Clinical Lecture

ON THE

INTERDIAGNOSIS OF INSENSIBILITY
FROM VARIOUS CAUSES.By REGINALD SOUTHEY, M.D. OXON., F.R.C.P.,
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GENTLEMEN,—Questions somewhat of the following kind are likely to be put to you one day, and are full alike of clinical interest and medico-legal importance :—What is the matter with an individual who has been picked up on the road insensible, irrational, or inarticulate, and about whom no antecedents whatever are known? Is he ill, drunk, drugged, suffering from brain concussion, or from coma after an apoplectic or epileptic fit?

Syncope, trance, catalepsy, and coma are names severally used by medical writers to designate states of insensibility which the lay public group vaguely under fits.

Syncope is fainting; it may be otherwise described as a condition of body in which there is a death-like pallor, with loss of muscular power and of consciousness. Trance is a state of death-like faintness in which, however, some consciousness is retained, although the person is unable to move or speak. A faint is ordinarily (when an individual has not lost a very large amount of blood) an exceedingly transitory affair. A person collapses rather than falls to the ground in a faint; his knees bend under him, he subsides into the sitting posture, his head drops forward, and by the time the head has thus sunk to the level of the heart, or below it, the circulation through the brain becomes sufficiently restored for consciousness to return. In a faint a person seldom bruises his face. Upon waking up he may feel sick or giddy, or alarmed, but his brain resumes its thinking function usually at once and entirely.

In states of trance—of which I possess no personal experience, but may refer you to accounts to be read in Smith's "Forensic Medicine," pp. 540-542, and Forsyth's "Medical Jurisprudence," pp. 125-130, 155, and pp. 165-188—it appears that the body is to all appearance inanimate, and there is no power to move a muscle, the limbs meanwhile remaining perfectly flexible. The peculiarity attached to this condition is that the individual may hear, see, and remember all that goes on about him. There is no perceptible pulse or respiration, and hence trance has been mistaken for death, but we possess no recent or philosophical observations upon this form of faint. The recorded cases do not tell us of the temperature, or whether or not the muscles react to galvanic stimulus. Burial alive in this condition, except at sea, is highly improbable, but in no case would you be justified in making a post-mortem or consigning a body to burial unless you were satisfied from the presence of rigor mortis or appearances of putrefaction that death was actual. Catalepsy, again, is a rare inanimate condition; still I once had a case under my care which accorded well with the descriptions that have been given of it.

A young man of nineteen was picked up in Fleet-street insensible, not fallen, but standing stiffly, unable to move or to articulate. Here are the features that distinguish this state, all which he presented in other similar seizures which he had for several days afterwards while in the hospital. Thus while carrying something about, or sitting up in bed, or attempting to poke the fire, his eyes would suddenly acquire a fixed gaze, and he would remain arrested as it were in his act. We could set his limbs in any posture we pleased, and there he remained like a mesmerised cock, to the amusement of the nurses and other patients. I watched him on several occasions. There was not the slightest twitching or spasm of any of his muscles. His pulse was slow, and so were his respirations, his extremities cold and flabby, the axillary temperature remained natural. He would bear pinching or pricking without flinching, and took no account of our bawling his name close by his ear. There was no reason to think that he was feigning, and it

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would be difficult, I fancy, to feign and act a disease so well as he did. To quote from Dr. Copland's Dictionary, vol. i., p. 292: "The statue-like appearance and muteness of the cataleptic alone suffice to distinguish this disease. There is neither the lividity of asphyxia, the pallor and general flexibility of syncope, the stertor of coma, the paralysis of apoplexy, nor the movement and dreamy mental automatism of somnambulism."

Coma, however, because arising from very various causes, is the single one of the several states mentioned by them as likely to offer much difficulty in diagnosis. Coma may be due—1st. To *pressure* exercised on the brain, arising (a) from within the ventricles, effusion therein; (b) from depressed fracture; (c) from effusions outside the brain between hair and membranes. 2nd. To *alteration* in molecular state of brain from some concussion, contusion, bruising, oedema, apoplectic extravasation. 3rd. To brain poisoning by insufficiently oxidised blood, uræmic blood, narcotics, anæsthetics, inebriants.

I cannot give you certain symptoms for diagnosing each of these causes of coma, but I may aid you in excluding some of them, and thus help you to arrive at a correct diagnosis in particular instances. The profound coma produced by serous effusion into the ventricles of the brain, to judge from what I have seen in dropsy, renal or cardiac, and by what happens in tubercular meningitis, is gradually reached through a stage of sopor. The individual at first is sleepy, then dreamy and slow of apprehension, difficult to awake, and finally incapable of being roused at all. Reflex movements may be excited for a while, later the face is pale, the temperature falls. The breath is either Cheyne-Stokes or principally diaphragmatic and stertorous. At first there is power to swallow, afterwards acts of deglutition fail to be coördinated. The pupils are non-characteristic, but more often dilated than contracted. (a) The coma due to depressed fractures, or to effusion of blood or pus or fluid between the brain and the membranes, is as sudden in its advent as its cause may have been. Usually the breath is stertorous, the pupils are contracted, and the temperature is normal or above normal, the skin perspires profusely. No or only slight reflex response is made to galvanic or cutaneous stimulation.

You will be guided to the diagnosis of fractured skull by bleeding or oozing of bloody serum from the ear or nose, or by ecchymosis of the eye, and towards large apoplectic effusion of blood upon the surface of the brain by the stertor of the breathing and complete absence of reflex irritability. (b) When the coma is due to alteration in the molecular state of the brain, local contusion, or apoplectic extravasations, the face is usually pale, the temperature fallen below normal; the reflex irritability, although variable, is apt to be greater on one side of the body than on the other; the pupils may be unequal, and evidence of some hemiplegia is generally discoverable in muscles of the face or eye.

It seems like hair-splitting in diagnosis to dwell upon what may aid you to distinguish between coma due to general compression of the brain substance and coma due to more localised brain injury, with general circulatory and nutritional disturbance, but I give you the best clues I can, such as have served me sometimes well; the evidence of any hemiplegia in a case of profound coma is always in favour of localised injury of brain rather than of general pressure.

The coma may, however, be due to some brain-poisoning, the sources of which I have grouped under heading 3. Now, deficiency of oxygen and excess of carbonic acid in blood produces coma, as, for example, in the inhalation of laughing-gas or nitrous oxide, when the face is black, the veins turgid, the skin hot, the muscles irritable, and reflex spinal irritability is heightened.

Again, the blood-poisoning may be due to the action of some gas inhaled which interferes with or arrests the oxygen-carrying properties of the blood-cells. Sulphuretted hydrogen and carbonic oxide both appear to act in this way. The circumstances of the accident, the conditions under which the person was found, as well as some significant clinical appearances, already discussed by me, should facilitate the recognition of this source of coma. This blood change, if profound, is not recovered from.

Brain-poisoning and coma by anæsthetics and inebriants are usually detectable by the odour of the breath of the comatose person.

You may occasionally feel a little uncertain as between

apoplexy and dead drunkenness. We had an illustrative case the other day in our wards. A man who had been intoxicated by rum was admitted, and very profoundly comatose he was. No reflex movements were excitable; his face was cyanotic; there were gurgling râles in his throat, and I believe he might have died suffocated if I had left him in the position in which the nurse had placed him in bed, for he had a large tongue and uvula, with somewhat swollen and cedematous fauces; and as he lay on a flat bolster, with his head back, his tongue slipped back in his mouth, and nearly closed the opening of the glottis. Propping him up with a bed rest, and pulling his tongue forwards, his breathing quickly improved, and his face assumed a less blue colour. Still for some ten minutes I was unable to get any movement of either leg by tickling the soles of the feet, and the pupils were unequal, one rather smaller than the other, and both quite irresponsive to the stimulus of light. It was true he smelt of liquor, but he was quite properly admitted by the house-physician, since treatment and lapse of time alone could decide whether his coma was due to alcohol alone, or to disease *plus* alcohol. You may remember what I ordered in that case, as a piece of practice which is prompt, rational, and attended by no possible risks—the throwing up the rectum of a pint and a half of cold water, with a tablespoonful of common salt dissolved in it. It brought him out of his profound coma at once, for he asked for a night-stool almost directly afterwards. The stomach-pumping and belabouring with a galvanic battery of a comatose person, who may be the subject of gravescent apoplexy, belong to the surgery of the past, when active practice corresponded too closely with lynch-law-like punishment.

In every case of coma count the respiration, observe the quality of the breathing, and its ordinary effect upon the blood by the colour of the lips. Examine the pulse carefully for quality, frequency, regularity. Touch the eyeballs, examine the pupils for reaction to light, their condition—dilated, contracted, unequal, irregular; tickle the soles of the feet, to test what amount of reflex muscular action can be coördinated. The breathing in most states of coma is heavy and slow; notice if it be stertorous, Cheyne-Stokes-like, wholly diaphragmatic, or if the intercostals act. Next take temperature in the axilla or rectum. A remarkably low temperature, four degrees or more below normal, betokens usually uræmic coma, acetonæmia, or grave concussion of the brain. After epilepsy, apoplexy, intoxication, the temperature is more often normal, or even slightly above normal.

Uræmic coma will again be separately distinguished most often by some slight œdema of eyelids or extremities, wax-like pallor, bronchitic sounds, very foul breath, smelling of beef-tea; a brown furred tongue, pearly conjunctivæ, and dilated pupils bespeak urea poisoning. Diabetic coma is indicated by oppressed noisy breathing, effected by abundant muscular effort, but attended by some lividity in evidence of impaired oxidising changes at the pulmonary capillaries, of which we do not at present understand the exact cause.

In the coma that has succeeded an epileptic fit you may expect to find bruises, torn or dirtied clothes, indications of the tongue having been bitten.

Finally, the mode of recovering consciousness after intoxication differs much from what is observed in persons recovering from the coma that succeeds an epileptic fit. After intoxication I have noticed that while the sense of hearing is recovered soon, the lower attributes of mind, the sensorial instincts, reappear early, the individual who was comatosely drunk hears correctly before he sees correctly, and sometimes before he can stand. It is curious to notice the manner in which he repeats a word of your question over and over again, like a parrot, and perceive how some grotesque idea grows out of the jingle or jangle of a phrase mumbled over more or less thickly.

How differently do the mental faculties recover after brain concussion and commotion as after epilepsy. How the intellectually reduced “egomet” is in a dreamy state and staggers in his gait, hears questions put to him correctly, answers them articulately, apparently rationally, but having done so straightway forgets what he has said, cannot tell where he is or what he is or was doing. He is conscious of some confusion in his own memory, and shows often some high measure of judgment in his distrust of himself, and says: “I cannot recollect where I was going.”

Intellectual wholeness or sanity lies outside to-day’s

lecture, and I mention this merely as a clue to past coma, whether from drink or cerebral commotion.

It remains for me to point out the distinctive features of opium coma. There is profound stupor, with closed eyelids, contracted pupils, upturned eyeballs; the face is pale, the skin cool, clammy; the forehead beaded with heavy perspiration; the limbs are lax, but, for a while, reflex motions are readily excited. The respiration now slackens, falls in frequency, perhaps, as I have seen it do, to as low an ebb as 6 per minute, the pulse remaining fair at 80. The coma now is very profound; you may prick, stimulate by galvanism, call, flick, stimulate by what means you please, little or no further purposive response is returned by coördinated muscular movements. The body will not walk; it is only dragged about, if the advice given has been to walk the man about. Next the lips get livid; the surface of the body colder; the breath is sobbing, and at long irregular intervals; the pulse hardly to be felt at the wrist, and death is imminent by asthenia or sudden failure of the heart’s action. Now for what may you mistake this coma, and what may be most easily confounded with it? Apoplexy into the pons Varolii, since, as was pointed out long since by Dr. Wilks, who illustrated the fact by several examples of cases that had fallen under his observation, apoplexy into the pons is attended by great contraction of the pupils.

If, then, in any case of profound coma, with extremely contracted pupils, you suspect opium to be its cause, but have no corroborative fact, no laudanum bottle, no opium-smelling substance, or mark of subcutaneous morphia syringing upon the body, or soot of opium-smoking inside the nostril to aid you, remember that the case may still be apoplexy into the pons, a source of coma not likely to be benefited either by bastinado, beating with towels, walking about, or all the extracts of coffee which can be pumped into the body at either extremity.

Clinical Lecture

ON A

CASE OF STRANGULATED HERNIA.

Delivered at Westminster Hospital

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GENTLEMEN,—The patient, Richard M—, whose case I bring before you to-day, was admitted into Northumberland ward on Wednesday, May 5th, with urgent symptoms of a strangulated left oblique inguinal hernia. He is a spare, thin, nervous-looking man, forty-two years of age, and by occupation a groom. The history that he gave was this. The hernia first appeared in the left groin about twenty years ago, coming down suddenly whilst he was jumping a fence. The size of the rupture was described as that of a pigeon’s egg, and the patient succeeded in reducing it himself about four hours after it had appeared. He was very properly advised to get a truss, and he seems to have worn it for about six months, when he left it off, supposing that his rupture was cured. The fact that the hernia did not return for nine years shows the immense importance and advantage in such cases of the immediate application of a properly-fitting truss. On its recurrence after this interval the patient states that he again succeeded in returning it, and that he resumed his truss. About two years ago the breaking of his truss led to his discarding it, and shortly afterwards the hernia came down whilst the patient was riding, and this time he failed to reduce it. He was admitted into the Great Northern Hospital, where he remained for three days. All attempts to reduce the hernia failed, and symptoms of pain and vomiting rapidly ensued. However, on the third day, when in a warm bath, the patient succeeded in reducing the hernia himself, and the symptoms ceased. His truss was repaired and resumed.