EMERGENCIES IN GENERAL PRACTICE

THE DIFFERENTIAL DIAGNOSIS OF COMA

BY

M. J. PARSONAGE, M.B., B.Sc., M.R.C.P., D.C.H.

Assistant Physician, Neurological Department, Leeds General Infirmary; Consulting Neurologist, Leeds Regional Hospital Board

Coma is a state of deep unconsciousness from which the patient cannot be roused by ordinary forms of stimulation. There is a total absence of any kind of psychologically understandable response to such stimulation as pinching the skin or vigorous shaking of the patient, but the term "semicoma" is sometimes used to describe those states of less deep unconsciousness in which there may be grimacing or withdrawal movements of a limb on the application of such stimuli.

Importance of an Orderly Approach

There are many possible causes of coma, but for practical purposes a broad distinction may be made between those cases in which coma has supervened as a late development in a patient suffering from a known disease and those in which it is an early and outstanding feature. Greater difficulties in differential diagnosis are likely to be encountered in the latter circumstances, and it is with such cases that this article is mainly concerned.

In dealing with an individual case of coma it is better to adopt a systematic but relatively simple plan of approach as opposed to one which aims at all-inclusiveness at the expense of being unwieldy and unnecessarily lengthy. A scheme based upon the following lines is therefore recommended: (1) An initial consideration of the likeliest causes in the particular patient to be examined. (2) The obtaining, wherever possible, of a history of the events immediately preceding the onset of the coma from any available source—that is, from relatives or witnesses. (3) A systematic physical examination of the patient. (4) A consideration of other less common causes if the underlying disease process is not immediately evident after careful appraisal of all the ascertainable facts.

The causes of coma can be grouped in order of importance as follows:

Cerebrovascular Catastrophes and Conditions Causing Cerebral Ischaemia.—Intracerebral and subarachnoid haemorrhage, thrombosis, embolism, hypertensive encephalopathy, terminal congestive heart failure, severe anaemia due to loss of blood or blood disease, leukaemia, Stokes-Adams syndrome.

Brain injuries, including their complications.

Metabolic Disorders and Endogenous Intoxications.—Diabetes, hyperinsulinism, renal and hepatic failure, Addison's disease.

Post-epileptic States.

Exogenous Poisons.—Barbiturates, carbon monoxide, alcohol, aspirin, arsenic, lead, alkaloids, petrol fumes, carbolic acid.

Intracranial Infections and Tumours.—Meningitis, encephalitis, intracranial abscess, primary and secondary neoplasms.

Physical Agents.—Heat hyperpyrexia, electric shock, caisson disease.

Psychiatric Disorders.—States of severe stupor in hysterical, schizophrenic, and depressive illnesses.

The History

Facts obtained by the interrogation of relatives or witnesses will often provide valuable indications concerning the likely

cause of the coma, and may well be indispensable if the patient's previous health has been regarded as good or is unknown to the practitioner.

In respect of antecedent diseases likely to be associated with the later development of coma it will be necessary to make specific inquiries, first, about such conditions as hypertension, cardiovascular and renal disease, diabetes, epilepsy, alcoholism, or drug addiction, and, secondly, with regard to abnormalities of mood or behaviour suggestive of pre-existing psychiatric or organic brain disorders. Thus a history of diabetes will immediately suggest the possibility of either hyperglycaemia or hypoglycaemia, while a history of hypertension or previous strokes will be in favour of a cerebrovascular catastrophe. On the other hand, a history of progressive loss of interest and increasing apathy associated with incontinence of urine may suggest a frontal lobe tumour; a story of recent disturbing emotional experiences in a subject who has previously exhibited hysterical symptoms will lead to suspicions of a hysterical trance state; bizarre behaviour associated with delusions or hallucinations will point to the possibility of the catatonic form of schizophrenia; while a history of increasing retardation coupled with expressions of guilt and self-recrimination or of previous similar episodes will suggest a severe depressive illness.

It must be emphasized that if the patient is truly comatose an organic cause must always be assumed; a state of severe stupor in which there is suppression of voluntary activity but not unconsciousness may be either the antecedent of an organically determined coma or a manifestation of a primary psychiatric disorder.

When coma has supervened in a person whose previous state of health is unknown or has been regarded as good, inquiry will need to be directed towards the circumstances under which it occurred. Indications of likely causes will be obtained if there is a history of recent head injury, haemorrhage, circumstances pointing to a suicidal attempt or exposure to poisonous fumes, or a recent alcoholic bout.

It is important to scrutinize all circumstantial evidence most carefully; sometimes it is seriously misleading. Thus the patient with evidence of head injury may have damaged his head through falling in a stroke, the diabetic is not immune from other disease, the hypertensive may have a cerebral tumour, and the smell of alcohol in the breath is not necessarily indicative of alcoholism. Fortunately, the physical examination will usually help to place such evidence in its correct perspective and should be systematically carried out in every case of coma.

Physical Examination

In the initial general inspection of the patient a search for signs of recent head injury is an important preliminary. Bruising or laceration of extracranial tissues may indicate that the injury is incidental, but bleeding or leakage of cerebrospinal fluid from the nose or ears and subconjunctival haemorrhage spreading forward from the back of the eye usually means a head injury severe enough to have led to a state of coma. The patient's colour should be noted (cherry-red in carbon monoxide poisoning, pale in many types of coma, but cyanotic when the coma is deep whatever its cause), and also the temperature and respiration (shallow in most forms of coma, but deep in diabetes and sometimes "hissing" in uraemia). Sweating is usually pronounced, but is strikingly absent in heat hyperpyrexia. The mouth and

tongue should be inspected for scarring (epilepsy) and pigmentation (Addison's disease), and any tell-tale odours in the breath noted. Lastly, the presence of neck rigidity should be sought and evidence of dehydration looked for.

A systematic survey of function in the various bodily systems should then be made. A brief examination of the nervous system can usually with profit be carried out first and need not as a rule comprise more than a scrutiny of the fundi with an ophthalmoscope, tests of pupillary and oculomotor functions, and a decision on the probable presence or absence of hemiplegia. The presence of the latter is established when it can be demonstrated that the limbs on one side show no movements of withdrawal when pricked, and fall more inertly than their fellows when passively raised and allowed to fall. Changes of diagnostic value that may be seen in the ocular fundi are those of renal or diabetic retinopathy, and the presence (or absence) of papilloedema may be ascertained. The significance of minor inequalities in the size of the pupils is not readily assessed, but a much enlarged pupil on one side showing little or no reaction to light is likely to be due to serious cerebral compression, while a complete unilateral third cranial nerve palsy (closed eyelids, fixed dilated pupil, and abducted eye) is sometimes to be seen in cases of subarachnoid haemorrhage. In all forms of true coma the tendon reflexes will probably be much reduced and the plantar reflexes bilaterally extensor.

In the examination of the cardiovascular system search should be made for evidence of cardiac decompensation, endocardial disease, and irregularities of rhythm, concluding with measurement of the blood pressure. The lungs should be examined for evidence of gross inflammatory or neoplastic disease and the abdomen palpated for the presence of rigidity or abnormal masses. A rectal examination will be necessary if prostatic disease is suspected in a uraemic state; and if early transfer to hospital is impracticable a specimen of urine should be obtained by catheterization and tested for the presence of albumin, sugar, and ketone bodies. Similar considerations apply to lumbar puncture, which will need to be undertaken sooner or later in all cases in which the diagnosis is not obvious; it should not, however, be performed if there is any likelihood that the patient has an intracranial space-occupying lesion.

Differential Diagnosis

The final diagnosis of the cause of the coma in any given instance will depend upon a correct interpretation and synthesis of all the ascertainable facts. In order to assist the practitioner in this task the more outstanding diagnostic features in some of the commoner conditions causing coma are given. Some of these conditions will be considered in more detail in later articles in this series.

Cerebrovascular Catastrophes and Conditions Causing Cerebral Ischaemia

Haemorrhage into brain substance occurs most commonly in late middle life, is usually fatal, and will generally be associated with severe hypertension. Hemiplegia is characteristic of *intracerebral* bleeding and is often associated with stertorous breathing and a slow, full, bounding pulse. The head and eyes are commonly deviated towards the side of the lesion, and retention or incontinence of urine is the rule. In *intrapontine* haemorrhage there is likely to be bilateral limb paralysis associated with pin-point pupils and hyperpyrexia. Spontaneous *subarachnoid* haemorrhage (usually due to rupture of an intracranial aneurysm) generally occurs at an earlier age; in such cases limb paralyses are far less common, but neck rigidity is usually more prominent and there may sometimes be unilateral palsy of the third cranial nerve. Papilloedema and retinal haemorrhages are seen in some cases, and blood is characteristically found in the cerebrospinal fluid; the latter may also sometimes occur in intracerebral bleeding, but in such cases the presence of hemiplegia and a more deeply comatose state are helpful diagnostic features.

When coma occurs in cases of *cerebral thrombosis* it is usually less profound and of more gradual onset than in

the case of intracerebral haemorrhage. Hemiplegia is often present, but hypertension is less common and there may be a history of previous strokes. An onset with sudden severe headache or vomiting is much more in favour of intracerebral haemorrhage than of thrombosis.

Coma is of uncommon occurrence in *cerebral embolism*. A semicomatose state may occasionally be encountered when there is occlusion of a large vessel such as the middle cerebral artery; diagnosis in such cases depends upon establishing the association of sudden unconsciousness and accompanying neurological signs (hemiplegia) with the presence of a cardiac or pulmonary lesion likely to be a source of emboli (coronary thrombosis, endocarditis, auricular fibrillation or flutter, pulmonary infarct, or suppuration).

Hypertensive encephalopathy may complicate benign or malignant hypertension, acute nephritis, and eclampsia. There is usually a subacute onset consisting of transient focal cerebral disturbances (amaurosis, aphasia, hemiplegia) followed by the development of convulsions and coma. The blood pressure is always raised, especially in malignant hypertension, when the diastolic reading is usually in the neighbourhood of 130–150 mm. Hg; papilloedema with or without the exudative changes of hypertensive retinopathy may be present in some cases, and urinary changes will be found when there is acute or chronic renal damage. The focal cerebral disturbances are much more transient than those occurring in cerebrovascular lesions, and the usually good response to treatment (venesection and lumbar puncture) may be a helpful diagnostic feature.

Brain Injuries

The patient remaining in a state of coma after the receipt of a head injury must be presumed to have sustained either a *contusion* of his brain or an injury complicated by *intracranial bleeding*. The latter is particularly likely if the patient has recovered from the immediate effects of the injury and subsequently lapses into deepening unconsciousness. This indicates increasing cerebral compression and will be associated on the side of the haemorrhage with a progressively dilating pupil which fails to react to light.

A history of relapsing impairment of consciousness ranging from drowsiness to coma suggests a *chronic subdural haematoma* which is usually a complication of a comparatively mild head injury, although such a history may sometimes be lacking. Males are much more commonly affected than females, and localizing signs are slight or absent until a late stage, although a dilated pupil is often found on the side of the haematoma.

Metabolic Disorders and Endogenous Intoxications

In *diabetic coma* the onset is gradual and follows a period of severe ketosis (dyspnoea, anorexia, nausea, vomiting, drowsiness, and diffuse abdominal pain). The patient is dehydrated and collapsed, acetone is present in the breath, and the tests for sugar and ketone bodies in the urine are strongly positive.

Coma of *hypoglycaemic* origin is always of rapid onset. Convulsions often occur in the early stages, and it is distinguished by the absence of both dehydration and ketonuria. Focal neurological signs (hemiplegia) may sometimes be present and sugar is occasionally found in the urine.

Coma due to *renal failure* is characterized by dehydration and dyspnoea associated with muscular twitching, convulsions, and albumin-laden urine of low specific gravity.

In cases of coma due to *hepatic failure* ("cholaemia") there is an antecedent phase consisting of malaise and jaundice followed by drowsiness, delirium, vomiting, muscular twitching, and a tendency to haemorrhage.

Post-epileptic Coma

This is only likely to be a serious problem in the presence of status epilepticus. Focal neurological signs may or may not be present, but in the absence of an antecedent history of epilepsy there will sometimes be evidence of previous tongue-biting. Both albumin and sugar may be found in the urine

and some degree of pyrexia is usual. Occasionally coma in an epileptic subject may be due to overdosage with anti-convulsant drugs.

Exogenous Poisons

Here the diagnosis will usually depend primarily upon circumstantial evidence and the exclusion of other causes of coma. Special features to be noted are the cherry-red complexion in *carbon monoxide* poisoning; the smell of stale alcohol in the breath in *alcoholic* poisoning; the association of fits, raised blood pressure, and a "lead line" in the gums in lead *encephalopathy*; the hyperpnoea and violet urinary ferric chloride reaction in *aspirin* poisoning; and the normal or subnormal temperature and pin-point pupils in *opium* poisoning.

Intracranial Infections and Tumours

Meningitis in its various forms can sometimes be a cause of rapidly developing coma, especially the fulminating *meningococcal* variety. Diagnostic difficulties may nevertheless be considerable when the temperature is subnormal and neck rigidity is inconspicuous. A purpuric rash is commonly present in *meningococcal* infections, while in other forms there may be a focus of infection elsewhere in the body. Usually, however, the diagnosis can be established only by lumbar puncture.

In cases of *localized intracranial abscess formation* coma is likely to be a late event. Headache of increasing severity and drowsiness are outstanding premonitory symptoms, mastoiditis and intrathoracic suppuration being the commonest sources of infection. Occasionally coma supervenes rapidly if the abscess ruptures into the cerebral ventricles or subarachnoid space, in which case there will be neck rigidity associated with high temperature.

Coma is also usually a late development in cases of *intracranial tumour*, whether it be primary or secondary in origin. Occasionally it may develop rapidly when there is cystic degeneration or haemorrhage into a tumour, but differentiation from intracerebral haemorrhage is usually impossible on clinical grounds alone.

Physical Agents

Heat hyperpyrexia is a serious condition due to failure of the heat-regulating mechanism as a result of prolonged exposure to high temperatures. It is usually fatal without adequate treatment and is characterized by progressive hyperthermia, absence of sweating, convulsions, circulatory collapse, and coma.

An individual accidentally subjected to the *effects of an electric current* may be rendered comatose. Diagnosis will depend upon the detection of skin burns and a consideration of the circumstances.

Mental Disorders

Severe states of stupor occurring in hysterical, schizophrenic, or depressive illnesses may sometimes give rise to diagnostic difficulties. In cases of this kind the patient is not likely to be truly unconscious, since he may show some evidence of mental activity or betray the fact that he is not entirely unaware of his surroundings. Diagnosis will depend upon the absence of physical signs and evidence of psychological disturbance in the history obtained from friends or relatives. Organic disease must be assumed to be present, however, if the patient is genuinely comatose, even if there is a history suggestive of mental disorder.

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PROFESSIONAL BOXING AND THE DOCTOR

BY

J. W. GRAHAM, L.M.S.S.A.

Administrative Steward, British Boxing Board of Control; Chairman, Medical Subcommittee, British Boxing Board of Control; President, Medical Commission, European Boxing Union; Member of Medical Advisory Committee to World Boxing Union

In an editorial comment in the *British Medical Journal* of December 25, 1954 (p. 1535), the hope was expressed that the controlling body of professional boxing would listen to the advice of its medical advisers.

In order to show that the professional controlling body does, in fact, listen to its medical advisers, I propose to give a short account on the control of professional boxing, with special reference to the medical aspects of professional boxing.

Prior to 1929 the control of professional boxing in the British Isles and on the Continent was practically non-existent. Owing primarily to the efforts of the late Lord Lonsdale, the British Boxing Board of Control was formed in that year. Lord Lonsdale became the first president. The constitution of the Board is a president, two vice-presidents, and a number of stewards, all of whom have some specialist knowledge of boxing and are not in any way financially interested in boxing. The stewards serve in an honorary capacity and meet their own expenses. Should a vacancy occur, it is filled by invitation from the president and the stewards.

An account of the ups and downs of the Board of Control from 1929 to the present time, although interesting, would be out of place here, but a short account of the present organization is necessary.

Organization of the Board of Control

The Board now consists of a president, two vice-presidents, and up to 25 stewards, divided into stewards of appeal and administrative stewards, and has through its regulations complete control of all professional boxing in Great Britain and Northern Ireland, except fairground boxing booths. It has the authority to issue licences at its absolute discretion to all persons connected in any way with professional boxing (boxers, promoters, managers, referees, trainers, seconds, etc.). Without a current licence no one can officiate in any capacity at a professional boxing tournament.

The Board functions from head offices in London and is assisted by eight area councils covering the whole country and Northern Ireland. The area councils are made up of licensed members of various categories—promoters, boxers, boxers' managers, seconds, etc., elected annually by the licensed members in the area. Each area council has two representative stewards, who are elected annually by the council members, and who represent the views of their councils to the stewards at board meetings.

Before the second world war the medical arrangements were rather sketchy. A principal medical officer was appointed who was one of the administrative stewards. His main duty was to advise the Board on any medical matter which cropped up and to examine boxers where necessary on behalf of the Board.

During the second world war the control of boxing suffered, as most of the stewards were engaged in one or other of the Services. After the war, when the Board got into its stride again, it was quickly realized that a medical organization was a necessary part of the controlling body, and by 1950 such an organization was instituted as follows. An honorary area medical officer was appointed to each of the