Sudden death / by Herbert French.

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SUDDEN DEATH.

BY

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Sudden Death.

Gentlemen,—I thought that to-day we would at any rate begin to discuss the subject of sudden death; and by sudden death in this connection I do not mean to include railway accidents, suicide, murder by means of firearms or prussic acid, and so forth; but rather do I want to deal with such medical and surgical conditions as may terminate suddenly in death. As instances of what I mean I would mention an apoplectic seizure due to cerebral hæmorrhage, or a sudden hæmoptysis due to rupture of an aortic aneurysm into a bronchus.

Before entering upon the discussion of those gross lesions which may end in sudden death, however, I must draw your attention to a group of cases which is intermediate between those of accident, suicide, or murder on the one hand, and those of organic disease on the other, namely, death from such things as fright, for example, or the so-called "status lymphaticus." There is no doubt that fright may kill quite healthy individuals. There have recently been in this hospital two cases of adult persons who were about to undergo surgical operations, and who died of fright. In one case the anæsthetic had just been begun, and in the other even that stage had not been reached. The patient was being got ready for the operation, and simply died. In each case there had been great fearfulness for the result of the operation, and the dread had so worked upon the nervous system that the latter simply ceased to carry on the

action of the lungs and heart. Such cases as these may be rare, but they are very distressing. Therefore I would ask you to be very considerate of the feelings of patients who are to undergo operations, particularly women — both these patients were women. A post-mortem examination in these cases reveals nothing beyond the morbid condition for the relief of which the operation had been advised, perhaps a fibroid of the uterus, or a carcinoma of the breast, and so forth.

Closely akin to the cases I have just been referring to are others in which I do not think fright plays a very obvious part, and yet, for some apparently inadequate cause, the patient dies. If I use the term "status lymphaticus" I think I shall bring to your minds the kind of case I mean. It occurs mostly in young people, children under the age of puberty, in whom it may be that an anæsthetic has been given for the performance of some minor operation such as circumcision; or perhaps an operation has been performed on the tonsils without any anæsthetic at all, and the patient simply dies. Neither of these operations is an adequate cause of death. Post-mortem, these patients are found to have rather large thymus glands, and swollen lymphatic glands throughout the body—inguinal, bronchial, mesenteric, and so forth. You know how, at the root of the mesentery, that part furthest from the intestine, there are always visible lymphatic glands. As you go from the root of the mesentery out to where it is attached to the intestine, the glands become smaller and smaller, and they ought to be invisible in an ordinary sense when you get close to the intestine itself. But in the people who have died of the so-called "status lymphaticus," you

will find even the smallest lymphatic glands close up against the intestinal wall quite visible, sometimes as big as small beans. Moreover, the tonsils, the Peyer's patches, the solitary follicles of the intestine, and the vermiform appendix, in fact, all the lymphatic structures in the body, may be enlarged. That is all we can find wrong in these cases. You will note that I speak of the status lymphaticus as "so-called." This is because I am not positively certain that the condition is pathological. I was speaking to you about this last week when we were making a post-mortem examination upon a girl of eight, who had been run over by a motor car and killed instantly. She presented all the features of a case of so-called "status lymphaticus," particularly the large thymus gland, and the large visceral lymphatic glands. It is not often we make post-mortem examinations on children who were apparently well a few minutes before death; and I would suggest that it is possibly normal for absolutely healthy children to have relatively large thymus glands, large lymphatic glands in the mesentery, and large Peyer's patches and solitary glands in the bowel. It may be that the serious illness that precedes the death of most of the children that we make post-mortem examinations upon, causes the lympathic tissues to shrink in such a way that we have come to regard the absence of the "status lymphaticus" as normal, whereas the exact contrary may be true. Upon the latter supposition, the sudden deaths of children from apparently inadequate causes might well be included amongst those of adults, due to fright, or to fearfulness of a prospective anæsthetic and operation. In any case we learn that we must beware of telling

anybody in connection with an operation that there is no risk in it whatever.

Having cleared away the question of suicidal, homicidal, railway accident, and poisoning deaths, on the one hand, and such intermediate conditions as fright, and the status lymphaticus, on the other, I want to pass on to consider what you, no doubt, thought was the chief subject I should talk about to-day—obvious lesions in the bodies of people who may have died suddenly. Theoretically, you can divide these into two groups, namely: first, those cases in which no one had any knowledge whatever of the presence of any disease, the person being apparently in perfect health immediately before death; and secondly, cases in which there was a diagnosed disease, whose existence had possibly been known of for years, yet in which, in the end, death may have occurred quite suddenly. A man may have an aldermanic dinner, and fall forwards dead in the course of making a speech after it; he was apparently a healthy man in whom, nevertheless, there was a latent disease which ended in apoplexy. On the other hand, a person may be suffering from aneurysm of the aorta which has been giving rise to symptoms for a long time, and one day it may rupture and cause sudden death. The distinction between these two types of cases is not merely theoretical; it is of practical importance, because, in the one class of case, the patient may be under treatment by a doctor, and the doctor may know what to expect, whilst in the other, no medical man may have seen the case during life, and an inquest may be necessary, as we were saying in the Forensic Medicine lecture this morning. Nevertheless, I do not propose to maintain the distinction

ere. I propose to take both classes of cases ogether, classifying them merely upon a patho- gical basis.

I think we can divide the causes of sudden eath into the following main groups :—

THE MAIN CAUSES OF SUDDEN DEATH

exclusive of murder, suicide, accident, fright, and status lymphaticus).

.. CAUSES IN CONNECTION WITH THE HEART OR WITH THE AORTA.

(*a*) *Muscular.*
- Fatty heart.
- Fibroid heart.
- Rupture of the heart.

(*b*) *Valvular.*
- Aortic regurgitation.
- Infective endocarditis.

(*c*) *Pericardial.*
- Acute pericarditis.
- Hæmopericardium.
- Adherent pericardium.

(*d*) *Coronary Artery.*
- Embolism.

(*e*) *Aorta.*
- Rupture of aortic aneurysm :—
  - Into pericardium.
  - Into trachea.
  - Into bronchus.
  - Into lung.
  - Into œsophagus.
  - Into pleura.
  - Into mediastinum.
  - Into abdomen.
  - Externally.
  - Into the pulmonary artery.
  - Into superior vena cava.
  - Into an auricle.

II. Causes in connection with the Brain.

Ordinary cerebral hæmorrhage.
Pontine hæmorrhage.
Cerebral embolism.
Cerebral embolism from infective endocarditis, followed by aneurysm on circle of Willis and secondary hæmorrhage.
Hæmorrhage into a tumour.

III. Lung Conditions.

Hæmoptysis from small pulmonary aneurysm in phthisical cavity.
Pulmonary embolism.
Very large pleuritic effusion.

IV. Laryngeal conditions.

Diphtheria + spasm.
Rupture of a quinsy.
Foreign body.
Acute suffocative œdema.
Syphilitic abductor paralysis.

V. Addison's Disease.

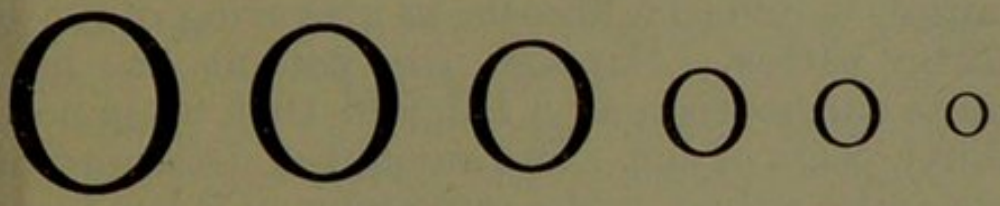
The first main group we will take is that in which there is something the matter with the heart or aorta. I am not going to confine myself rigidly, however, to the order in which I have written the above list. One of the first affections of the heart which will occur to you here is that which is often called myocarditis, but which would be more accurately designated myocardial degeneration. Sometimes this is a fibrous degeneration of the myocardium, sometimes it is rather a fatty infiltration or a fatty degeneration. Perhaps fatty hearts are more liable to cause sudden death than are fibroid hearts. The heart change is demonstrated by making microscopical examinations of the muscles. I would have you remember that

a fatty heart may cause sudden death soon after labour. It is a very distressing occurrence, for your patient is a woman who has passed through her labour normally, and all seems to be going well when she suddenly dies, and everyone begins to ask why this has happened. Nobody was prepared for the catastrophe, very likely not even the medical man. I want you to realise that this disaster can occur as a result of a person having a fatty heart. In the volume of the Guy's Hospital Reports for the year 1873 there is a paper headed "Sudden death from syncope soon after labour," and I will read you an illustrative case. In November, 1870, a patient, aged 34, was attended in her ninth confinement in the Lying-in Charity of Guy's Hospital. Labour was tedious, the uterus acting feebly. There was a little oozing of blood before delivery, but after the child was born this soon stopped. As the woman appeared faint, one of the obstetric residents was sent for, and he gave her brandy with a little opium. The gentleman who attended her remained for a time, and then, as all seemed to be going well, he left. He was again sent for shortly afterwards, and on his arrival he found the patient dead. It was difficult to ascertain the time of death, as there was no struggling. The husband and friends, who had been in the room the whole time, were not aware when death took place; they thought she had fallen into a deep sleep. The writer of the paper I have referred to ascertained from the husband that his wife, previously to her confinement, had suffered from palpitation of the heart and attacks of dyspnœa, and on that account was often obliged to stop whilst walking in the street and sit down on a

doorstep. At the post-mortem examination next day it was found that there was much fat, especially in the abdominal wall; no coagulum in the heart's cavities or vessels; the lungs were healthy, the heart had the usual quantity of fat on its surface, but the muscular tissue was evidently the seat of fatty degeneration. The kidneys and all the other organs were healthy to the naked eye. It is very difficult to make sure of a diagnosis of fatty heart during life, but a catastrophe such as this reminds us of the necessity of treating rather carefully people who complain of attacks of dyspnœa, in consequence of which they are often obliged to stop when walking in the street.

Let us now consider aortic regurgitation. It is the most dangerous of all the individual valvular heart lesions, and it is the most likely to cause sudden death. This leads me to broach a question which you may say is in no way concerned with sudden death. It is so important, however, that I want to speak to you about it. It is this: we do not always sufficiently practise in the wards the determination of the *amount* of a valvular lesion present in the particular case before us. We are too apt, upon hearing the bruit, to label it aortic regurgitation, without making a really good attempt to measure the *degree* of the regurgitation, that is to say, to determine whether the volume of blood that leaks back is a small or a large proportion of that which the ventricle pumps up. In the post-mortem room, a fortnight ago, there were two consecutive cases of people over seventy who had had rheumatic fever when girls, and who had definite mitral stenosis. They had mitral valves which were, roughly speaking, two-thirds the

size they ought to have been. That degree of stenosis does not very seriously hamper the circulation, but a greater contraction would have made itself felt many years earlier. We want words in this connection, which are comparable to paralysis and paresis, to represent different degrees of stenosis. Stenosis should represent an advanced degree, while there should be another term to indicate the lesser degree. If, allowing for the necessary reduction in scale that the exigencies of printing require, the normal aperture of the mitral valve were represented by an "O" half as large again as the one marked "1" in the following diagram :—



1 2 3 4 5 6

then it is clear that the mitral valve, as seen and measured in the post-mortem room, would be stenosed if it were reduced to the size of O No. 1, and that it would also be stenosed if it were reduced to the size of O No. 6.

Cæteris paribus, however, the prognosis in any particular case is bound to be worse with mitral stenosis of degree "six," than it is with mitral stenosis of degree "one," and so on. Similarly, there may be aortic regurgitation in which, for every ounce of blood which is pumped forward, there leaks back a drachm, and with that the circulation goes on very well. On the other hand, there may be a condition in which, out of every eight drachms pumped out

by the left ventricle, seven drachms leak back before the next systole, and that is a very serious thing. You have only to get a very little further and the circulation stops, because almost the whole of what the heart pumps into the aorta leaks back into the heart again. The patient is then bound to die. That is what happens in bad aortic reguritation.

There are two main varieties of aortic disease—the rheumatic and the syphilitic. Which of these has the worst prognosis from the point of view of sudden death? The syphilitic. If you will think a moment you will see why this is. You may have a bad prognosis in rheumatic aortic regurgitation, but a rheumatic valvular lesion, whether aortic or mitral, is due to a fibrosis, to a scarring of those aortic valves. Suppose the patient has had acute rheumatism as a lad, and that he is now thirty years of age. The amount of deformity of his aortic valves is the same now as it has been for years past. The scarring is the result of an inflammation which ceased years ago, supposing always that there has been no recurrence of the rheumatism. The heart will finally fail simply because it cannot maintain its hypertrophy for ever, but the amount of aortic regurgitation will not increase. In a syphilitic case, on the other hand, the changes in the aortic valve are not the fibrotic scarring that results from an inflammation that has ceased; they are, on the contrary, due to atheroma, and this atheroma, having developed, is almost certain to progress. Syphilitic aortic regurgitation, therefore, increases as time goes on, and thus, no matter how much the heart may hypertrophy, there comes a time when the leakage is so great that death cannot but occur. This syphilitic atheroma is a

progressive lesion that sets in between 35 and 45 years of age in males who work hard, and it is apt to advance even in spite of the administration of iodide of potassium and mercury. Syphilitic aortic regurgitation, therefore, is much more likely to cause sudden death than is aortic regurgitation due to a former rheumatic endocarditis, other things being equal. One must remember, however, that upon the top of what was a simple rheumatic endocarditis, followed by a scarring of the valve, there may develop an acute fungating endocarditis, which is accompanied by an ulcerating process that often eats a hole through the valve, and thus produces so sudden an increase in the degree of regurgitation that sudden death results. I need scarcely remind you that there may be a similar rupture of the valve when the disease is due to atheroma, as Dr. Lauriston Shaw has recently told you in his clinical lecture on that subject.

I have now to consider two much rarer conditions, namely, rupture of the heart and rupture of the aorta. They both come under one heading in a way, because they are both most liable to occur in syphilitic patients. Whereas you may know that an aortic regurgitation patient has heart disease, and he may be under your care for years before he suddenly dies, you cannot diagnose, as far as I am aware, the man who is going to have rupture of the heart, because death from this cause usually comes on in an apparently healthy person. It is the kind of case that is medico-legally important, because the disaster is likely to occur in a man who is engaged in some work, such as going up a ladder with a hod of bricks on his shoulder; the weak spot in the heart may rupture and the man may fall down and break his neck, the medico-legal

question then being whether the condition of the heart caused him to die and then fall, or whether he died as the result of a fall. Rupture of the heart, when it occurs, nearly always takes place at one spot, namely, in the region of the apex of the left ventricle. You will find in each case of ruptured heart that the wall is very much thinner at that spot than it ought to be, and you will find generally that it is fibrous there instead of muscular. These three specimens are all from cases of the kind, and they are all precisely similar to one another.

There may or may not be laminated blood clot adherent to the interior of the fibrous patch in the myocardium. The cause of the weak fibrous patch is always one or other of two things: either there is syphilitic endarteritis of the coronary arteries, so that the part near the apex which is supplied by their distal branches does not receive sufficient blood, and consequently undergoes a gradual atrophy from malnutrition; or there has been in the wall at that spot a gumma, which gumma, while it was there, displaced the heart muscle fibres, and when it healed made a fibrous patch, just as a healed gumma will cause a fibrous scar in the liver or in the skin. As the result of some physical effort there suddenly occurs so great an increase in the work the heart is called upon to do that a rupture takes place through this weak fibrous scar at the apex of the left ventricle. Death results almost instantly, because enough blood will get out into the pericardium to fill the latter with blood under considerable pressure; and if you get a bigger pressure outside the heart than there is inside the auricles, the circulation is bound to stop. Acute pericarditis may kill in the same way.

If it gives rise to effusion, that need not necessarily be bad for the patient. But if that effusion takes place so quickly that the pericardial sac cannot bulge enough and make room for the effusion, then the fluid is under such great pressure that it squeezes the auricles, and the circulation ceases. When a heart ruptures, it is not the amount of blood lost which kills the patient, but rather the fact that the blood gets into the pericardium outside the auricles under a pressure that is greater than that within the auricles. I would ask you to look particularly at this specimen, labelled "aneurysm of the heart." The cardiac aneurysm is due to the bulging of the fibrous scar before it bursts. If you look at the specimen carefully you will find that the wall of the left ventricle close to its apex has not only become very much thinner than it should be, but that it has bulged out into a saccular aneurysm. Please note how nature tried to save that heart from bursting. You will see inside it a laminated blood clot adherent to the fibrous patch; this clot, if it could have been given time to organise sufficiently, would have repaired the weak spot. The natural process of thus plastering up, or patching over, the thin place in the ventricular wall shows extremely well in this other specimen also.

The next cause of sudden death we will discuss is rupture of the aorta, or of an aortic aneurysm. Cases of aortic aneurysm may be classified in several different ways, but for our present purpose I should like to divide them into the following three groups, namely: first, those in which, previous to rupture, the existence of aneurysm was entirely unsuspected owing to there being neither symptoms nor physical signs

of it; secondly, those in which the symptoms and signs have led to a correct diagnosis of aneurysm previous to death; and thirdly, those in which, although the aneurysm has given rise to symptoms and signs, the latter have been interpreted as due to something else than aneurysm; in other words, the diagnosis has been wrong. Should the aneurysm rupture, the occurrence is a complete surprise in groups one and three, though it would have been anticipated in group two.

I wish you to realise that aneurysms of the aorta are very variable in size. When they are as big as Tangerine oranges, or larger still, there may be a definite pulsatile tumour which cannot be mistaken; and besides this, the abnormal mass may compress different structures in the neighbourhood, and thus give rise to various pressure symptoms, such as those which result from obstruction to a bronchus or to the œsophagus, or to the trachea, or to interference with and degeneration of nerves such as the left recurrent laryngeal, the left cervical sympathetic, or the brachial plexus, so that, in addition to pain, there may be changes in the voice, inequality of the pupils, or atrophy of some of the muscles of the shoulder and upper arm. It is important to realise, however, that an aortic aneurysm may be so small that you may not know it is there. You may pass a man for life assurance one day and he may die next day from the rupture of an aneurysm that was entirely unsuspected. This is a case of sudden death in an apparently sound person, and therefore a much more striking occurrence than is the sudden ending there may be in a case of aneurysm that has already been diagnosed from symptoms or pressure signs. The commonest

site for a small aneurysm of this kind is in the first part of the aorta immediately above the aortic valves. You will remember that the pericardium extends along the aorta for an inch or so above the level of the aortic valves. You will therefore realise that there is ample room for a bulge the size of a small walnut, or even larger, to develop upon that part of the aorta that lies within the pericardium, as in this specimen, and for it to rupture into the pericardium. The case could not then be diagnosed from rupture of the heart itself, except upon post-mortem examination.

Death occurs abruptly, because the blood effused into the pericardium, outside the auricles, is under such pressure that the latter are unable to go on working. Every year cases of this kind are brought into the front surgery dead. Here is a specimen of rupture of a small aortic aneurysm; there were no symptoms until sudden death occurred. You may ask in what sort of patient this aneurysm is most liable to occur. It is commonest in strong labouring men who perform hard intermittent work, who drink hard, and who have had syphilis. The importance of this from the point of view of the Employers' Liability Act is obvious. Aneurysm may, of course, occur without there being either history or evidence of syphilis, but in any case of aortic aneurysm the chances are as much in favour of syphilis as they are in cases of locomotor ataxy. Syphilis weakens the wall by the production of atheroma, identical in nature with the weakening of the valves that gives rise to syphilitic aortic regurgitation. Alcoholism accentuates this change, and intermittent hard manual work causes intermittent increases in blood pressure,

which make the weakened vessel wall first of all bulge, and finally rupture.

I need not spend much time in discussing cases of aneurysm in which the lesion has been diagnosed correctly long before death occurs. It is clear that if an aneurysm, known to exist, is bulging against the trachea, it may cause erosion of the latter, weakening the wall until, finally, there is a hole through it, and the patient suddenly brings up quarts of blood, and dies. It may similarly open into the œsophagus, the pleura, the mediastinum, the retroperitoneal tissues, and so on. Probably the rarest site of rupture of an aortic aneurysm is externally through the skin. One instance of its doing so, however, is recorded by these wax models from our museum. Although the majority of aneurysms that rupture elsewhere than into the pericardium have produced symptoms or signs previous to their rupture, I must remind you that even here the bulge of the aorta may be small enough to have caused no obvious pressure effects, so that the rupture and sudden death occur unexpectedly. Tremendous hæmoptysis or terrific hæmatemesis may occur in this way in persons who have been enjoying what seemed to be perfect health.

I show you here a specimen in which you will see an aortic aneurysm which will barely admit the tip of my little finger; you see it is bulging against the trachea, just above the bifurcation. There is no real obstruction to the airway; and there were no symptoms at all until the patient had profuse, sudden, and fatal hæmoptysis. Sudden death may also occur when an aortic aneurysm opens into another vessel, such as the pulmonary artery, the superior vena cava, or into some portion of the heart, such

as the right auricle. Specimens exemplifying each of these accidents I show you here.

As an example of the third group of cases, in which sudden death occurs in a patient who has had definite symptoms previously, but in whom some other diagnosis than aneurysm has been made, I will tell you of a mistake which I made when I was house-physician. It was so instructive a mistake that I learned much from it myself, and I hope you will learn from it, too. I want you to remember that there are aneurysms which do give rise to symptoms, but those symptoms, unless you are careful, you may attribute to another cause. The most difficult aneurysm of the aorta to diagnose is one on the descending thoracic aorta, beyond the left recurrent laryngeal nerve, in the posterior mediastinum below the fifth dorsal vertebra. The only tubes it can obstruct there are the œsophagus and the left bronchus; as the œsophagus is mobile, it often gets away to one side and escapes obstruction. The aneurysm usually causes pain, because it is eroding the vertebræ, but the pain, being felt between the shoulders, is apt to be discounted on account of the commonness with which dyspepsia causes similar pain under the left scapula or between the shoulders. With aneurysm, however, it is a very constant pain, and it is usually associated with a decided tenderness on palpation at some particular spot on the left side behind, thus differing from the pain of dyspepsia. It is also important to remember that an aneurysm in the descending aorta is almost certain to obstruct the lower divisions of the bronchus on the left side. The physical signs you get from this are impairment of the percussion note over the lower lobe of the left lung, with deficiency of

the vesicular murmur in the same region, with or without a few crackling râles. All the abnormal physical signs point to something being wrong in the left lower lobe, and they simulate the signs of thickened pleura with fibrosis of the lung or something of that sort; indeed, the obstruction to the bronchus by the aneurysm actually does cause fibrosis of the affected lobe, with signs precisely similar to those that would result from a pleuritic effusion that had been slow to clear up. Moreover, long before the aneurysm ruptures there is irritation of the stenosed bronchus, and the sputum may become streaked or stained with blood. Picture to yourselves, therefore, a man who complains of pain in the left side of his chest behind, who has foul purulent sputum streaked with blood, who is thin, has slight pyrexia, who is losing weight, and who has an impaired note with deficient vesicular murmur all over the left lung, and a few crackling râles to be heard here and there, and you will see that you might easily diagnose the case as phthisis, as I did. The sputum was examined for tubercle bacilli, and it was reported that in addition to many pyogenic cocci, a single acid-fast bacillus had been seen. The patient I am speaking about was in 35 bed, Stephen ward, and on the diagnosis of chronic phthisis, when his general condition seemed to have improved as much as it was likely to, he was allowed to get up three or four days before Christmas to assist in decorating the ward with ivy and holly. His excitement increased his blood-pressure sufficiently to burst his undiagnosed aortic aneurysm, and he suddenly died. The flood of blood that came from his mouth and nose so terrified everyone in the ward that the Christmas festivities were changed

to gloom. So remember, therefore, the importance of accurately accounting for a localised pain in the back, with physical signs suggesting partial obstruction to the left lower bronchus.

I have only one or two more observations to make about cardiac cases. In the museum we have an example of a very exceptional form of cardiac rupture—namely, rupture of the auricle. We must bear in mind particularly that rupture of the heart generally takes place at one spot—*i.e.*, the apex of the left ventricle. A rupture of the auricle is a very exceptional case. Another interesting specimen from the Museum, rarer even than rupture of the auricle, is a perforation of the aorta by a fish-bone. The case may remind you of a scene in a certain novel in which one person is represented as stabbing another, the end of the dagger protruding into the aorta. The novelist called in an imaginary medical man, who recommended dilute hydrochloric acid by the mouth to dissolve away the end of the dagger. The treatment was carried out, and the patient got quite well. The specimen I have just mentioned is that of a thoracic aorta laid open to show upon its posterior surface a ragged laceration. On the reverse of the specimen the œsophagus exhibits a deep ulcer, while below is mounted the fish-bone which was the cause of the trouble. The patient, aged 22, was a trooper in the 2nd Life Guards, who died five days after swallowing what he thought was a piece of gristle with his food. He first complained of indigestion and a feeling of heaviness in the chest, and on the symptoms becoming worse he was admitted into the hospital. A day or two after admission, while leaning over and talking to another patient, he suddenly brought up

quarts of arterial blood and died. This may fitly come into the category of sudden deaths in the apparently sound, as the symptoms were not thought to be serious.

Having discussed the cardiac and aortic causes of sudden death in a former lecture, let us now pass on to consider brain conditions that may end in a similar way. First and foremost of these comes apoplexy, or cerebral hæmorrhage. This is an affection of the elderly, though it is possible to have a cerebral hæmorrhage in the young, and it is commoner in men than women. Aldermanic banquets are responsible for quite a number of cases of cerebral hæmorrhage, especially when there is added the mental excitement of making a speech. The main features of cerebral hæmorrhage are so familiar to you all that I need not dwell upon them here. The commonest place in which to find a hæmorrhage in the brain is said to be the internal capsule, but the point I wish to lay most stress upon is that an internal capsular hæmorrhage does not begin in the internal capsule, and it is erroneous to talk as if it had its origin there. It starts outside the internal capsule. There is an artery called the lenticulo-striate, which enters the brain upon its infero-external aspect from the Sylvian fissure, running forwards and inwards, first to the lenticular nucleus and then across the internal capsule to the caudate nucleus. It is this artery that leaks and gives rise to hæmorrhage. If we cut almost half way down the brain in a horizontal plane we expose the internal capsule and its surroundings in a way that I may represent to you on the black-board thus.

Now let me represent the various degrees of the lesion in ordinary apoplexy. If we have a very small hæmorrhage which does not prove fatal, we are sometimes able to follow up the patient until he dies, perhaps five or six years afterwards, from another cerebral hæmorrhage. We may then find the effects of the former hæmorrhage as well as of the one that afterwards proved fatal. A specimen in the museum shows the remains of such an old hæmorrhage here in the lenticular nucleus, external to the internal capsule, and this is the commonest place for it to start. As it increases it extends along the course of the vessel towards the internal capsule, until finally a big hæmorrhage destroys the internal capsule and all the structures in its neighbourhood, extending into the third ventricle, across into the opposite lateral ventricle, and also down the iter of Sylvius into the fourth ventricle. The chief result in any case is a hemiplegia, but it depends entirely upon how big the hæmorrhage is what the course of the case will be. One may distinguish at least four degrees of this common form of cerebral hæmorrhage, namely, (1) that in which there is only a small extravasation of blood, confined to the lenticular nucleus, and barely compressing the internal capsule at all; (2) a slightly larger hæmorrhage, still confined to the lenticular nucleus, but exerting decided pressure up against the internal capsule; (3) a bigger hæmorrhage, no longer confined to the lenticular nucleus, but extending and destroying neighbouring structures, including the internal capsule, but not vital centres in the medulla or elsewhere; (4) a hæmorrhage so extensive as to be incompatible with the continuance of life. You will understand from these diagrams

what I mean, and you will realize how some people may get complete paralysis of one side of the body for a very short time—a day or so—and then lose it completely; other people may have apoplexy, with complete paralysis of half the body which may last ten days, a fortnight, or three weeks, and yet get absolutely well; others, again, may suffer from cerebral hæmorrhage causing a hemiplegia from which the patient never recovers—there may be temporary improvement, but not complete cure; and, finally, there are very bad cases that rapidly end in death. In the first, the slight instance, in which the blood exerts little, if any, pressure against the internal capsule, the reason of the hemiplegia is the sudden upset of the balance of circulation, most marked where the hæmorrhage is, so that the adjacent internal capsular fibres are for the moment prevented from carrying impulses, whilst as soon as the balance of circulation is restored, the fibres conduct again and the patient seems to be perfectly well. The next degree of hemiplegia, which takes a few weeks to get better, but ultimately recovers completely, depends on there being something squeezing up against the internal capsular fibres and compressing them without destroying them; they will not conduct impulses until that pressure has been removed, that is to say, until the clot has become partially absorbed—a week or two after the seizure. The third degree will never recover completely, though there may be slow improvement even for months after the apoplexy occurred. The greater the number of internal capsular fibres that have actually been destroyed, the greater will be the degree of hemiplegia that persists, in spite of som

attempt at innervation by the other hemisphere of the brain.

One other point about cerebral hæmorrhage may be new to you, and that is its possible association with glycosuria. You may be called to see a man who is comatose, and in trying to determine the cause of the coma you will probably examine the urine. Not infrequently you will find that the latter contains sugar in abundance. Remember that glycosuria in cases of coma by no means necessarily indicates diabetes; for if the extravasated blood in a case of cerebral hæmorrhage finds its way into the lateral ventricle, as it often does in a serious case, it is easy for it to travel thence into the third ventricle and thence by the iter of Sylvius to the fourth ventricle, where it may interfere with the surrounding centres and lead to Claude Bernard's "Stichpunkt" glycosuria.

Pontine hæmorrhage may cause glycosuria in exactly the same way; and sudden death from pontine hæmorrhage is due to damage to the vital centres in the medullary region. I would ask you to remember how the thermometer assists you in distinguishing between pontine hæmorrhage and opium or morphia poisoning; in each case there is coma, with flaccidity and pin-point pupils, but with the former there is pyrexia going on to hyperpyrexia, and with the latter hypothermia.

Let us now discuss another affection of the brain which may lead to sudden death — embolism. It sometimes happens that a person dies suddenly as the result of cerebral embolism. Nine out of ten times the cause of that is something the matter with the heart, so that although it may seem to be a cranial condition, it is primarily a cardiac cause of sudden death. Of

the heart lesions which are not malignant, mitral stenosis is that which is most commonly associated with cerebral embolism, but more important and serious than simple mitral stenosis is infective endocarditis. You might expect cerebral embolism to be fatal every time it occurs, but that is not by any means the case; indeed, in mitral stenosis the embolism may cause hemiplegia without even any loss of consciousness. Occasionally, however, cerebral embolism proves immediately fatal, and at the autopsy it may be very difficult to detect anything wrong with the brain at all. Death has occurred so abruptly that the brain tissue has had no time to become softened or discoloured, and the embolus may be entirely overlooked unless the vessels are cut up and examined with great care.

In infective endocarditis cases the embolism may not itself prove fatal, and yet the patient may die suddenly a week or ten days later with cerebral symptoms suggesting cerebral hæmorrhage. This may occur even in quite young patients—a boy or a girl who has had a valvular lesion, upon the top of which fungating endocarditis has developed.

This specimen will show you what happens in those cases. You see here a small but ruptured aneurysm of the internal carotid artery. The patient had fungating endocarditis from which cerebral embolism resulted owing to a piece of the clot from the affected valve being carried up into the middle cerebral artery. The clot contained micro-organisms which multiplied and softened the wall of the artery. The vessel bulged at the softened spot, and in that way there was produced an aneurysm, which, as its walls were more and more softened and thinned,

finally leaked, causing a cerebral hæmorrhage. The patient suffered from hemiplegia and coma as the result of the original embolism, but after the first day recovered consciousness and seemed to be doing famously ; but a week later a sudden ending occurred synchronously with the bursting of the cerebral aneurysm.

Another point in regard to intracranial lesions that I would ask you to remember is the possibility that when you diagnose ordinary cerebral hæmorrhage you may be dealing with a much graver condition, namely, hæmorrhage into a tumour. Try and find out whether a tumour is present ; and therefore do not omit to look at the optic disc to see if there is any optic neuritis. A cerebral tumour does not always give rise to obvious symptoms unless it either irritates the motor cortex and causes Jacksonian epileptic fits, or else interferes with the motor cells, so that there is paralysis ; or unless by its mere bulk it causes enough increase in intracranial pressure to produce headache, vertigo, vomiting, optic neuritis, and general convulsions. There may be a glioma or a sarcoma growing insidiously in the brain, and the patient may have no more symptoms than increasing headaches. In such a tumour there are often many thin-walled blood vessels, and one or more of these may rupture and cause death from what may seem to be a simple cerebral hæmorrhage.

Leaving the brain now and passing on to lung conditions that may cause sudden death we find at least three that are important ; these are (1) pulmonary embolism, (2) pulmonary hæmorrhage, and (3) very large pleural or pleuritic effusion. Pulmonary embolism may vary much in degree ; multiple small emboli in the lungs are by no means necessarily fatal ; their gravity

depends on how ill the man is, on what is the matter with the patient already, and on whether the emboli contain bacteria which go on to produce abscesses in the lung. The kind of pulmonary embolism which is most serious occurs mainly in the practice of those who operate much. Its percentage incidence is fortunately small; but when it does take place it occurs about the tenth day after some major operation, particularly abdominal, or about the same time after a normal or a difficult labour. It generally occurs after there has been some movement of the patient. If there has been thrombosis in the iliac veins, for instance, after an operation on the vermiform appendix, movement may detach a big clot which has not had time to become organised, and now and then in hospital a patient has had the operation wound heal by primary intention, and has been progressing famously in every respect until, on beginning to sit up in bed, he suddenly falls back dead. The clot, suddenly detached in this way, has been carried along and impacted in a main pulmonary artery. It is important, therefore, not to be in too great haste to move people after operations, or after labour, particularly if there may have been any inflammatory focus near big veins, as is the case after appendicitis. What will such lungs show at the inquest? How can you tell there has been a suddenly fatal pulmonary embolism? I want you to realise that, unless you are careful, you will find nothing to show for it at all. You might expect, perhaps, that there would be an obvious infarct in the lung, but death occurs so suddenly that the lungs themselves have no time to alter from their normal appearance. When you suspect that there has been a large pulmonary embolus,

you need to cut open the pulmonary artery inch by inch, and even then if you are not careful you will wash away the only thing which will tell you the cause of death, that is, a large irregular pale clot which is of such a shape that it was obviously not formed in the place in which you find it. You need to be extremely careful in autopsies of this kind.

With regard to rapidly fatal pulmonary hæmorrhage, the only cause of that, apart from aneurysm of the aorta rupturing into the lung or into a bronchus or the trachea, is the bursting of a small aneurysm on a pulmonary arteriole, especially in cases of chronic phthisis with cavitation. The tuberculous process eats away the lung tissue gradually, and the part which is in closest relation to the blood-vessels is eaten away last. The vessels may remain after all the surrounding lung tissue has been eaten away; and in many cases of phthisis you find that there are big irregular cavities, with bands of tissue running across them from side to side, referred to in books on pathology as "bridles." I show you examples of these in these specimens from the museum. Inside each bridle there is a pulmonary arteriole. If the blood were clotted inside the latter it would not matter, and in ninety-nine out of one hundred the gradual extension of the infective process causes thrombosis before the bridle is ulcerated away. Sometimes, however, the wall of the arteriole becomes so softened by being bathed in the pus within the cavity in the lung that its wall becomes bulged out into one or more small aneurysms of which I show you specimens here, and when bleeding occurs, even from such a small aneurysm as this, it may be very rapidly fatal. Bleeding in cases of phthisis may not be

at all dangerous itself if it is due only to the opening of capillaries or veins and not of arteries; but when the blood is escaping from a pulmonary arteriole it is a very serious thing indeed. Nevertheless it is very difficult to tell clinically which is going to be a fatal pulmonary hæmorrhage and which is not.

In illustration of this point, I should like to tell you of a personal experience I had when I was H.-P. I had to deal with two apparently identical cases at the same time in the front surgery. Both were cases of phthisis, and both were coughing up blood. One patient was a man, the other a woman; the physical signs were very similar in each. It so happened that I had a male bed available, but not a female bed, and I had to send the female case home. The male case I took in. The man died that night of an extraordinarily severe hæmorrhage, and the woman recovered entirely for the time being. There was nothing to choose between the two cases, so far as could be judged clinically; it was only an accident that the one taken in was the one that was to prove suddenly fatal. It might well have been the other way about. The point is that it is impossible to tell clinically how serious a pulmonary hæmorrhage in a case of phthisis is going to be, and if you carry in your minds these museum specimens of pulmonary aneurysms you will, I am sure, treat all such cases with the very greatest care.

There are a few more causes of sudden death mentioned in the list I gave you in the former lecture, but I have not time to go into them in detail. You should be familiar with the fact that spinal cord conditions are fatal in proportion as they implicate the origin of the phrenic nerve; when that is destroyed the patient dies,

for he ceases to breathe. Such a death may occur suddenly, but it does not always do so; it may result from injury, hæmato-myelia, or fracture of the cervical spine. Inflammations of the grey matter of the spinal cord may spread upwards at a great pace, and the resulting death may be almost sudden. Landry's paralysis, also known as acute ascending paralysis, may possibly be an example of this—fatal cases being so acute that the tissues have had no time to alter their appearances from the normal.

Do not forget laryngeal conditions as a cause of sudden death. Of these I may mention five in particular. Remember that in laryngeal diphtheria the patient may seem to be doing quite well, and you may decide that the obstruction to breathing is not bad enough for tracheotomy; and yet, in addition to the mechanical stoppage of the larynx due to inflammation, there may presently be added obstruction from spasm of the laryngeal muscles, with the result that the patient who appeared to be doing so well may be dead in less than a minute. People, especially children, with any laryngeal inflammation causing dyspnœa are never safe to leave alone, and tracheotomy instruments should always be ready at their bedside. Secondly, apart from inflammations of the larynx, you should be familiar with the fact that two types of acute œdema of the larynx may occur, viz., (1) inflammatory œdema; (2) acute œdema occurring in Bright's disease, or in a peculiar hereditary complaint known as angeio-neurotic œdema. This acute œdema may cause sudden death. Thirdly, there are cases of extraordinarily sudden and severe cases of inflammatory œdema of the larynx, acute suffocative œdema, as it is called. This is a streptococcal condition,

extraordinarily virulent; and the patient may be asphyxiated in a short time unless prompt surgery gives an airway. Fourthly, paralysis of vocal cords may be a cause of sudden death. In syphilitic subjects particularly, special portions of the brain may suffer from nerve-cell degeneration. If the centre controlling the movements of the vocal cords is attacked, it is a most unfortunate fact that it is always the abductor muscles of the pharynx that get paralysed first, so that before the adductors become paralysed there may be a sudden bringing together of the cords by adductor spasm, and unless tracheotomy is performed instantly the patient cannot breathe, and death occurs as certainly as if he were killed by strangulation. You will appreciate the fact that there may be next to nothing abnormal to be found post-mortem in such a case, and the cause of death may be thought to be unnatural unless a clear clinical history is obtainable. Fifthly, be very careful how you hasten to open a quinsy by plunging a scalpel into a tonsillar abscess. It is possible that when you put a knife into a large abscess in the tonsil, a large amount of pus may squirt out, and if the patient happens to inhale at the same time, a large quantity of that pus may be inhaled into the larynx, a very serious matter, which may cause sudden death. I have seen a case in which this dire accident occurred.

Before concluding I wish to touch for a moment upon an entirely different condition, that of Addison's disease. It is well known that this is a complaint in which the main symptoms are progressive weakness or asthenia, pigmentation of the skin and of the buccal mucosa, and fainting attacks. There may also be vomiting and irregularities of the heart's

action. The disease is to all intents and purposes incurable, and we may be asked how long a given case of Addison's disease is likely to live. The patient may live for months or years; he may die of some intercurrent malady such as pneumonia; he may develop general tuberculosis; but I want you to know that a patient suffering from Addison's disease may seem to be just as well to-day as he was a year ago, and yet may be dead before to-morrow. Addison's disease, therefore, is a possible cause of sudden death.

I am obliged to leave a great deal unsaid, and to pass by many causes of sudden death quite unmentioned; but I trust that at least one or two of the points that we have discussed may some day be of use to you.