An Address

ON

POST-MORTEM EXAMINATIONS WHICH DO NOT REVEAL THE CAUSE OF DEATH.*

By FREDERICK J. SMITH, M.D., F.R.C.P.,
Physician to the London Hospital.

THE subject of this address is only one chapter out of a very large question upon which my attention is closely engaged—namely, the *post-mortem* examination from a medico-legal point of view—but it is perhaps the most important chapter, for within it may come many cases of concealed murder which cannot be detected by an autopsy, or at least are not likely to be detected except by either a happy chance or preternatural ingenuity on the part of the medical man.

One might put the question, Is the occurrence of an autopsy without pathological appearances to account for death an example of any large class of deaths, or is it an accident due to carelessness or an incomplete survey of the conditions found?

The daily press of the evening genus, and more particularly the halfpenny species, would lead one to suppose that the latter is the case when one finds it gravely reported that an expert specially engaged by one of His Majesty's coroners stated that a man died from "threatened apoplexy." One can imagine that the appearances were really compatible with death from natural causes, but that something more in the nature of a definite diagnosis was required from one with such expert knowledge. It is statements like this that bring expert evidence into ridicule. But there can be no question but that the first hypothesis is the correct one: one has only to think of a few of the diseases of the nervous system—epilepsy, for instance—or some of the chronic scleroses; or, again, of some of the zymotic diseases, such as whooping-cough, to realize at once that in the absence of the clinical history of the case a *post-mortem* examination, done with the most scrupulous care, will fail to reveal the cause of death in the absence—aye, and even in the presence—of a prolonged microscopical research. Or take, again, numberless vegetable poisons, without something to go upon suggesting a prolonged and careful chemical analysis, what chance would there be of detecting death from any alkaloid, or similar substance, possibly with the exception of strychnine? I know of none that would reveal its presence to naked-eye inspection. The argument need not, however, be laboured, for it is only in the mind of ignorance and quasi- or pseudo-specialism, that has for its own notorious reputation to twist the indefinite appearances of natural death into some alleged definite indications of the precise cause of death, that any doubt exists. The larger one's acquaintance with natural *post-mortem* appearances the greater is one's hesitation to attribute these to some fixed and definite cause acting while life was still present, or even to ascribe death to some gross change found.

Our records at the London Hospital for a period of some nineteen months finally put a crown of proof on the argument. During these nineteen months there were 2,123 *post-mortem* examinations performed carefully and recorded with exactitude, and in no less than 102 there were found no naked-eye changes whatever to which death could be attributed. I propose to briefly analyse these 102 cases, and then to consider some reasons why such need not surprise us.

There were in the period covered by my report 150 cases of death ascribed by clinical evidence to marasmus—that is, wasting without obvious clinical cause—and diarrhoea and vomiting, and of these no fewer than 83 showed nothing whatever in any internal organ to which death could be ascribed, and in all these it would have been impossible from autopsy alone to explain why the thread of life had snapped when it did.

The remaining 19 cases were of various denominations, the most frequently occurring being prematurity, of which there were four examples. It might be argued that immaturity can be detected by inspection, and so it can in many cases born at six, seven, or even as late as eight months, but if immaturity be taken in its physiological sense of incapability to live an extrauterine existence, there will be found several in which by inspection alone it would be hazardous to assert

positively that such was the case. I have, for instance, 5 such cases, aged respectively 1, 8, 18 days, and 5 and 8 weeks, the first of which was born during eclampsia of the mother. Besides these children there were one at 7 weeks, in which a guess was made that the child might have had congenital syphilis; one at 4 months, with liver and spleen somewhat larger than usual, but nothing else; one at 20 months, in which there was a doubtful history of laryngismus stridulus; and two at 2 years each, in one of which there was some slight evidence of rickets, but in the other every organ in the body seemed perfectly healthy, and no reason could be ascribed why life, which had lasted for these two years, should not have continued. The remaining 5 cases were adults. One, a girl aged 22, was known to have had acute mania, another, a man of 25, was seen to faint with the heat, and died at once, and the other three remained without a label from lack of clinical and *post-mortem* evidence, and would in former days have been described as death from the visitation of God.

I maintain if this be the experience in the *post-mortem* room of a hospital with a large medical school attached, a similar experience must be common in places where no critics are present to ask questions or to discuss the morbid appearances disclosed. There is no keener critic than a body of medical students. The reasons why we do not hear of more verdicts, "cause of death unknown," is, first, that when a medical man who has seen the patient during life does the autopsy he is quite competent to say that what he found *post-mortem* was quite compatible with the symptoms he had observed during life and can label the disease accordingly; and, secondly, that when a stranger does the autopsy he magnifies natural appearances into morbid ones and makes a statement accordingly, a statement which nobody cares to or perhaps can controvert.

How such cases occur is after all not very difficult to answer, for if one takes a general survey of all the possible causes of death, one is immediately struck by the very few absolutely tangible and definitely certain causes of death of which the naked eye makes us at once aware.

Two or three limbs torn off or crushed and mangled, an internal organ lacerated, with the pleurae, peritoneum, or meninges filled with blood; severe cerebral haemorrhage or extensive softening, a few forms of extensive kidney destruction, pus deluging a serous membrane, a consolidated lung, cancer blocking a tube or causing perforation thereof, a few cases of corrosive poisoning, are nearly all in which we can positively assert that such a condition is immediately or after a very short interval quite incompatible with a continuance of life. In the great bulk of cases, probably about 95 per cent. of all cases of death, kidney, heart, lung, stomach, may any or all of them be grossly diseased, and we may say that the disease was the cause of death, but only because we have known something of the symptoms that preceded the fatal termination; they are none of them in their nature necessarily fatal within a given short period of time, and our evidence before the law must run, "I found gross disease of such and such organ, and it would be sufficient to account for death sooner or later, and may have caused it now."

Apart from this line of reasoning we have well-known physiological, physical, and physico-chemical principles upon which to rely. The basis of the first being the fact that cellular and somatic death or the death of the individual in the ordinary sense of the word and the death of the individual cells of the organs by no means coincide, the interval between the two events being very variable, possibly amounting to several hours; in physiological experiments this is well shown. The physical principle is the well-known law that fluid will when left alone sink down to a position of stable equilibrium; in other words, and directly applied to our subject, blood while liquid will sink and soak into the lowest possible part of the vessels and organs of the body, filling them to the highest available point, and thus simulating *ante-mortem* congestion to such an extent that the microscope may be necessary to distinguish between a vital reaction to an irritant and a mere physical phenomenon. The physico-chemical principle is the well-recognized fact that cells—both those necessary to life and those less important ones—may be slain by substances of a foreign nature gaining access to them and stopping their vital functions without leaving any traces behind them; this subject has been the object of many physiological researches, and proved up to the hilt, but one might illustrate it by asking what changes would any one expect to find after a hypodermic dose of any powerfully poisonous alkaloid such as conine, nicotine, morphine, or strychnine, and yet assuredly death is

* Delivered before the Medico-Legal Society, December 12th, 1905.

directly due to the physico-chemical action of these substances upon nerve cells.

We may consider these points from a practical point of view by taking the individual organs of the body, those at least, such as the brain, heart, and lungs, which are necessary to life and disease of which may cause death; indeed, it is usually asserted that death only takes place through one or the other of them, an assertion which fairly well expresses the truth, though in some, such as haemorrhage and septicaemia, it might be difficult to assert that one only was the ringleader in the catastrophe.

The brain: A dead body is invariably laid on its back, and hence the meninges at the base of the brain become full of blood, usually liquid, and unless care be taken, it may be assumed that meningitis or cerebral congestion of a fatal character was present before death.

The gross organic changes such as haemorrhage, gross softening tumours, abscess, pus on meninges, tubercle and adherent meninges, are easily detectable and not likely to be overlooked, but such diseases as epilepsy, convulsions due to peripheral causes, leave no traces by which they can be recognized. An excess of submeningeal fluid or excessive appearance of puncta cruenta may make one suspect that they owe their origin to *ante-mortem* influences acting fatally upon the brain, but in uraemia, in bronchitis and emphysema, in pneumonia, in morbus cordis, I have frequently found the brain in a condition indistinguishable from that which might easily be caused by these troubles, and if one thinks for a moment about the actual dying of the patient—the real essential *why* did he die in these cases—it is very easy to see that they frequently do die in all sorts of diseased conditions actually and proximately because of the serious disturbance of the normal physiology of the brain.

Meningitis itself when early requires a good diffuse daylight for the recognition of the simple dry greasy appearance presented in such a condition, and I have more than once been in doubt whether it was present or not, and should have overlooked it—even when tubercular in origin—had I not had the *ante-mortem* symptoms to guide me.

With regard to the heart, it is an established axiom in physiology that it never stops in systole but always in diastole, and yet *post mortem* it is very frequently found in a condition of apparent contraction due to rigor mortis, and as rigor mortis is universal in muscle the following is a justifiable statement: If the heart be found flabby, with other muscles stiff, it is probable that there is something in the condition of the heart muscle itself to account for it, some form of degeneration—fatty, fibrous, or simple—but to state that this was the cause of death is going much beyond anything we have justification for.

Take any disease you like—and one can undertake to say that the heart may in two consecutive cases be found in the one contracted and in the other flabby, in the one a good colour and in the other a bad colour—and who can say without the clinical history whether the heart was the real cause of death or whether it was merely what one may call an accessory to the fact. In carcinoma brown atrophy is a very common appearance; in cirrhosis of the liver, in fatty liver, in old age with atheroma, a fatty heart is frequent, but fatty degeneration takes a long time, and who by naked eye or even with the aid of a microscope can say at what stage of the diseased process the organ failed from absolute incapacity, or that it has been only unequal to some unusual strain? Again, the opposite is true, and cancer, cirrhosis, etc., may kill, and yet the heart be found quite healthy. Are we, then, in the one case to say the heart killed and in the other the independent disease? Such a position is one of pure guesswork, and only the clinical history can decide.

It is when the lungs are considered that the greatest difficulties are met with in deciding between appearances that may have been the ultimate cause of death and those which may be an accidental accompaniment of death, and also those which are mere *post-mortem* effects. There can be no question but that when somatic death takes place—death, that is, of ordinary parlance—all the cells of the body do not die at the same time, and from the mucous lining of the bronchial tubes a certain amount of mucus is exuded *post mortem*, and it is impossible to estimate whether the mucus thus found was enough to cause death or not. Again, supposing, as is frequently the case—and I believe almost universally—a little pus can be squeezed out of the medium-sized and smaller tubes, are we, therefore, to say that bronchitis was the cause of death? I hold very strongly we are not justified in so doing unless we have an account of the symptoms imme-

diately preceding death, for these give just the information we require.

Haemorrhage into the lung, tubercle, consolidation, subpleural ecchymoses are all obvious and evident enough, and their indications may be translated into lethal expressions; but what about oedema of the lungs? It often kills, I have no doubt; but it is equally often found in septicaemia, uraemia, apoplexy, etc., when its indications are, to say the least, very doubtful. Supposing, then, it is the only gross change found, are we justified in saying it was the cause of death? Assuredly not without the clinical history of symptoms preceding dissolution. And we are bound to include oedema of the lungs amongst those *post-mortem* appearances which leave death unexplained.

Again, what is known as hypostatic congestion of the lungs may be what its name implies, a pure and simple mechanical effect capable of taking place *post mortem*, though often enough in certain debilitated conditions of the system it is a very real *ante-mortem* phenomenon easily capable of killing, and it wants very great experience of clinical history combined with *post-mortem* findings to say how far it was the cause of death. The phenomenon, moreover, can hardly be distinguished from the very early stage of pneumonia; in both there are precisely the same dark red, almost black, condition of the bases of the lungs or of one lung, and it requires very nice discrimination and long experience to say that degree of resistance to the finger is natural, and that is pathological; in fact, again, without the symptoms and the temperature that preceded death, I believe the distinction to be oftentimes impossible.

One cannot pass over in silence the extreme difficulty one meets with in deciding from naked eye anatomy whether the kidneys are or are not healthy. I admit that gross changes are common enough and easy enough to detect, and yet one often has to call the kidneys normal to the naked eye when the clinical history has shown that they at any rate allowed much abnormal material to filter through them, and that they were the clinical cause of death whatever may have been the proximate and essential cause.

Even if gross changes be observed we are again face to face with the difficulty—they must at some time lead to death, but have they caused it now?

These cases are very important, and it follows from what I have said that they occur with moderate frequency; we have seen also the reasons why they occur, and we have now to consider their importance from a medico-legal point of view. This may be summarized in a sentence, though details must be discussed rather more fully. That procedure of a coroner's court by which a verdict is arrived at on the evidence of the pathologist alone is simply a farce, for no such person, even if skilled, can by naked-eye evidence alone determine the true cause of death in a completely satisfactory manner. It is absolutely essential that with him should be associated the medical man who last saw the deceased alive. Do not let me be misunderstood. I am not denying the value of expert pathological evidence; I am merely stating that it is insufficient for the needs of justice and the safety of the public, and requires to be supplemented by other medical evidence; but I do go so far as to say that if only one person can possibly give evidence at an inquest, that one should be a medical man who has seen the person alive, even if it be only in his death agony.

The real genuine pathological appearances of organs as opposed to the mere accidental exudations and congestions, so often distorted by self-assertive would-be experts into evidence of the cause of death, are not likely to be overlooked by any medical man, though I must admit that it is possible they may be, for I have myself seen carcinoma of the sigmoid leading to perforation of the gut remain entirely unperceived by a medical man; but this class of mistake is of infinitely less importance than that of attributing a death from morphine poisoning to bronchitis, which is a very possible or even probable mistake which might be made by one who relied upon *post-mortem* evidence only.

Let us imagine a person in South-West London, for example, is taking medicine containing morphine and that some evilly-disposed person gives an overdose: the medical man in attendance feeling that the disease should not have proved fatal may refuse a certificate; a *post-mortem* examination is made and an inquest held without any reference to or communication from the medical man. Alleged signs of bronchitis are found and a verdict in accordance is given, and a foul murder is hushed up probably for ever. The mere possibility of such an occurrence makes one shudder, and yet the

ignoring of the medical man in attendance makes such, one might say, probable. In my own experience within the last few months a case in point has occurred: Dr. Wynn Westcott asked me in September last to make a *post-mortem* examination on two children who had died after taking liqozone, a strong solution of sulphurous acid. On autopsy I could find no possible nor even probable cause for death, and had one not known that repeated doses of simple irritant poison may leave no traces in the alimentary canal beyond those attributable to mere *post-mortem* changes I should have been obliged to say that I could find nothing to account for death had I not had the invaluable, nay indispensable—if justice was to be done—support of the evidence of Dr. T. W. Morcom Harnels, who had attended the deceased children for some three or four days, with all the symptoms of irritant poisoning. It was on his evidence combined with mine that a correct verdict was given—namely, poisoning from the effects of taking liqozone in repeated doses.

It may be urged that the mere fact of no cause being found on autopsy *prima facie* sufficient to account for death, should make the pathologist suspect some insidious form of poison, but a reference to my analysis of cases will at once show that such a connexion is by no means necessary, and common experience leads in the same direction, especially, perhaps, in that terrible cause of infant mortality so-called overlaying. I believe that in the majority of such cases there are no pathological conditions found after death.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

UNION OF SEVERED NOSE.

In September, 1904, a lady, aged 24, lost control of her bicycle, and was thrown through the plate-glass window of a shop. I saw her about twenty minutes afterwards, and found the end of her nose was missing, while her upper lip was so gashed that it was attached to the face by the internal mucous membrane only. Ten minutes later the tip of the nose was handed to me, having been found on the shop floor, and brought across the road to where the patient lay; it measured about three-quarters of an inch from side to side, half an inch vertically, and was under an eighth thick, but fortunately contained almost no cartilage. Being quite cold, it was placed in warm water for a few minutes, then washed in 1 in 40 lysol, and secured in position by four catgut stitches. The wound was then painted with tinct. benzoin. co., and a few shreds of cotton-wool, saturated with this fluid, applied. The same treatment was used for the lip. The patient was kept in bed for ten days, and allowed fluid nourishment only. Within twenty-four hours she could feel the point of a pencil when brought in contact with her nose, and acuteness of sensation gradually increased, until in about four days it was normal. The wound healed by first intention, and there was no discharge from it until a fortnight after, when the superficial layers of the skin sloughed off. Under a dressing of quarter strength boracic ointment the epidermis regenerated in a few weeks, and, excepting a little redness at the junction of tip with nose, and a slight nick in the right nostril, the organ looked normal. The lip healed by first intention. Now, several months after the injury, there is nothing abnormal to be seen, except a slight not readily observed deficiency at the margin of the nostril.

Shotley Bridge.

J. MURRAY RENTON, M.A., M.B. Edin.

THE ALBUMINURIA OF PREGNANCY.

MRS. C., aged 21, a primipara, expecting her confinement on April 1st, came to see me several times on account of repeated attacks of urticaria. She was very constipated, and the uterus was enormous. Twins were suspected.

On March 20th she had a good deal of oedema about the feet and ankles. Her urine proved to contain much albumen, and was passed in very small quantities and at long intervals. She was given a diuretic and carefully dieted. The following day she suddenly discovered she was unable to see with the right eye. Two days later, as she developed a marked internal strabismus of the same eye, the diuretic was made stronger, purgatives administered, and the patient kept in bed.

No improvement took place, and on March 26th the left eye

became similarly affected, though loss of vision was not complete.

On ophthalmoscopic examination an unusually pale condition of the fundus was found, but no evidence of haemorrhage or albuminuric retinitis. She was now passing more urine, with slightly less albumen, and the bowels were acting better. Labour commenced on March 27th.

In due course I ruptured the membranes, and after a gush of liquor amnii she called out in a joyful voice that she could see with her right eye. Shortly after she was delivered of twins, and, the second child being born, I noticed that the squint was much less marked, but she told me that she could see "two of me."

The rest of the labour, except for considerable haemorrhage, was uneventful. On its termination she could see with both eyes, but the left eye was still turned inwards a good deal. During the puerperium the oedema which, after she was kept in bed appeared in the lumbar region, rapidly disappeared.

The squint persisted until April 5th, when it too vanished, and vision became normal. The urine was collected carefully as soon as possible, and the albumen found to be lessened in quantity. She was given an iron tonic, and by April 14th there was no longer any trace of it, while her face, which had been red and puffy, had resumed its normal appearance. She has since remained in good health. The two babies were healthy and well developed. It seems remarkable that she should have escaped eclampsia.

London, N.W.

JOHN SPURWAY, M.B., C.M. Edin.

SUDDEN DEATH AND THE THYMUS GLAND.

THE following case recently occurred in my practice: An urgent message was sent me from a house a few doors away. I got there in three minutes, but found the patient—child—dead. A first glance at it showed it to be an extremely well-developed infant, about 8 months old. On each cheek was a dusky patch; the lips were slightly blue, but the finger nails of a natural colour, and there were no other signs of asphyxia. I immediately commenced artificial respiration, and persisted in it for twenty minutes, but without success. The mother, it appeared, had fed the baby from the breast, and had then put it to bed, apparently in perfect health. About a quarter of an hour afterwards she went to look at it and picked it up dead. Close questioning disclosed nothing pointing to any previous disease. The child was always thought to be extremely healthy; it invariably, however, turned on its face to sleep, and had a habit of throwing its head back in its mother's arms. An examination was made by myself, with the kind help of Dr. H. Roger Smith, the usual family doctor. The brain and all the organs were found to be healthy, with the exception of the thymus gland. This occupied the entire region from the thyroid to the diaphragm, or, by the rib marking, to the upper border of the seventh rib. On removing it from the trachea, it was found to consist of three lobes; it measured 4 in. in length, 2½ in. across, and ¾ in. thick, and weighed 2 oz. The trachea was full of half-digested milk. The enlarged thymus may perhaps explain why the child used to assume the attitudes which have been described. They were probably those in which the pressure on the thymus was least felt. In Osler's *Medicine*, under "Diseases of the Thymus Gland," it is stated that enlargement of the gland is a recognized cause of sudden death in infants, owing either to its direct pressure on the trachea or to its pressure on the pneumogastric causing spasm of the glottis. In this case, however, there was no flattening of the trachea or evidence of pressure *post mortem*. There appear to be no symptoms by which the condition can be diagnosed with any assurance during life.

Hampstead.

EDWARD JESSOP.

A NEW TREATMENT FOR "HOUSEMAID'S KNEE."

A FEW days before the case of accidental rupture of a housemaid's knee seen by Dr. Pogson I had a similar one in a much older patient.

Mrs. J., aged 68, was under my care for osteo-arthritic pain of the left knee from June 13th to July 11th, 1905, by which time it had quite subsided. On July 24th she came to show me a typical medium-sized, painless enlargement of the left bursa patellae which had arisen during the preceding fortnight. I told her to give up kneeling, and the following day went away for a holiday.

I next saw her on August 16th, and she told me that the swelling had remained stationary till about August 12th, when she accidentally fell on the stairs and heard it go pop.