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Contributors

Bolton, Charles.
Tweedy, John, 1849-1924
Royal College of Surgeons of England

Publication/Creation

Edinburgh : Young J. Pentland, 1902.

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THE HEART IN DIPHTHERIA.

By CHARLES BOLTON, B.Sc., M.D.

Reprinted from the Edinburgh Medical Journal. *Edinburgh and London*, Young J. Pentland, *April*, 1902.

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By Charles Bolton, B.Sc., M.D., *Resident Medical Officer, University College Hospital.*

Cardiac failure is one of the most important as well as the commonest of the toxic effects of diphtheria. A large majority of patients have at some time or other, during the course of the disease, signs of heart failure, and death during the acute toxic stage invariably results from primary cardiac failure, excluding, of course, an accidental cause, as asphyxia or lung disease owing to involvement of the larynx. Since the introduction of antitoxin into the treatment of diphtheria, it is much less common than formerly to see death resulting from implication of the larynx, a larger proportion of these cases being saved than in the pre-antitoxin days; and it may be stated that now the majority of deaths from diphtheria are the result of heart failure, for it cannot be doubted that many of the cases which now show signs of cardiac failure would have died had they been treated without antitoxin.

Although in diphtheria the acute toxic stage is not sharply separated from the convalescent stage by a fall of temperature or marked alleviation of the constitutional symptoms, the one blending insensibly with the other, it is advisable to speak of cardiac failure as occurring in the acute and convalescent stages respectively, because in these two stages it presents differences which will be referred to later on. In the milder cases, as soon as the fauces are free from membrane, the acute stage may be said to have ended, and the patient to be convalescent; in the more severe cases the membrane takes longer to separate, and even though the throat may have been clean for several days, the patient may still die from the acute toxæmia. The general statement may be made, that in acute diphtheritic toxæmia, when uncomplicated, death usually occurs within the first fortnight of the disease, life being prolonged a day or two over this period in a few cases; hence the patient can never be said to be safely over the acute stage of diphtheria until after the first three weeks.

Cardiac failure in diphtheria either leads to death by syncope, which may be quite sudden but is generally preceded by a progressive failure of the heart, or is rendered manifest by some disturbance in the regularity and alteration in the frequency of the pulse, with or without the physical signs of cardiac dilatation. Œdema and other symptoms of heart failure have been described, but these, if they ever occur, must be extremely rare.

Heart Failure leading to Death.—*Death from acute diphtheritic toxæmia.*—As stated above, death resulting from the severity of the toxic poisoning is due to a primary failure of the heart.

The class of cases which die in this way comprises essentially the severe faucial cases, in which the nose is usually also involved, and sometimes the larynx. Primary laryngeal cases rarely show heart failure, whereas wound diphtheria is very fatal through cardiac failure. Cases of severe toxæmia in which there are subcutaneous hæmorrhages, with or without bleeding from the mucous membranes, are apparently always fatal during the acute stage, from progressive heart failure. On looking up the records of the cases which have died in this manner in University College Hospital during the past eight years, I find that the average time of death was the tenth day of the disease, the earliest being the third or fourth, and the latest the fifteenth day; and it may be stated generally that, except in a few cases, death usually takes place some time during the first fortnight of the disease.

Heart failure in the majority of cases sets in a few days before death; and although death may occur on the same day as the first signs are noticed, yet life may be prolonged for a week or even longer after the symptoms of heart failure have appeared, and all hope of saving the patient has been abandoned. The time of onset of the cardiac failure in these cases is some time during the first week of the disease, especially about the sixth day.

The first sign is noticed in the pulse, which becomes irregular in force and rhythm, the compressibility being always increased. It is as a rule rapid, but at times it is slow. Sometimes before death it becomes remarkably slow, and this slowing is a very fatal sign. The pulse becomes progressively weaker and more irregular till it is imperceptible, and in this condition the patient may linger for hours or even days, to finally die from syncope. Whilst the pulse is irregular the patient may die quite suddenly if any extra strain is thrown upon the heart, as for instance vomiting or struggling. The cardiac impulse may be diffuse and slapping, but this is not always seen. Occasionally the patient may experience severe præcordial pain, which is by no means common. The face is remarkably pale and waxy looking, the lips are cyanosed, and, as the pulse fails, the extremities become cold and the patient restless; the respirations sometimes have a peculiar sighing character. Consciousness is retained to the end, the patient in some cases looking quite bright, so that the friends are hopeful and will not believe the prophecy of death. Vomiting may or may not occur, and, when it does, it usually commences after the irregularity of the pulse has indicated the onset of heart failure. The urine generally contains albumin, and in a few cases it may be entirely suppressed for a day or more before death. The temperature in the worst cases is often low.

As an example I will quote the following case:—

Case 1.—V. P., female, æt. 5; admitted to University College Hospital, 17th March 1901. The illness commenced on 11th March with headache, a sore throat being first noticed on 15th March. On

admission, the tonsils, uvula, and part of the soft palate were covered with blackened membrane; the breath was foul, and the cervical glands were enlarged on each side; there was a little rhinorrhœa. The face was very pale, the lips were a little cyanosed, and there were several subcutaneous hæmorrhages on the trunk and limbs; the patient was prostrate but perfectly conscious. The pulse was very irregular in force and rhythm, 128 in frequency, and at times almost imperceptible. The cardiac impulse was a little diffuse, the sounds being feeble and corresponding with the pulse; no murmurs were present. The lungs were normal, and there were no symptoms of laryngeal involvement. The urine contained a thick cloud of albumin. The temperature was 103°. Antitoxin, 9000 units administered on admission.

Progress.—The pulse became progressively weaker, and at noon on the following day was quite imperceptible; the extremities were cold; the patient became restless, and died at 4.30 P.M. (eighth day of disease). There was never any vomiting, and 7 oz. of urine were passed, between admission and death. On the last occasion that the pulse could be counted it was 116 in frequency.

In very many cases the signs of toxæmia are not so much in evidence. The throat may have become quite clean and the patient appear to be perfectly well, when the pulse will become irregular and the heart progressively fail, death occurring in the manner described above. It is the opinion of the writer that these are cases of a milder degree of toxæmia, many of which would probably have recovered had antitoxin been given at an earlier period of the disease.

The next case is very instructive, as affording an example of the effect of vomiting upon the heart. On the eleventh day of the disease vomiting began, and the heart then showed marked signs of dilatation; on the thirteenth day an attack of vomiting determined death by sudden syncope.

CASE 2.—L. F., female, æt. 8½; admitted to University College Hospital, 12th September 1901. The onset of the disease was 7th September; and on admission the tonsils, uvula, and faucial pillars were covered with thick discoloured membrane; there was profuse rhinorrhœa with excoriation of the nostrils, and the cervical glands were enlarged on each side. A subcutaneous hæmorrhage was present on the right leg. The pulse was weak but quite regular, and the cardiac impulse was in the fifth left intercostal space internal to the nipple. Temperature 101°·6. Antitoxin, 8000 units administered.

September 13.—Pulse 128 in frequency, and a trifle irregular in force and rhythm. Bleeding from the vagina, epistaxis, and a subcutaneous hæmorrhage on the left shoulder.

September 14.—Pulse 144, irregular. Cardiac impulse extends to nipple line. 12 oz. of urine, mixed with blood, passed since admission.

September 15.—Pulse 108, much weaker, but at present more regular. Two fresh subcutaneous hæmorrhages.

September 17.—Vomiting commenced, and now the cardiac impulse was ½ in. outside the nipple line; no murmurs. Pulse 120, irregular.

Fauces almost clean, a small patch of membrane still adhering to the right tonsil. A fresh hæmorrhage. Vaginal bleeding.

September 18.—The pulse is much weaker and more irregular. On the whole the rate is slower, varying from 78 to 104; and on examining the pulse every hour, its irregularity is found to vary considerably during the day, sometimes being almost regular, and soon after becoming extremely irregular. Cardiac impulse still half an inch outside nipple line. The extremities are cold and the face is very pale, but the child is quite bright and conscious. Vomited twice.

September 19.—This morning patient asked for a little jelly, and shortly afterwards vomited considerably, when the pulse suddenly became imperceptible, the face livid, and the pupils dilated; the heart could not be heard, and after a few gasps at intervals the patient was dead (thirteenth day of disease).

The next case illustrates a slowing of the pulse before death.

Case 3.—L. D., female, æt. 3; admitted to University College Hospital on 27th June 1901. The onset of the disease was 24th June, and on admission there was membrane covering both tonsils and faucial pillars; a little rhinorrhœa, and enlargement of the cervical glands on both sides; several subcutaneous hæmorrhages. Antitoxin, 6000 units administered, and repeated on the two following days.

June 28.—Pulse irregular, and especially so after syringing the throat.

June 29.—Pulse 108 to 130, weaker and more irregular. Hæmorrhage from the bowel.

July 1.—At 11 P.M. last night the pulse was found to be 60. At 3 A.M. this morning it was 62, and at 7 A.M. it was 58; after this the patient was pulseless, and died quietly during the afternoon (eighth day of disease).

Cardiac thrombosis.—Cardiac thrombosis is a rare cause of death in diphtheria. It was formerly thought that sudden death in this malady was due to this cause, but the fallacy of this view is now apparent.

Three cases of cardiac thrombosis are recorded by Dr. Woollacott;[1] the first of which I had the good fortune to witness, whilst a medical officer at the Homerton Fever Hospital. They were all severe cases, one being complicated by scarlet fever and suppurative adenitis, and the other two by faucial ulceration, so that a secondary infection was possible in each case. Two died within the first three weeks of the disease, and the third on the twenty-sixth day. From these cases it appears that the chief symptoms of this condition are severe præcordial pain, with some variety of altered respiration and restlessness, the onset of the pain being more or less sudden. Sudden death did not occur, the heart failure being gradual, and the patients surviving for several hours after the onset of the pain. The pulse was rapid but regular before the heart began to fail, and, when the heart dilated, the pulse became

[1] *Lancet*, London, 1899, vol. i. p. 1217.

irregular. There was vomiting in each case preceding the above symptoms. Paralysis was present in two of the cases.

In two of the cases infarcts were present; in one, in the kidney and spleen, the thrombus being situated at the apex of the left ventricle; and, in the other, the infarcts were in the lung, a thrombus being found at the apex of each ventricle and in the right auricle. In the case without infarction the clot was situated in the right ventricle at the apex, and in the right auricle. The clots were breaking down, and had evidently been there long before the onset of the pain, which Dr. Woollacott attributes to dilatation and distension of the heart.

Embolism of the popliteal artery, with gangrene of the limb, and hemiplegia due to cerebral embolism, have been recorded in diphtheria.

It is quite conceivable, as Dr. Woollacott points out, that sudden death might easily arise; but such was not the course of events in these cases.

Death during convalescence.—After the acute stage of the disease has passed, death from heart failure is usually associated with paralysis, and is especially seen in cases of generalised paralysis affecting the palate, pharynx, larynx, and diaphragm. It is by no means the invariable rule for patients extensively paralysed to die from heart failure, as many die from lung complications; and, when this does happen, the heart failure is frequently found to be secondary to some strain, as for instance vomiting or the shock caused by severe pain, as in a case of which I have knowledge in which very severe pain in the loin, with hæmaturia due to gravel in the kidney, undoubtedly accelerated the death of the patient; or it may be secondary to paralysis of the respiratory muscles, and probably this is the commoner mode of death in paralysis. In other cases the heart failure is only a part of the generally enfeebled condition of the patient, and presents no peculiarities. This is especially seen in those cases of marked wasting in which food cannot be retained by stomach or rectum. The heart failure in these cases of paralysis is evidenced by irregularity of the pulse, as in the acute stage, but there is no progressive enfeeblement of the circulation and death within a definite period of time as in the acute toxic cases. At this late period of the disease it is by no means so uniformly fatal, as a patient may be rendered pulseless by vomiting, and yet recover.

Sudden death.—Sudden death from syncope may occur at any stage of diphtheria, but the most alarming cases are those which occur when convalescence is established and the patient is thought to be quite free from danger. These sudden deaths are all referable to some strain which has been thrown upon a heart which is unprepared to meet it.

As will be shown later, primary heart failure, unassociated with paralysis, occurs at an early period of the disease, and is manifested

in the milder cases chiefly by irregularity of the pulse; in some cases this irregularity is very persistent, and may last for many weeks, the patient all the while appearing to be and feeling perfectly well, and therefore all the more likely to encounter strain. Strain will determine the onset of cardiac dilatation and irregularity of the pulse in a patient who has previously shown no signs at all, and it is therefore not surprising that sudden death should occasionally occur in patients who have up to then appeared in perfect health. It is probable, if the heart and pulse were carefully examined in many of these cases, that some signs would almost invariably be found.

Seven cases of this nature are described by Dr. Lees,[1] of which I will quote three:—

"A child of 3 years of age had suffered from diphtheria, but was thought to be convalescent. She was standing by a window, when some one entered the room a little abruptly. She turned quickly round, and fell dead on the floor."

"A medical man in good practice attended a child for diphtheria. As she did not recover as quickly as he expected, he sent her to the seaside for change of air. She fell dead on the sands."

"A girl of nearly 11 years old had a severe attack of diphtheria. She looked pale after this, and was kept in bed for eight weeks. She was then allowed to rise. In five minutes after rising she was dead."

Heart Failure not leading to Death.—A very large proportion of patients suffer from cardiac failure at some stage of the disease, and this is evidenced by irregularity or intermittence of the pulse, with or without the physical signs of dilatation of the heart. It is rare to have pain or other symptoms, the patient in most cases feeling quite well, and in many cases being quite lively, whilst the pulse is irregular. Primary heart failure occurs most commonly during the acute toxic stage of the malady, and at this period it may be associated with an early onset of paralysis.

Cardiac failure in the acute stage.—The first signs of heart failure are commonly noticed between the end of the first week and sometimes during the third week of the disease; a large number of cases commencing at the end of the second week. In some cases they may be seen as early as the fourth day.

The pulse is on the whole rapid and compressible, and becomes more or less irregular; it remains irregular for about two to six weeks, but the irregularity may last for four to five months, or, on the other hand, for only a little over a week. The heart may show signs of dilatation.

In the following case the pulse became irregular on the fifth day, and was quite normal again on the fourteenth day, the heart showing no signs of dilatation.

Case 4.—E. H. B., male, æt. 5; admitted to University College

[1] *Brit. Med. Journ.*, London, January 5, 1901, p. 7.

Hospital on 14th November 1901. The onset of the disease was 11th November, with vomiting and feverishness. On admission, there was a patch of white membrane on each tonsil, on the left side spreading to the adjoining part of the pharynx, and a little on the uvula. There was no rhinorrhœa. The cervical glands were a little enlarged on each side. Temperature 100°·4. The pulse was quite regular and the heart normal. Antitoxin, 6000 units administered.

November 15.—The pulse has become irregular; no signs in the heart; a cloud of albumin in the urine.

November 19.—Fauces quite clean; pulse still irregular.

November 24.—Pulse again quite regular; knee-jerks present; no signs of paresis.

December 27.—Discharged quite well; no other symptoms having developed.

During the period of irregularity the pulse was examined every hour, and varied in frequency from 70 to 120 during the twenty-four hours, being quite regular at times. On the whole the irregularity was associated with a slowing of the pulse, but this was by no means an absolute rule. Towards the end of the time the pulse became irregular about once a day only.

Some of the readings for November 17 are as follows:—

Time.	Frequency.	Regularity.
3 A.M.	80	Regular.
6 ,,	94	Somewhat irregular.
11 ,,	84	Slightly ,,
7 P.M.	...	,, ,,
8 ,,	78	,, ,,
9 ,,	120	Regular.
9.30 ,,	74	Irregular.
10.30 ,,	80	Much less irregular.

When the heart shows the physical signs of dilatation, the cardiac impulse is displaced outwards towards the left anterior axillary line, and is more or less diffuse and slapping in character. The percussion note extends a little further to the left than normal. On auscultation the first sound is short and sharp, and sometimes a systolic murmur may be heard at the apex, the pulmonary second sound being accentuated. The point on which most stress should be laid is the diffuse character of the cardiac impulse and its displacement outwards, especially when it has previously been found in the normal situation.

In the following case the pulse became irregular on the ninth day of the illness, and was quite normal on and after the forty-eighth day. The heart showed signs of dilatation on the ninth day, and was normal again on the thirtieth day. After the fauces were clean the child was quite well and lively, and it was only on examining the heart and pulse that anything could be found amiss.

CASE 5.—S. B., female, æt. 5½. Admitted to University College Hospital on 4th November 1901. The onset of the disease was 31st October. On admission there was a patch of white membrane on each tonsil, the uvula and soft palate being quite clean. There was no rhinorrhœa, and the cervical glands were enlarged on each side. Pulse 100, and quite regular. Cardiac impulse in the fifth left interspace internal to the nipple. Temperature, 100. No albumin in the urine. Antitoxin, 8000 units administered.

November 8.—Fauces quite clean. The pulse became irregular at 4 A.M., and on examining the heart at 11 A.M. the impulse was in the anterior axillary line; there were no murmurs; the first sound was practically normal, and the pulmonary second sound was accentuated. Knee-jerks present; no signs of paresis.

November 13.—Cardiac impulse just external to the nipple line; pulse irregular.

November 15.—A systolic murmur heard at the apex; impulse in the nipple line.

November 18.—Impulse again outside nipple.

November 20.—Impulse in nipple line.

December 3.—Up to this date the pulse had been more or less irregular all day, but after this it became quite regular during the whole day, and was only irregular at night; this irregularity gradually disappeared by the 18th December, when the patient was discharged quite well. The knee-jerks were present during the whole of the time, and there were never any signs of paresis; there was never any albumin in the urine. During the period of irregularity the pulse was examined frequently during the day and night, and was found to vary very much both with regard to frequency and irregularity. On the whole, the irregularity was more marked with a slow than a rapid pulse, although sometimes it was quite as evident in the latter case; and on the whole the pulse was both slower and more irregular during the night than during the day.

The following figures represent the record from 5 A.M. on 13th November to 8 A.M. on 14th November :—

Time.	Frequency.	Regularity.
November 13—		
5 A.M.	71	Very irregular.
7 ,,	120	Regular.
8 ,,	90	Irregular.
9 ,,	...	Slightly irregular.
11 ,,	86	,, ,,
3 P.M.	96	,, ,,
5 ,,	108	Regular.
9 ,,	82	Irregular.
10 ,,	79	Slightly irregular.
November 14—		
2.30 A.M.	66	Very irregular.
4 ,,	88	Irregular.
6 ,,	104	Regular.
7.15 ,,	96	Slightly irregular.
8 ,,	89	Irregular.

When paralysis comes on in the acute stage of the disease, it is usually preceded by some irregularity of the pulse.

In the following case the pulse became irregular on the sixth day of the disease, and on the thirteenth day paralysis commenced. The pulse was still irregular on the hundred and twenty-fourth day when the patient was discharged, and the knee-jerks were also absent; the paralysis disappeared on the seventy-fourth day. The heart showed signs of dilatation about the same time as the pulse became irregular. The irregularity of the pulse was not preceded by vomiting, which sometimes happens in these cases.

Case 6.—A. G., female, æt. 3 years and 7 months. Admitted to University College Hospital on 29th August 1901. The onset of the disease was 27th August. On admission there was a large gelatinous-looking piece of membrane on the right tonsil; profuse rhinorrhœa with enlargement of the cervical glands on both sides. Pulse 103, and quite regular; heart normal. Temperature, 103. Urine free from albumin. Antitoxin, 8000 units administered.

August 31.—Membrane has extended to the uvula and left tonsil; pulse quite regular. Antitoxin, 8000 units administered.

September 1.—Pulse irregular in force and rhythm, and compressible; cardiac impulse in fourth left intercostal space in nipple line.

September 2.—Impulse outside nipple line, and an apical systolic murmur present; knee-jerks present.

September 8.—Fauces clean, a little superficial erosion of the mucous membrane being present at the base of the uvula. Nasal voice, but palate moves a little on phonation; pulse more irregular.

September 9.—Palate quite motionless; pulse is less irregular since syringing of the throat has been stopped.

September 17.—Patient is very languid and limp. Cardiac impulse $\frac{3}{4}$ in. outside nipple line; systolic murmur still present.

September 24.—An occasional strabismus is noticed to-day.

September 27.—Marked internal strabismus; pupils dilated.

October 2.—Knee-jerks absent for first time. Cardiac impulse in the nipple line. Breathing during the night was very irregular, and frequently interrupted by pauses. Diaphragm acts quite normally.

October 13.—The breathing has frequently been irregular during the night. Squint disappearing; pulse still irregular.

October 18.—Cardiac impulse outside nipple line.

November 8.—Squint practically gone; voice normal and palate moves well. Knee-jerks absent; child much more lively and stronger. Cardiac impulse in nipple line.

December 16.—Child quite well; knee-jerks, heart, and pulse as before.

December 28.—Patient contracted scarlet fever and was sent to the fever hospital, the knee-jerks still being absent and the pulse irregular. The pulse in this case varied in the same manner as in the two previous cases. It rapidly changed from a condition of regularity to irregularity several times during the day, and the frequency also varied considerably; the irregularity in this case occurred more often with a rapid pulse than in the two previous cases. Towards the end of the time the pulse

was irregular only during the night. When the child started vomiting at the onset of the scarlet fever, the pulse was very irregular during the whole morning, and after the vomiting ceased it regained its former condition.

On 26th November some of the pulse records were as follows:—

Time.	Frequency.	Regularity.
November 26—		
7 A.M. . . .	100	Regular.
8 ,, ...	97	Slightly irregular.
8.45 ,, ...	...	Regular.
11 ,, ...	100	Slightly irregular.
3 P.M. . . .	76	Very ,,
9 ,, ...	96	Slightly ,,
November 27—		
7 A.M.	96	Regular.

I have examined the pulses of very many other cases, of which the above are examples, in the same way, and have found them to agree in the following facts:—

1. During the twenty-four hours the pulse alters considerably in both degree of irregularity and in frequency.

2. These changes occur frequently, and often at very short intervals (half an hour or so); sometimes the change can be noticed under actual observation, and it often appears as if the irregularity comes on in definite attacks.

3. On the whole the irregularity is most marked at night, and usually persists during the night after the pulse has become quite regular during the day.

4. As a rule, when the pulse becomes irregular, there is a diminution in its frequency, but this rule is by no means absolute. Sometimes the pulse may be irregular when the frequency is increased or diminished, and in other cases the pulse is rapid all the time.

5. The irregularity coming on in the acute stage is a primary affection, but is nevertheless greatly affected by strain, as vomiting and struggling when the throat is syringed or food administered by the nose.

6. The irregularity does not conform to any type; every variety and degree of irregularity, with sometimes intermittence, occur together.

7. As a rule the patient appears quite well, and suffers no inconvenience or distress on account of the heart failure.

In the following case there were undoubtedly attacks of heart failure. On the eleventh day of the disease vomiting commenced, and the pulse became irregular; paralysis started on the sixteenth

day. The attacks were certainly not in any way connected with the entrance of saliva or food into the larynx, as there was no coughing or other symptom of respiratory disturbance.

Case 7.—A. P., male, æt. 2; was admitted to University College Hospital on 17th March 1901. Onset of the disease was 16th March, and on admission there was membrane on both tonsils and uvula, with enlargement of the cervical glands on each side. The voice was a little hoarse, and there was a trace of recession of the lower portion of the chest. Pulse quite regular and heart normal. Temperature 99°. The urine contained half albumin on boiling and setting aside. Antitoxin, 6000 units administered.

March 21.—Fauces quite clean.

March 26.—Occasional vomiting; pulse irregular.

March 31.—Several times during the day the child has become cyanosed, the pulse being barely perceptible. Palate does not move very freely. No change in the character of the respirations. Cardiac impulse in the fifth interspace outside the nipple line.

April 13.—Internal strabismus. The attacks of cyanosis and pallor of the face with a feeble pulse continue.

April 17.—Knee-jerks absent. Diaphragm normal. No signs of paresis anywhere except the eyes and palate.

April 22.—Child is generally limp. Vomited twice during the day. Has not had a bad cyanotic attack for ten days.

April 26.—Vomiting stopped, and pulse perfectly regular; still very limp, but paralysis is clearing off. After this, recovery was gradually completed.

Apart from irregularity, the frequency of the pulse may be affected alone. As a rule, in acute diphtheria the pulse is as a whole rapid, but exceptionally it may be slow.

In the following case the pulse was rapid till the eighth day of the disease, after which for a fortnight the highest record was about 72 in the morning, and later in the day it varied between 48 and 64; the frequency rose after this, but until the date of discharge it sometimes fell to 60 at night.

Case 8.—N. H., female, æt. 10; was admitted to University College Hospital on 2nd September 1901.—The onset of the disease was 31st August, and on admission the right tonsil was inflamed and the left covered with thick membrane, at the upper part creeping on the soft palate. The cervical glands were enlarged on each side, and there was slight rhinorrhœa. The cardiac impulse was in the fifth left intercostal space internal to the nipple line, and the sounds were normal. The pulse was 124 and regular. Temperature, 100°·4. No albumin in the urine. Antitoxin, 8000 units administered.

September 6.—Fauces clean.

September 8.—Slight nasal voice; pulse slow during the night.

September 10.—Nasal voice distinct; palate not drawn up on the left side on phonation; knee-jerks present; pulse regular but slow during the night.

October 12.—No other signs of paralysis have developed, and for the past few days the voice has been normal; knee-jerks have never been lost. The frequency of the pulse has risen, and for the past two days it has not been below 76, the highest record being 92. The patient made an uninterrupted recovery.

Cardiac failure during convalescence.—Heart failure, as evidenced by an irregular pulse, with or without signs of dilatation, is as a rule associated with paralysis during this period of the disease; or it may be determined by some strain which is thrown upon the heart, as for instance getting up too early, undue excitement, or vomiting. It may come on as late as six or eight weeks after the onset of the disease. Tachycardia is sometimes seen during convalescence; slowing of the pulse is less common.

Irregularity of the pulse may occur after the paralysis is established; or the paralysis may be preceded by an irregular pulse, which is often determined by vomiting.

The following case of paralysis preceded by an irregular pulse will be quoted, as it is unique in that the paralysis was limited to the external ocular muscles:—

Case 9.—I. S., female, æt. 4; was admitted to University College Hospital on 15th September 1901. The onset of the disease was 10th September. On admission there was yellowish grey membrane on each tonsil; the cervical glands on each side were enlarged; there was hoarseness of the voice, with stridor and retraction of the lower part of the chest. The pulse was regular, and the cardiac impulse internal to the nipple, the heart sounds being normal. Temperature, 99°·4. A cloud of albumin in the urine. An asphyxial attack occurred, and tracheotomy was performed. Antitoxin, 8000 units administered. After this the child coughed up membrane through the tube, and on 24th September there were present the physical signs of pneumonia. The tracheotomy tube was removed the next day, and on 4th October the signs of pneumonia had disappeared; the pulse was quite regular, the knee-jerks present, and the child lively and well. On 12th October the pulse became irregular, and on 19th October there was marked internal strabismus, the palate was normal, and there were no other signs of paresis; accommodation was normal, as the child could shove a pin through a tiny hole in a piece of paper at every attempt quite easily and without hesitation; the knee-jerks were present, and there were no signs of cardiac dilatation. The squint had practically disappeared on 29th October, and on 6th November the pulse was quite regular. There were never any other signs of paralysis, and the knee-jerks were never lost. The child was discharged, and on 29th November was brought up to the hospital for inspection; she was then perfectly well.

In this case the pulse was very frequently examined, and was on the whole more uniformly rapid than in those previously quoted, and no definite relation of irregularity to any particular degree of frequency could be detected; it changed considerably during the

twenty-four hours, and finally became quite regular by day, whilst still irregular at night.

As an example of the effect of strain, in determining the onset of heart failure, the following case will be quoted; it is also interesting in that the patient finally developed marked ataxy of the legs.

Case 10.—S. W., female, æt. 12; was admitted to University College Hospital on 9th October 1900. The onset of the disease was 6th October, and on admission both tonsils, uvula, and part of the soft palate were covered with membrane. The pulse was quite regular, and the heart and urine normal. Temperature, 99°·8. Antitoxin, 8000 units administered. On 16th October the fauces were clean, the pulse quite regular, and the heart normal. On 22nd October the patient was allowed to get up in blankets, sitting in a chair; four days later the pulse was found irregular, and between 90 and 100 in frequency; the cardiac impulse was displaced ½ in. outside the nipple line, and slapping in character; a systolic murmur was present at the apex, and the pulmonary second sound was markedly accentuated; the patient complained of pain and weakness in the legs; knee-jerks present. She was put to bed. A nasal voice developed a fortnight later. On 8th December the pulse was between 72 and 90, and quite regular, the murmur had disappeared, and the cardiac impulse was in the nipple line; the knee-jerks were now absent. On 18th December the patient got up again and could walk, but there was marked ataxy of the legs; no other signs of nervous affection were present. On 6th January 1901 she could walk without ataxy, and the heart and pulse were quite normal.

After a consideration of the various forms of heart failure occurring during the different stages of diphtheria, as exemplified by the cases whose records have been described and discussed above, the very great danger to which patients during the whole course of the disease are liable, as a result of acute degeneration of the neuro-muscular mechanism of the heart, is at once apparent. This danger can only be rendered evident by a thorough and systematic examination of the heart and pulse in every case of diphtheria, however mild it may appear to be; and it can be best guarded against by keeping the patient in bed, or at least perfectly free from all excitement and strain, as long as there are any signs of heart failure, as shown by an irregular pulse or the physical signs of cardiac dilatation.

The time at which a primary heart failure is to be feared is during the acute stage of the disease, and the only prevention of this consists in the use of efficient doses of antitoxin at as early a period as possible. After the acute stage has passed, and convalescence has set in, the heart failure is generally secondary, and must be dealt with as mentioned above.

In conclusion, I wish to express my thanks to the physicians of University College Hospital for so kindly allowing me to make use of these cases.