this article:

PHYSIOLOGICAL:

In health the heart beats with a frequency varying within comparatively narrow limits for each individual. At birth the rate is 140, but this falls with each succeeding year until from 10 to 15 years of age the average is 90, while in the adult it usually varies from 65 to 75.

In women and in small persons the rate is slightly quicker.

The standing position, the early stages of effort, bodily or mental, swallowing, digestion, high external temperature, diminished atmospheric pressure, cause an increased frequency of the heart's action; Emotion, fear, anger, painful impressions accelerate it. Low arterial tension causes a compensatory increase in the number of the heart's contractions per minute.

Tarchanoff, Carpenter and others describe cases of persons who could quicken their hearts' action at will, by concentrating their attention on the heart beat. In Tarchanoff's case a rate of 120 per minute was reached, in other cases as high a rate as 160 is noted.

A natural tachycardia is found in some persons who are described as healthy.

West/

West gives a case with a rate of 120, male.
Allbutt " " " " 120, male.
Balfour " " " " 150, male
Binswanger " " " 150, female.

Acceleration of the heart's action has been often noted in pregnancy, apart from any other manifestation of disease. Cases are reported with a persistent rate of 100, and some as high as 120 - the rate falling to 70 or 80 after delivery.

The action of drugs in quickening the heart rate is often very complicated, as they may affect it from so many points of attack. Their action varies greatly with the dosage.

The Atropine Group, quickens the heart's action by paralysing the inhibitory endings of the vagus in the heart.

The Alcohol Group has a vaso-dilator action, it stimulates the heart reflexly from the mouth, gullet, and stomach, and it is a quickly available food. In long continued excess it may cause neuritis of the inhibitory terminations of the vagus in the heart, with consequent acceleration of the heart's action.

Ammonia accelerates the heart's rate reflexly. It also stimulates the vaso motor centre.

Nitrites act as accelerators chiefly through dilating/

dilating the vessels.

The Aromatic Volatile Oils have a slight reflex accelerating action on the heart.

Thyroid Extract has a powerful accelerating action.

Tobacco, Tea, Coffee, taken in continued excessive quantities cause small, frequent, irregular contractions of the heart.

In the later stages of poisoning by most drugs, the heart's action is greatly accelerated. A rate of 180 to 240 per minute is recorded in a case of carbolic acid poisoning by Kronlein - this acceleration lasted for 2 hours before death.

PATHOLOGICAL:

Most departures from health are followed by increased frequency of the heart's action. This is specially evident in diseases of the heart and blood vessels, and of the nervous mechanism which controls them.

Thus in diseases of the Pericardium, Myocardium, Endocardium, in valvular affections with failure of compensation especially of the Mitral valve, in cardiac dilatation and heart failure we meet with acceleration of the heart beat - also in arterio-sclerosis/

sclerosis with its consequent alterations of the blood supply to the cardiac nerves and centres.

Pyrexia per se causes a quicker rate, apart from any action on the vaso motor and cardio motor centres.

In Acute Specific Diseases there are many factors at work in the same direction; pyrexia, toxaemia, vaso-motor dilatation, exhaustion. Most of these diseases have been followed by neuritis, some more often than others, especially diphtheria, variola, septicaemia, measles, typhoid, typhus, influenza, pneumonia.

In some cases the neuritis has attacked the terminations of the Vagus in the heart as in Dr Frost's case, where three weeks after an attack of diphtheria the heart beat was 180 per minute. For a week it remained between 160 and 180 and then fell gradually. During this time the patient felt quite well and was anxious to get up and go about. There was no other nervous disturbance in this case.

The tachycardia noted as following influenza has been ascribed to affection of the heart muscle by the toxin, but the analogy of other organismal tachycardias points rather to a neuritis.

Bidon's case had a heart rate of 120 per minute.

Grödel's " " " " " " 130 " "

In/

In other cases the toxins appear to poison the cardio motor centre, as in some cases of typhoid in which the acceleration of the heart is not proportional to the temperature which may be as high as 104°, while the pulse is between 90 and 100. These cases shew an unusually high mortality.

Illustration of septicaemia tachycardia is found in Fiessinger's case of a woman who suffered from puerperal septicaemia. Eight days after the subsidence of the fever the heart's contractions were 190 per minute. After 10 days furthur the rate fell to 80. There was no recurrence.

In Lowit's case a girl had an ulcer the size of a bean on one tonsil. In a week later the heart rate went up to 252 per minute. respiration 28 - she recovered in 2 to 3 weeks and had no recurrence. There was no organic disease of the heart.

Peripheral neuritis occurs in Beri-beri, and tachycardia is frequently observed.

Toxins from perverted glandular secretions, from deranged metabolism, or disordered processes of digestion or excretion may affect the heart's action similarly. Thus peripheral neuritis occurs in diabetes, and cases of tachycardia are occasionally observed in this disease.

In Exophthalmic Goitre with increased thyroid secretion/

secretion tachycardia is constantly present, though not due to a peripheral neuritis.

Tuberculosis, Cancer, and diseases accompanied by cachexia and exhaustion often shew a high heart rate; 168 was noted by Traube for some weeks in a case of extensive tuberculosis; 175 by Eccles in a case of cancer. Toxaemia too may be a factor in these cases.

Gout is often associated with heart disease and arterio sclerosis. Many cases of tachycardia have been noted in gouty patients, the heart rate seldom being very high.

Osteo-Arthritis, according to Duckworth, is occasionally complicated by Tachycardia.

NERVE LESIONS:

Compression of one or both vagi by tubercular glands, or by mediastinal cancer or sarcoma is recorded in 30 cases. Martius collected 24 of these.

150 was the highest pulse rate per minute in these 24.

120 to 130 was the rate in most of them

120 to 130 " " " " Renaud's case.

120 to 130 " " " " Hayem's "

130 to 140 " " " " Ludet's "

176 " " " " Luzet's "

It/

It is noticeable that the rate is fairly uniform for so large a number of cases.

Jaquet's case, which had a pulse rate of 176 was complicated by fever.

Weil's case, with a rate of 216, was in the last stages of disease, with tracheo-stenosis, and here the rate fell to 150 when the tracheal condition was relieved. In all these cases the tachycardia was persistent.

Compression of the vagi by aneurysm has been noted in Ziemssen's case, the left vagus was pressed on and flattened by an aneurysm - the pulse rate was 100. Talamon's case, where the right vagus was pressed, had a pulse described as "very frequent".

Peripheral neuritis of the vagus has been already alluded in connection with acute infectious diseases.

In Alcoholic Neuritis the vagus terminations in the heart may be involved, as in Dejerine's case, where the heart rate was 140 to 150 per minute, and the post mortem examination revealed degeneration of these fibres.

In lead poisoning acceleration of the heart is noted in Fraenkel's case of a man suffering from lead colic, who had a heart rate of 180, and who developed ordinary lead paralysis a few months later.

Tumours/

Tumours, Haemorrhage, Thrombosis - of the Brain or Medulla - may cause tachycardia.

186 per minute was the rate in Bitres' case. The patient, a male, 17 years old, was epileptic. He had this tachycardia for one month, after which the rate fell gradually to 90. At the post mortem examination a tumour was found in the cortex, involving the right leg and right arm areas.

160 per minute was the rate in Bunzl-Federn's case. The patient, a male, 32, had paralysis of the eye muscles, ptosis, loss of light reflex, no syphilis. The attacks lasted 30 minutes and recurred at irregular intervals. Dentu records a case of a coachman who was thrown from his carriage and fell on his head. The next day for ten hours his heart rate varied between 200 and 240. There was no recurrence.

Kelly gives a case of a man who injured his head by falling 40 feet. After this he had often a heart rate of 216, with profuse sweat and congestion of the head and neck. The attacks lasted a day and came twice a week.

Cerebral Meningitis is accompanied by a slow pulse in the early stages, but with the effusion and exhaustion of the later stages an extreme frequency may come on, varying from minute to minute with slight causes, the arterial tension being low.

In Irritative lesions of the Spinal Cord vaso motor disturbance is common, and with it is often associated acceleration of the heart's action. Instances are given of a rate of

140 in Glosso-labio-laryngeal Paralysis Ducheune. Charcot.

180 in Cerebro Spinal Meningitis Descroizilles.

160 " Acute Ascending Paralysis-Féré.

130 " Progressive Muscular Atrophy -- Charcot.

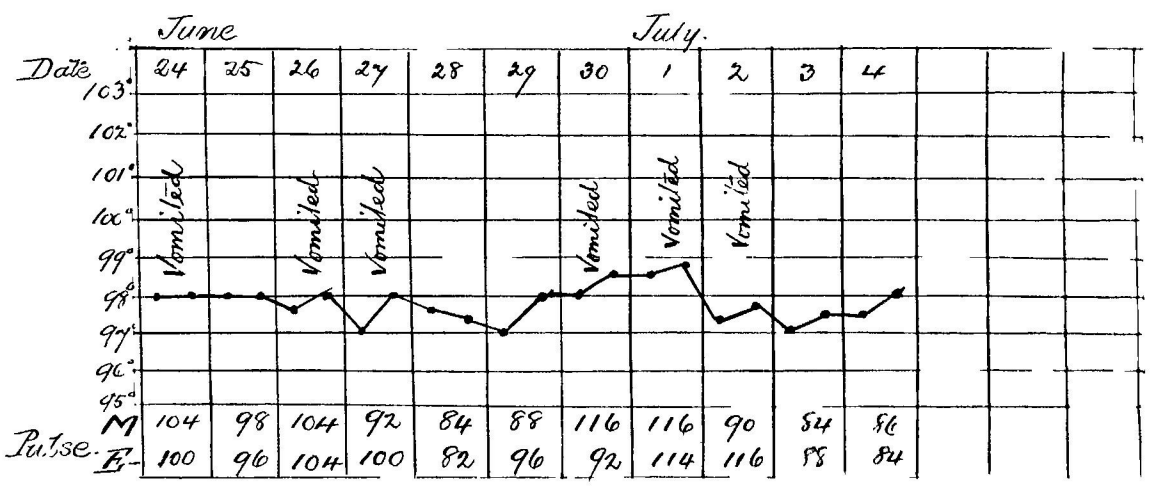
220 " Disseminated Sclerosis - Proebsting.

100 " Acute Myelitis - Kohler.

120 " Syringo Myelia. Ioffroy and Achard.

In Locomotive ataxia slight acceleration of the heart's action is common in the early stages of the disease, accompanying Gastric Crises, and usually in cases of Cervical Tabes. In 25 cases of Dr Byrom Bramwell's the pulse averaged from 87 to 90.

The subjoined chart shews the relation of the acceleration to the Gastric Crises in one of these cases.



Cases of Locomotive Ataxia have been noted where the frequency of the pulse was 120, and in one case 150. These higher rates occurring in the later stages of the disease, and being due according to Basso to degeneration of the intermedio lateral tract and adjacent nerve fibres in the upper part of the cord, traceable up to the medulla where it is near the accessory, the Glosso-pharyngeal, and the Vagus nuclei.

Exophthalmic Goitre is characterised by an increased heart rate, often 120 per minute, though it may be as high as 160. I. G. Dill has collected 11 cases of paroxysmal tachycardia with heart rates of 200 to 250 occurring in sufferers from Exophthalmic Goitre. Hoesslinn describes a similar case with a rate of 200 to 250.

In Neurasthenia tachycardia is one of the many expressions of the asthenic condition of the nervous system. Increased excitability, increased rapidity of fatigue causing considerable reactions to slight stimuli. Determann, Lehr Hofmann note 245 cases of Heart disturbance out of 575 cases of neurasthenia.

In Hysteria the heart's action is very easily disturbed and great acceleration is often observed.

In Epilepsy the rate may be slow, or it may be accelerated./

accelerated. Duchaussoy gives a case with a persistent rate of 120. Teissier gives a case in which paroxysms of tachycardia occurred at intervals for 12 years without any epileptic attacks. Epilepsy then came on and the attacks of tachycardia ceased. Talamon notes a case of tachycardia in which Epilepsy supervened for 2 years, during which time there was no tachycardia. He gives another case of Epilepsy where paroxysms of tachycardia came on independently of the convulsive attacks.

Leared's case had tachycardia during the Epileptic attacks.

Convulsions react on the heart rate similarly to severe exertion.

Chlorosis and Anaemia are accompanied by increased heart rate and great mobility of variation. Malnutrition of nerves and nerve centres, and of the heart muscle itself being contributory factors.

REFLEX TACHYCARDIA.

Many cases are caused by irritation in some other organ.

A Nasal Polypus was the cause of irritation in Spencer Watson's case of persistent tachycardia with a rate of 150 per minute. When the polypus was removed the heart resumed at once its ordinary action without further treatment.

Tapeworm/

Tapeworm was the irritant in Payne Cotton's case of recurrent tachycardia with a rate of 160 to 230 per minute, along with gastric disturbance and constipation. Under treatment the tapeworm was expelled and thereafter the tachycardia ceased.

A Floating Kidney was observed during life in Samson's case of tachycardia. There was no valvular disease of the heart but for six weeks the heart's action was 200 to 260 per minute. At the P.M. examination nothing pathological was found beyond the floating kidney.

A Floating Kidney in Taylor Seymour's case was associated with a heart rate of 260; in Eccles case with a rate of 245.

Gall stones are the irritant in several cases, the tachycardia coming on during the spasm of pain.

168 was the heart rate in Rommelaere's case.

216 " " " " " Sollier's case.

Distension of the stomach or intestine may affect the heart and abdominal aorta by direct mechanical pressure; the condition may also affect the heart reflexly. Hyper-acidity or other disturbance of the alimentary tube may act similarly reflexly on the heart. In these cases the heart disturbance disappears immediately on the removal of the primary irritation/

irritation in the alimentary canal.

200 was the heart rate in Klemperer's case of acid dyspepsia. He treated it with Soda Bicarb. and the heart rate fell in sequence.

220 to 250 was the rate in Preisendorfer's case. The dyspepsia here was consequent on loss of teeth and over-eating. Not only the dyspepsia was abolished by strict diet, but also the tachycardia.

It is noticeable that there is no recorded case of tachycardia in the graver diseases of the stomach and intestine.

Aspiration of the abdomen in a case of cirrhosis of the liver quoted by Dehio was followed next day by a heart rate of 180 per minute. The tachycardia lasted six days, when the rate became less frequent.

Uterine affections have often a similar reflex action. Here also the removal of the uterine disturbance is followed by the prompt disappearance of the tachycardia.

Menstruation suddenly interrupted was associated with a heart rate of 200 in Moon's case. Menstruation was re-established 14 days later and the heart rate fell to 80.

Prior to the first menstruation in a girl of 13, for four days the heart rate was from 212 to 236.

Menstruation/

Menstruation then came on and the tachycardia ceased. Draper's case.

During menstruation a pulse rate of 200 is noted in two cases, viz., Laache's case; Bowles' case.

Menorrhagia was accompanied by a heart rate of 200 in Eccles' case.

Retroflexion of the Uterus was the causal condition in a case with a pulse rate of 120 per minute. The uterus was replaced and then the tachycardia ceased. Theilhaben's case.

Uterine tumour was the irritant in the case noted by Taylor Seymour. Here the heart rate was 200.

Following immediately on Delivery, Gerhardt's patient had a pulse rate of 220 per minute for several hours, unaccompanied by pyrexia or excessive haemorrhage. The condition lasted some hours and did not recur.

Following instrumental delivery under chloroform Stocker's case had a heart rate of 200. Respiration 18 per minute. This continued 5 days, after which the rate fell gradually to 88.

Following immediately on abortion with haemorrhage Glanz's patient had a heart rate of 180 for some hours. Digital pressure of the Vagus in the neck was followed by a lowering of the rate to 112. There was no recurrence.

Many cases of tachycardia of slighter degree are noted in women at the age of puberty and at the menopause, which doubtless are also reflex in many instances.

PAROXYSMAL TACHYCARDIA.

There are still other cases of Tachycardia which cannot be classified as Reflex, Toxaemic, Specific: no lesion has been found in the nervous system to account for them, nor are they necessarily accompanied by organic heart disease.

First noticed by Payne Cotton in 1867, this variety of tachycardia has since been discussed by Bouveret, Larcena, Herringham, Hofmann, Martius, and others.

French writers accept Bouvert's nomenclature and speak of it as "Essential Paroxysmal Tachycardia". These cases are characterized by an extreme acceleration of the heart's action (usually 200 beats or more per minute; in some cases a rate of 300 is recorded) coming on suddenly, lasting minutes, hours, days or rarely weeks, ending usually in a sudden return of the heart's action to its ordinary rate.

These paroxysms recur at irregular intervals, and in the meantime the patient enjoys his usual health.

Organic/

Organic heart disease may coincide, more often it is absent. In the attack there is often a feeling of tightness in the chest, seldom any actual pain. The face is usually pale and there may be slight dyspnoea. Occasionally cramp and numbness in the limbs is complained of .

The pulse is small, weak, uncountable - the heart beats from 180 to 300 times a minute; there is diffuse praecordial pulsation and pulsation in the epigastrium and the neck. There is no murmur, the heart sounds are equal in tone and intensity, and equidistant - the diastole being shortened relatively to systole.

The patient rarely feels palpitation or distress. There is usually no giddiness, no pyrexia, no great increase in respiration rate. Dyspepsia or vomiting are often noted immediately preceding the attacks.

There are no signs of pulmonary congestion unless the attack is prolonged over 3 or 4 days. In longer attacks secondary symptoms of backward pressure shew, and venous stasis manifests itself. In 40 cases it is stated that there was no enlargement of the heart dulness - in 38 cases enlargement of the dulness is recorded, most of these being cases accompanied by organic heart disease. An increased secretion of urine is reported in 12 cases, more often it/

it is diminished. Albuminuria was found in the urine in 6 cases, sugar in 1.

Profuse sweating was observed in 6 cases.

At the end of the attack there is generally a feeling of exhaustion.

The tolerance of the heart for this extreme acceleration is very remarkable. In some of the cases these recurrent attacks have been borne for more than 30 years, in several for longer than 20 years.

Sex:

In 99 cases where the sex is noted, 58 are males, 41 females. Herringham in his 53 cases had 30 males, 23 females.

Age: In 97 cases where the age is given:

None	were	seen	from	1	to	10	years	of age.
5	"	"	"	10	to	20	"	" "
22	"	"	"	20	to	30	"	" "
25	"	"	"	30	to	40	"	" "
22	"	"	"	40	to	50	"	" "
16	"	"	"	50	to	60	"	" "
7	"	"	"	over	60.		"	" "

In 67 cases where the age at which the first attack came on is given:

4	cases	began	from	1	to	10	years	of age
12	"	"	"	10	to	20	"	" "
18	"	"	"	20	to	30	"	" "
15	"	"	"	30	to	40	"	" "
12	"	"	"	40	to	50	"	" "
4	"	"	"	50	to	60	"	" "
2	"	"	"	over	60		"	" "

These attacks had been borne:

for	more	than	30	years	in	3	cases.
"	"	"	20	"	"	5	"
"	"	"	10	"	"	15	"
"	"	"	5	"	"	15	"

Thus it is a disease of all ages, but chiefly of adult life. Though the tachycardia is borne for long, there are very few patients who live beyond 60.

In twelve of these reported cases, death ensued during an attack.

Trade or employment has no special effect.

Most of the cases occur in patients living in comfortable conditions.

In six cases hereditary transmission is recorded - in one family there were individuals of four generations affected. In seven cases there were other nervous disorders also present. Syphilis is recorded in three cases, but in one of them the tachycardia was noted years before the syphilis was acquired. There was a previous history of soft sore in two cases. Typhus was antecedent in five cases, pneumonia, empyema, malaria, were each recorded twice, influenza three times. One case began after Belladonna poisoning, and the paroxysms recurred for 15 years. One case dates from the taking of chloral for insomnia. In 12 cases there is a record of excess in alcohol or tobacco, or in both. In one case sexual excess is stated. In 67 cases no

valvular/

valvular heart disease is present - in 37 cases there is a history of heart disease.

The first occurrence of tachycardia usually follows severe exertion, prolonged strain, or great exhaustion. Later in the case slight provocations bring on an attack. Excitement, exertion, cold, stooping, getting up from bed, are frequent exciting causes. In other cases a drive or a railway journey brought on an attack, while in a few cases attacks came on in sleep, possibly precipitated by the assumption of a bad position or the excitement of a dream. In the later stages still the paroxysms may come on without any exciting cause. Thus the attacks become more frequent - they also become more prolonged and more severe.

There is at all times great irregularity in the duration of the attacks, and though they tend to lengthen, at times very short attacks may occur late in the case.

In two cases, repeated pregnancies were borne well by women subject to tachycardia - and several cases of pneumonia and infectious fevers recovered in spite of the patients suffering from tachycardia.

Two cases of paroxysmal tachycardia have come under by own observation:

Case I. was a professional man, aged 28, tall, strongly built, not neurotic. He was a famous foot-ball player and had been selected to play for his country. He took no alcohol, smoked little, and when I first saw him he had suffered from no acute disease. He had no valvular disease of the heart. He was 22 when he began to experience sudden short attacks of tachycardia, lasting from 5 to 30 minutes. He described the onset as instantaneous, the end as sudden. The rate of the heart's action was 160 to 180 per minute - his usual rate being 70. They were brought on sometimes by stooping, at other times they came on during the night, when he was in bed. During the attack he had a feeling of discomfort, but no actual pain; his face was pale; there was no dyspnoea, vomiting or gastric disturbance. The pulse was weak, of low tension, but countable at the wrist. There was no murmur audible over the heart, no increased area of cardiac dulness.

He was able to cut short the attacks by taking a deep inspiration and then holding his breath till he was blue in the face.

Most attacks came on when he was exhausted by long unbroken periods of work - but he was able to take severe exercise without causing attacks until two years later, when he suffered from acute rheum-

atism/

atism. After this time his mitral valve became incompetent. The lesion was compensated, hypertrophy of the left ventricle shewed itself, and he felt little inconvenience resulting, save that he could no longer take violent exercise on account of the breathlessness and palpitation which it caused.

Since the acute rheumatism the attacks of tachycardia have become more violent, more frequent, more prolonged. They are now accompanied by dilatation of the right side of the heart, the dulness extending to one inch to the right of the right edge of the sternum. After the attack the dulness soon recedes to the right sternal edge.

Holding his breath has now less effect in cutting short the paroxysms, and often fails to do more than lower the rate for a few minutes, after which it returns to the quickened frequency. Pressure on the vagus in the neck by the fingers has no effect. Strophanthus gives most relief of anything, but the attacks now continue from one to six hours.

This patient also suffers from Raynaud's disease. Slight cold causes tingling in the fingers of both hands, followed by numbness and inability to pick up small objects, though his fingers still appreciate heat and cold. This continues until all the fingers are quite blue. This conditions lasts

one/

one to two hours. In other respects the peripheral circulation is good.

The patient is alive and doing his work, but he regulates his life so as to avoid cold and chills as much as possible.

I have met with no other record of Raynaud's disease in connection with tachycardia, though several cases are noted of profuse sweating and other vaso-motor disturbance, of migraine, of epilepsy, associated with tachycardia.

The recurrent paroxysmal disturbance of the circulation, excited by insignificant causes - the transient nature of the attacks - the return to ordinary conditions between the attacks - the absence of any evidence of degenerative disease - are the outstanding features of both paroxysmal tachycardia and of Raynaud's disease.

When, as in this case, we find both diseases in the same person, we naturally ask may they not both be manifestations of the same undue irritability of the vaso-motor centres, which leads it to partial spasmodic over action in response to slight irritation.

The second case was under my care in Professor Fraser's Wards of the Royal Infirmary.

Patient/

Patient is a well-to-do Commercial Traveller, aged 42, little and thin. He lives in hotels, and indulges freely in alcohol and in tobacco. There is no history of any hereditary disease. He has not had rheumatism nor any organic heart disease. He had gonorrhoea and syphilis 14 years ago. For the last seven years he has had attacks of vomiting and gastric pain recurring every 6 to 12 weeks. For six months he has had a constant feeling of constriction round the abdomen, just below the level of the umbilicus - also sharp, darting pains down his thighs.

He has never noticed anything about his heart save a little palpitation on exertion.

He came to the Royal Infirmary complaining of vomiting which had lasted two days and of weakness.

On examination he was pale, anxious looking. There was no cyanosis, no dyspnoea, no giddiness, no palpitation, no praecordial pain. His pulse was uncountable owing to rapidity and weakness. The rate of the heart's action was 220 per minute, counted through the stethoscope on the apex. There was no murmur. The heart sounds were equal in intensity and tone and equidistant.

The right border of the heart was 1 inch to the right of the right sternal edge.

The left border was at the left nipple line.

The/

The upper border was at the second interspace. The whole praecordia pulsated visibly at each heart beat. There was epigastric, episternal, carotid pulsation. The liver reached 2½ inches below the costal margin, in the right nipple line. The artery walls were thickened. His voice was weak and husky. He had no cough. There was oedema of the bases of both lungs - no evidence of tuberculosis or pneumonia. The temperature was 98.6. Respiration 24. The urine was scanty, alkaline, sp.gr. 1014, free from pus, albumin, sugar, bile.

The vomited matter was milk, partly digested, with mucus, and it was yellow with bile. There was no affection of the mouth or gums. Sexual power was unaltered.

Sight was impaired slightly. The left pupil was small and contracted. The light reflex was absent, though both eyes reacted to accommodation, the left one much less than the right. There was no disease of the fundus. The external ocular muscles were unaffected.

The sphincters of the bladder and anus were weak.

The abdominal and cremasteric skin reflexes were not obtained. There was no knee jerk. The muscular sense was good.

In addition to the sensory disturbances already noted/

noted, there was found numbness of the soles of both feet with lessened sensibility to touch in the soles. There was no tenderness on pressure on the calves of the legs, or in the arm muscles. Appreciation of pain and temperature was unaffected. The muscles of the legs were rather poor, but still not weak. The gait was not ataxic, but was rather hesitating and uncertain. He could stand with both feet together and his eyes shut.

Though urged to stay and go to bed at once, he insisted on first seeing to his business affairs, returning to his hotel and coming back to the ward two hours later, more exhausted than before.

Next morning his heart's action was 100 per minute, arterial tension improved, but still very low. Taking too large a drink of milk he vomited very energetically, and at once his pulse rate went to 200, and for 13 days it ranged between 182 and 210 per minute. He was given iced milk in small quantities and was ordered

Sod. Bromid.	gr. xx.	
Phenacetin	gr. v.	t.i.d
Nepenthe	m. xx.	

Three days after admission, the heart rate was 210. Respiration 24. Temperature 97.5. The face neck and chest were deeply cyanosed. The jugular veins/

veins were engorged, but not pulsating. There was a circle of engorged superficial veins round the trunk at the level of the attachment of the diaphragm. The right border of the heart was two inches to the right of the right sternal edge - the other borders unaltered He was dull and dazed. Urine and faeces were passed in bed involuntarily.

Six days after admission: Condition unchanged. Heart rate 192. Respiration 24. Temperature 97.8. He was ordered in addition, Tinct Belladon. m.v., t.i.d

Eleven days after admission: He took milk and beef tea freely and retained them - otherwise no change. Heart rate 185, Respiration 18., Temperature, 96.5

Fourteen days after admission: Heart rate 190, respiration 20, temperature 95°.

Belladonna was stopped. At noon he began taking Tinct.Strophanth (B.P. 1885) m.v., four hourly. Pulse at 8 p.m. 190.

Fifteenth day: Pulse 128, respiration 20, temperature 97.

Sixteenth day: Pulse 89, respiration 20, temperature 97. The arterial tension had improved. The pulse now approximates to an ordinary one; urine is/

is much increased; oedema is less; cyanosis has disappeared. He is clear headed and anxious to get up. The sphincters are still weak but no longer are urine and faeces passed in bed.

During convalescence there were occasional attacks of acceleration of the heart rate, lasting up to one hour, the pulse attaining a rate of 120 to 130 per minute.

He was discharged the 41st day., Pulse 90, respiration 16, temperature 98.

Six weeks later he wrote to say he had had no recurrence and was able to do his work.

He continued working for nine months, and then had another attack which necessitated his staying in bed six months. Since that he has for 12 months been in his ordinary health.

The sensory disturbances already described are greater, and he has increasing unsteadiness in walking .

In this case there is a history of syphilis. Some of the symptoms of locomotor ataxia are present, others are absent. Alcohol and tobacco had been taken freely.

To recapitulate some of the heart phenomena: on admission the heart rate was 220 per minute; next day/

Day after admission - Rate 100.

4 Days after admission- Rate 210.

23 days after admission- Rate 86.

31 days after admission - Rate 80.

day it fell to 100, but on severe vomiting it rose suddenly to 200 and stayed for 13 days between 182 and 210. Then it fell gradually, the first decline being noticed 18 hours after treatment by strophanthus was begun.

Sodium Bromide, Phenacetin, Belladonna has been given previously without evident effect.

Where tachycardia has been noted in cases of Locomotor Ataxia the heart rate has exceeded 120 per minute in but one instance, and in that case it was 150.

We know from the researches of Pierret, Déjerine, Pitres, Oppenheimer and Siemerling that peripheral neuritis is common in tabes, affecting the sensory nerves ending in muscle, skin and joints.

If the tachycardia were persistent, and if the heart rate were between 120 and 150, we might attribute it to neuritis of the inhibitory terminations of the vagus.

Charcot accounts for these tachycardias in Tabes by ascribing them to irritation of the sympathetic in the cervical cord by the ascending tabelic lesion - which he says may also through the vaso-motor system disturb the circulation in the medulla and so affect the cardiac centres directly.

Basso/

Basso, Tripiére, Pierret, find the explanation of the tabetic tachycardia in degeneration of the intermediate lateral tract and adjacent nerve fibres in the upper part of the cord, traceable up to the medulla in close proximity to the vagus nuclei.

This degeneration has been found in cases of tabetic tachycardias.

These possible explanations are of considerable interest generally, but they are of less moment in the case under consideration. In this case the acceleration of the heart was so extreme, and so paroxysmal as to be widely different from the recorded cases where the average rate was from 100 to 113.

Further, in the recorded cases the tachycardia was either persistent, or if it did come and go, it was always in close relation to gastric or other visceral disturbances, coming with the visceral attack, going when the visceral condition went.

Quite otherwise was it in the present case, the tachycardia persisting 12 days after the complete cessation of gastric troubles.

The number of cases of Locomotor Ataxia seen in hospital is very large. They are met with by the hundred. In hospital they are under careful continuous observation. As we find so few cases recorded of/

of severe tachycardia among them, it is probable that it seldom occurs in extreme degree. If in the very large number of cases observed there has been none at all approaching the present case in acceleration, in paroxysmal character, in continuance long after the subsidence of the accompanying gastric disturbance - we may fairly conclude that the conjunction of paroxysmal tachycardia and locomotor ataxia in this case was accidental - that the cause of the extreme tachycardia is not to be sought in the antecedent locomotor ataxia, but as in other cases, with unknown origin it is to be looked for in some alteration of nutrition or other predisposing disturbance of the cardiac centres.

Bristowe gives a case where there was a similar accidental combination of tachycardia and syphilis - the tachycardia having begun at the age of 8, the syphilis having been acquired at 18.

Post Mortem examinations have been made in 13 cases of tachycardia. In some of these the sectio was done by an ordinary practitioner, who describes the macroscopic appearances merely. In three cases the heart nerves, vagus and sympathetic were more carefully examined. In one case (Hoffmann's) a very careful series of observations was made of the brain, medulla/

medulla, cord, nerves. Modern methods of staining being employed and comparison sections from healthy nerves and brain were submitted to the same stains at the same time. No lesion of any part of the nervous system was found.

In six cases dilatation of the heart was reported, in some cases with hypertrophy. Myocarditis was found in six cases, little in some cases, more in others. Fatty degeneration was noted repeatedly The valves were usually competent.

There are notes of one P.M. examination where tachycardia had been associated with a floating kidney. Here nothing pathological was found beyond the displaced kidney.

PROGNOSIS:

Prognosis in tachycardia associated with heart disease is governed largely by the heart condition. The accompanying lesion is usually mitral, when the prognosis is less unfavourable than in case of aortic incompetence. The existence of myocarditis renders the prognosis very grave. Myocarditis was found at the post mortem examinations in every case where death had occurred during an attack. Any extreme degree of dilatation similarly affects the prognosis

If in the intervals between the attacks the heart/

heart is not much enlarged, if the pulse is regular with a moderate arterial tension, the prognosis is more satisfactory. The frequency, intensity, and duration of the tachycardia in so far as they are increased, affect the prognosis unfavourably there being so much the greater danger of further damage to the heart and of ultimate heart failure.

The tachycardia occurring in the course of infectious diseases is always ominous, though many cases of recovery are noted where the acceleration has come on 2 or 3 weeks after the crisis of the disease.

In these cases there has been time for the general strength to recuperate, and they are indications rather of neuritis of the vagus than of acute dilatation.

In cases associated with compression of the vagus by tumours, tachycardia is usually an indication of the last stage of the disease.

In exophthalmic goitre the prognosis is serious in proportion to the degree of acceleration. If the rate keeps high in spite of treatment, death follows from exhaustion.

In neurasthenia and hysteria the prognosis is good. The Weir Mitchell treatment by extra feeding coupled/

coupled with massage, often proves very efficacious.

In reflex cases the prognosis is good, the tachycardia disappearing on the removal of the source of irritation.

In paroxysmal tachycardia while many cases shew a long endurance of the attack, there are few cases of complete cessation. One case is given where they ceased for one year, and in another case for seven years, but in both the tachycardia returned and continued.

The tendency of the attacks to increase in intensity, duration, and frequency, causes a strain that wears out the heart even if sudden death does not come during an attack.

There is also the chance that organic heart disease may at any time be superadded.

So that every case is serious, and in those cases accompanied by heart disease the prognosis is very unsatisfactory.

TREATMENT:

The first necessary is complete rest, bodily and mental, and in particular the avoidance of anything that is known to be an exciting cause.

Nutrition needs especially to be maintained, while avoiding any risk of undue strain on the stomach/

ach. The bowels must be carefully regulated. Any source of irritation which may act reflexly on the heart must be removed.

The various mechanical means of treatment are all simple and readily applied, so they should be tried.

Deep breathing, followed by forced holding of the breath, was tried in 12 recorded cases. In two it failed completely; in two it caused temporary lowering of the rate, and in two it answered for several years, then failed to cut short later attacks, though it still lowered the rate for the moment; in six cases it was completely successful, and continued to be so. It is particularly effective in neurotic cases.

Pressure over the heart combined with compression of the abdomen is said to have cut short the attack in two cases. Low position of the head below the level of the body was said by Honigmann and by Mayer to be successful. Pressure by the fingers on one vagus in the neck was tried in 13 cases, and proved successful in 7 of them. The pressure was applied at the level of the upper edge of the thyroid.

Icebags over the heart, mustard poultices to the praecordia, and to the nape of the neck have not been effective.

Of drugs Strophanthus, Digitalis, Morphia, and the Bromides are most useful. Morphia acting chiefly to/

to relieve the anxiety and unrest. Digitalis is effective but slow in its action. Grodel says in his case it made the man worse. Farquharson had no good result from it after along trial. Faisaus tried it in vain, and then had recourse to Strophanthus which succeeded.

These three cases are exceptions to the general experience. Strophanthus has always been effective The B.P. 1885 preparation is preferable to the tincture at present used. Amyl Nitrite and the Nitrites are contra-indicated.

The irregular duration of the attacks is a confusing element when estimating the effect of a drug, as the end of the attack may coincide with the administration of a drug that has not been effective.

Belladonna, Caffeine, Ether, Ether Spray, Faradisation of the neck are the remedies relied on by the French medical men - but they have not proved generally so useful as strophanthus and digitalis. These two drugs by their direct action on the heart muscle slow the heart rate, but their peculiar effectiveness lies in the fact that by increasing the heart's output they supply the brain and so the heart centres with an increased amount of blood, thus remedying the medullary anaemia which is a cause of the condition in many cases.

CAUSATION OF TACHYCARDIA:

The direct nervous mechanism of the heart includes the cardiac centres in the medulla, the cardiac branches of the vagus, the accelerator nerve from the sympathetic, the ganglia in the heart, and afferent nerves from the heart.

Indirectly the vaso-motor nerves exert a powerful influence on the heart, by their effect on arterial tension, and by their affecting the blood supply of the cardiac centres.

In an ordinary healthy person the heart tends to beat at a rate of 180 per minute, but this is checked by the cardio-inhibitory centre, which through the inhibitory fibres of the vagus, exercises a constant restraint upon the heart, and reins it in to a rate of 65 to 75 per minute. In proportion as this inhibition is lessened from time to time, so does the heart rate increase in frequency, until when the inhibition is wholly removed, as by atropine, a rate of 180 may be obtained, so that a great range of frequency is possible by means of this varying inhibition alone. A few accelerating fibres run in the vagus. But in addition there is an accelerating centre which sends its impulses to the heart through the accelerator nerve from the sympathetic system.

Experiments/

Experiments in man are not available, but in other mammals section of the accelerator nerve does not slow the heart, so this nerve ordinarily is not in action.

When its distal end is stimulated in the neck, acceleration of the heart's action comes on gradually, when the stimulation is stopped the heart returns to its former rate gradually. The greatest acceleration thus obtained is equivalent to increase of 50 to 70% in the rate.

The exact function of the heart ganglia in man was for long doubtful. Recent research would teach us to regard them as part of the great group of ganglion cells situated on the course of the efferent nerves supplying the viscera, and as being devoid of special function in any great degree, other than that of nutrition to the nerve fibres proceeding from them (Schafer) Afferent fibres from the heart connect with the nervous centres.

The quickening of the heart which occurs physiologically is due to lessening of the activity of the cardio-inhibitory centre in response to lessening arterial pressure, to increased CO_2 in the blood, or to afferent impressions from the heart or from sensory nerves.

The tachycardia occuring in the course of organic heart disease is usually a compensatory action,

quickened action atoning for the diminished effectiveness of each contraction, and so this tachycardia is often a proof of the nervous mechanism of the heart being intact. The tachycardia which is seen in acute infectious diseases is due partly to pyrexia, to vaso-motor dilatation, to exhaustion; but a more important part still is due to the organismal toxins. These affect the heart muscle, or poison the nerve centres, or act like atropine, or cause a neuritis of the inhibitory terminations of the vagus in the heart corresponding to the peripheral neuritis more frequently seen affecting the terminations of the motor nerves. The incidence of this neuritis does not appear to depend on the severity of the primary disease, but on peculiarities in the specific organism, such as determine the special characteristics of different epidemics of influenza - and also on the degree of vital nutrition in each case.

When this tachycardia appears in the acute stage of the disease, it is often a sign of degeneration of the heart muscle - the neuritic tachycardia more often comes on in 2 to 3 weeks after the acute stage.

In the peripheral neuritis of alcohol, lead, or other poisons, the tachycardia is again often due to vagus neuritis.

The tachycardia occurring in case of compression of the vagus by glands or tumours, is due to lessened inhibition/

inhibition. The rate in these cases never attains 180, which was obtained by Von Bezold in vagus paralysis by atropine. This may be due to some fibres escaping the general destruction.

The tachycardia seen in irritative lesions of the spinal cord has been already discussed in connection with my second case.

The tachycardia of exophthalmic goitre is accompanied by perspiration, diarrhoea, and vaso-motor dilatation, indications of disturbance of the sympathetic. But in view of the alterations which occur in the thyroid, and of the fact that similar results can be produced experimentally by the administration of thyroid extract, we may regard this as a toxic tachycardia.

The tachycardia of neurasthenia and anaemia is due to impaired nutrition. The heart muscle suffers and the nutrition of the nerve elements is altered. Probably there is some chemical change which so affects them as to produce irregular action. Further, these neurotic states keep the innervation of the vessels in a very sensitive condition.

The tachycardia reported in epilepsy may be due to spasm of the arteries supplying the cardio-inhibitory centre, spasm being so general a characteristic/

teristic of epilepsy. The anaemia thus caused with the resulting tachycardia being analogous to the loss of consciousness caused by anaemia of other areas of the brain.

Reflex tachycardia occurs physiologically in response to painful sensations, but with a less degree of acceleration than in the cases already described. Spencer Watson's case of nasal polypus previously given, is the only case of this affection recorded in connection with tachycardia, yet how large a number of nasal polypi are dealt with every year.

Similarly uterine troubles are the common lot of women, yet relatively few are accompanied by tachycardia, and these reported cases are in no point exceptional otherwise. How comes this tachycardia in these few quoted cases, and not in every one?

No lesion has been found post mortem in any part that would account for it.

The analogy between reflex tachycardia and reflex epilepsy is suggestive. Many cases are reported of epilepsy induced by irritation of the teeth, nose, or ear, by worms, by gastric irritation, the passage of gall stones, and in one instance it followed aspiration for pleuritic effusion. In these cases a predisposition to epilepsy would be granted - and in the cases of reflex tachycardia we need to assume similarly a predisposition.

PAROXYSMAL TACHYCARDIA.

Seeing that the sympathetic supplies the accelerating, the vagus the inhibitory impulses to the heart, while the heart ganglia are without special function save a nutritional one (Schafer), it is no wonder paroxysmal tachycardia should be attributed to paralysis of the vagus or to irritation of the sympathetic, or to a conjunction of both causes.

Others have found the explanation in suggested paralysis of the vagus terminations in the heart.

Herringham supposes disease of the nerve endings in the heart muscle, in addition to fibroid disease of the myocardium to be a reasonable hypothesis.

Talamon looks on the disease as essentially epileptic, heart acceleration replacing muscular convulsion.

Bouvert calls it "the intermittent manifestation of a persistent functional disease."

Debove, Boulay, Hoffmann, Larcena, ascribe it to a bulbo-spinal lesion at once paralysing the cardio-inhibitory and stimulating the sympathetic.

Is it to be attributed to a lesion located in the heart, affecting the muscle, the heart ganglia, or the nerve endings?

If myocarditis were the cause we should find signs of it in the intervals between the attacks, but irregularity of the heart's contractions which

characterizes myocarditis is conspicuously absent save in the last stages. The myocarditis found post mortem in these cases was invariably recent, and probably it accounts for the fatal termination - whereas the paroxysmal tachycardia in one case had been recognised for 24 years (case No.3), in another for 20 years (Case No.4). The degeneration of the heart muscle is secondary to the tachycardia.

That the cause is paralysis or degeneration of the heart ganglia, or of the vagus nerve endings in the heart is opposed by the slowing of the pulse on pressing the vagus in the neck, treatment practised successfully in seven cases. The intervals of good health between the attacks are incompatible with a neuritis.

No degeneration has been found in these parts of the nervous system, though it was carefully looked for in several cases post mortem.

That the ganglia have any function that could conceivably affect the heart rate to this extent is denied by recent researches of Schafer and of Engelman. Alteration in the calibre of the coronary arteries has been suggested - but in no case has it been found post mortem.

Paralysis of the vagus in its course would involve other vagus branches, in this case too we should not/

not get the slowing of the heart rate on pressing the vagus in the neck, further no degeneration of the vagus has been found post mortem in cases of paroxysmal tachycardia.

Irritation of the sympathetic is another hypothesis. Little is known of the pathology of the sympathetic but experimental stimulation of the accelerator nerve gives an increased heart rate gradual in onset and in disappearance, and not of long duration - thus widely different from that seen in parozysmal tachycardia.

Bulbar Lesion is not recorded accompanied by Tachycardia. No such lesion has been found post mortem in any of the cases.

In the cases of paroxysmal tachycardia the post mortem examinations throw little light on the vexed question of causation, and thus they harmonise with the absence of clinical evidence after the subsidence of an attack.

Probably different factors are at work in different cases of paroxysmal tachycardia, but in all cases there must be a predisposition.

The analogy between paroxysmal tachycardia and Raynaud's Disease is striking. In both there is circulation disturbance, paroxysmal, transient, recurrent, with intervals of good health between attacks: in both there is an entire absence of any evidence/

The two cases following immediately on delivery (page 16) are akin.

The successful treatment of many cases by Strophantus and Digitalis bears out this view, their effect being to cause a larger supply of blood to the brain.

A predisposition to tachycardia may be inherited as in cases 6, 7, 8, 9, 10.

In other instances though no direct inheritance can be traced, we find evidence of other disturbance of the central nervous system, indicative of a general neuropathic inheritance. Two patients had relatives who were insane, one patient was himself insane, epilepsy was noted in four cases, in two cases there is a history of hysterical hemiplegia.

Altered nutrition of the cardiac centres, influencing the nerve elements so as to cause irregular and defective action may in other cases predispose to tachycardia.

This predisposition may in some instances be due to the action of toxins - the cardiac centres having a special susceptibility for these special poisons, as other particular parts of the nervous system have for curara or atropine.

The large percentage of cases of paroxysmal tachycardia/

tachycardia accompanied by valvular disease of the heart may be due to there being two factors in these cases tending in the same direction. In addition to the predisposition the continuous afferent impulses from the injured heart, from the unfilled vessels, from the unfed tissues of the body, all tending to call forth increased frequency of the heart's contractions to compensate for their insufficiency.

In all cases after the first paroxysm there would be a residual tendency, which would increase at each repetition.

In harmony with this is the fact that while the first attack of paroxysmal tachycardia is usually precipitated by some severe exertion or strain, succeeding attacks are brought on by slighter exciting causes, and in the later stages they occur spontaneously.

To sum up,

1. The cause of paroxysmal tachycardia is not uniform in every case.

2. A predisposition is present in all cases. What that predisposition is eludes us.

3. The paroxysmal tachycardia is probably due to disturbance in the cardiac centres. The analogy between paroxysmal tachycardia and other paroxysmal diseases attributed to central causes supports this view.

4. In some cases this action is secondary to alterations in the blood supply of the centres, as in the case associated with Raynaud's disease, and others.

5. The predisposition may be caused by altered nutrition of the nerve elements of the cardiac centres.

6. Special toxins may have a predilection for the cardiac centres, and so cause the predisposition.

7. In cases of paroxysmal tachycardia associated with heart disease we have two factors tending in the same direction, hence the large number of cases of this kind.

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REPORTED CASES OF PAROXYSMAL TACHYCARDIA.

	Author.	Publication.	Sex	Age	Rate	Other particulars.
1.	Preston	Present thesis	M.	28	160-180	Raynaud's disease associated.
2.	Preston	Present thesis	M.	42	180-210	Locomotor ataxia associated.
3.	Brieger	Charitée Annalen 1888.	F.	33	250	Attacks began at 9 years of age.
4.	Hoffmann.	Die Paroxysmale Tachycardie, Wiesbaden.	F.	44	204-244	Attacks began at 24, attacks alternated with migraine
5.	Hoffmann.	do.	F.	39	198-208	Attacks alternated with migraine.
6.	Freyhahn.	Deutsche Med.Woch. 1892.	F.	42	200-228	Inherited.
7.	Faisans.	Soc. Med. des Hopitaux, 1890.	F.	50	200	Daughter had tachycardia.
8.	Faisans.	L'Union Médicale, 1891.	F.	25		Mother had tachycardia.
9.	Oettinger.	Semaine Médicalè, 1894.	M.	53	180	Tachycardia in 4 generations, grandmother, mother, self, son.
10.	Bowles.	Brit. Med. Journ. 1867.	F.	45	180-250	Sister was insane.
11.	Buckland.	Trans.Clin.Soc., Vol. 25.	F.	11	215	Grandmother insane Father had general paralysis.
12.	Kelly.	Med, & Surg. Reporter, 1896.	M.	60	216	Followed injury to head.
13.	Talamon.	Semaine Médicale 1891.	M.	53	200	Followed injury to head
14.	Huppert.	Berlin Klin.Woch. 1874.	M.	40	200-224	Patient maniacal.
15.	West.	Lancet, 1870	F.	26		Sister was insane.

No.	Author.	Publication.	Sex	Age	Rate	Other particulars.
16.	Huber.	Arch.f.Klin.Med. 1891.	F.	22	258	Had two attacks of Hysterical hemiplegia
17.	Teissier.	Ass. p. l'avance des Science, 1876			150	Tachycardia of 12 years, succeeded by epilepsy.
18.	Talamon.	Sem. Méd., 1891.				Tachycardia occurred during epileptic attacks.
19.	Talamon.	Semaine Médicale, 1891.	F.	65	150	Epilepsy replaced Tachycardia.
20.	Allbutt.	System of Medicine	M.	48	166	Epileptic.
21.	Allbutt.	System of Medicine	F.	45	160-280	No valvular heart disease.
22.	Bensen.	Berlin.Klin.Woch, 1880.	M.	52	180-200	do.
23.	Bouveret.	Revue de Médecine 1889.	M.	50	200-230	Excessive use of tobacco & coffee.
24.	do.	do.	F.	36	200-240	Died in attack.
25.	do.	do.	M.	45	200	
26.	Bressler.	New York Med.Rec. 1888.	M.			Valvular heart disease
27.	Brisbane	Lancet, 1875	M.		185	do.
28.	Bristowe.	Brain, Vol.X.	F.	30	180-246	
29.	do.	do.	M.	19	240-250	No valvular heart disease.
30.	do.	do.	M.	34	160-200	Valvular heart disease
31.	do.	do.	F.	25	200-250	do.
32.	do.	do.	F.	55	224	Died in later attack.
33.	Broadbent.	B.M.J., 1867.	F.	66	168	Valvular heart disease.
34.	do.	"The Pulse"	F.	49	152	History of Rheumatism

No.	Author.	Publication.	Sex	Age	Rate	Other particulars.
35.	Broadbent.	"The Pulse"	F.	25	200	
36.	do.	do.	F.	45	200-240	
37.	do.	do.	M.	36	215-230	Hemiplegia ensued three months later.
38.	Brown.	B.M.J., 1867.	F.	41	160-292	Died in later attack.
39.	Bunzel-Federn.	Prag. Med. Woch. 1891.	F.	33	192.	Valvular heart disease.
40.	Castaing	Tachycardie, Paris	F.	26	200	
41.	Cavafy.	B.M.J., 1875.	M.	32	228.	Heart disease (valvular) Followed long starvation in Siege of Paris.
42.	Cotton.	B.M.J., 1869.	M.	35.	200	
43.	Debove	Soc.Méd des Hopitaux, 1890.	F.	26	184-200	
44.	Edmunds.	B.M.J., 1867.	M.	35	200	
45.	Farquharson	B.M.J., 1875.	M.	28	210	Valvular heart disease.
46.	Fraenkel	Berlin Klin. Woch. 1875.	M.	30	180	
47.	Fraentzel.	Charitée Annalen 1889.	M.	35		History of Typhus. Died in later attack.
48.	Fritz.	Tachycardia Paroxysmalis, Zurich.	M.	41	240-320	Myocarditis, Excess in tobacco.
49.	do.	do.	M.	40	180-200	History of Typhus.
50.	do.	do.	M,	41	180-224.	Heart disease (valvular)
51.	Gibson	Diseases of the Heart & Aorta.	M.	71	250.	
52.	Grödel.	Berlin Klin. Woch, 1890.	M.	44	148	
53.	do.	do.	M.	57	180-200	
54.	Hampeln	Deutsch.Med.Woch. 1892.	M.	47	240-280	
55.	Haussler.	Corresp. f. Schweizer-Aertze, 1894.	M.	54	200-216	History of Rheumatism.

No.	Author.	Publication	Sex	Age	Rate	Other particulars.
56.	Herringham	Edin. Med. Journ. 1897.	F.	11	240-260	Began at 6 years of age. No heart disease.
57.	Hochhaus	Arch. f. Klin. Med., 1892.	M.	26	200-210	
58.	Hoffmann.	Die Paroxysmale Tachycardie, Wiesbaden.	M.	29	200	History of Sexual Excess
59.	do.	do.	M.	36	180-200.	Neurotic - Grandmother weak mentally.
60.	do.	do.	F.	38	204	
61.	Honigmann.	Deutsche Med. Woch., 1888.	M.	31	160-210	Valvular heart disease. Treated by low position of head.
62.	Huchard	L'Union Médicale, 1879.	F.	60	250-300	
63.	Kelly	Med. & Surg. Reported, 1896.	F.	22		
64.	do.	do.	M.	50	210-220	Followed on influenza.
65.	Kredel-Riegel	Arch. f. Klin. Med. 1892.	F.	16	144	Valvular heart disease.
66.	Laache	Norsk Mag. f. Laegevidenskaben 1898.	M.	23	200-240	Valvular heart disease. Excess in tobacco.
67.	do.	do.	M.	53	170-208	Hemiplegia came late and then tachycardia ceased.
68.	do.	do.	M.	47	160.	Valvular heart disease Died in attack later.
69.	Langer	Wien.Med.Woch. 1881.	M.	52	200.	
70.	Larcena.	Tachycardie, Paris.	F.	43	180-200	History of chorea.
71.	Loeser.	Virchow's Archiv. 1896.	M.	51	160-196	Valvular heart disease. Excess in alcohol.
72.	Martius	Berlin Klin.Woch. 1889	M.	27	200	
73.	Mayer.	New York Med. Monatsschr. 1893.	F.	26	234	Valvular heart disease.

No.	Author	Publication	Sex	Age	Rate	Other particulars.
74.	Nothnagel	Arch. f. Klin. Med., 1876.	M.	35	160-182	Valvular heart disease.
75.	do.	do.	M.	28	208-248	do. do.
76.	Nunnely	Lancet, 1871	M.	59		
77.	Oliver.	B.M.J., 1891.	M.	28	190-230	Valvular heart disease.
78.	Osler.	Medicine.	M.	87	200	Began at 37 Treated by strong coffee.
79.	Pechardre	L'union Médicale 1889	M.	57	280-300	Preceded by an aura
80.	Perrin.	do. 1890.	F.	60		
81.	Pribram	Neurose des Vagus	F.	20	200-300	
82.	Rommelaere.	De l'acceleration Cardiaque. Bruxelles.	M.	55	170	
83.	Rosenfeld.	Congr. f. innere Med. 1893.	F.	40	180-200	Valvular heart disease.
84.	Roth.	Prag. Med. Woch. 1884.	F.	70		Began at 18. Valvular heart disease.
85.	Spengler.	Deutsche.Med. Woch. 1887.	M.	26	250	
86.	Strauss	Charitée Annelen 1898.	M.	39	200	Excess in alcohol.
87.	Trechsel.	Rev.Méd. de la Suisse. Romande 1893.	M.	43	170	History of typhus.
88.	Tuczek.	Arch. f. Klin. Med. 1878.	M.	36	190-208	
89.	Watson.	B.M.J., 1867.	M.	45	216	Died in later attack.
90.	West.	Lancet, 1870.	M.	33	250	Valvular heart disease.
91.	do.	Lancet, 1890.	M.	32	252-300	do. do.
92.	Williams.	Bristol Med.Jour. 1897.	F.	62	200-240	do. do.
93.	Winternitz.	Berlin.Klin.Woch. 1883.	F.	41	230-260	No valvular heart disease.
94.	Tunker.	do. 1877.	M.	22	208	Valvular heart disease.