death was not conclusive, so it is undesirable to base any conclusions on the figures; but experience of large numbers of these cases allows an examiner to group the probable causes of death with reasonable accuracy for registration purposes.

Cases of sudden death	404 cases
Cardiovascular group	357
Respiratory diseases	21
Nervous system diseases	8
Gastro-intestinal diseases	3
Miscellaneous causes	15

Here the outstanding feature is the proportion of deaths (85%) attributed to cardiovascular causes. This is not surprising when one considers that most of these were of people collapsing in the street or at work, or found dead in bed. In ordinary times many of these cases would have come to necropsy, but as nearly half occurred during the war years, when post-mortem examinations were reduced to a minimum, they were ascribed to the most likely cause as adjudged from the previous history and mode of death. In all, 318 deaths were thus considered to be due to coronary thrombosis and myocarditis; 11 to valvular diseases; 22 to cerebral haemorrhage; one to ruptured aneurysm, and five to pulmonary embolism.

Deaths from External Agencies (305 Cases).—These deaths were due to the following causes:

Injuries:	
Fractured skulls	101
Ruptured organs	25
Fractured limbs and spine	25
Burns	14
Exposure	2
Other causes	4
Drowning	70
Accidental carbon monoxide asphyxias	22
Associated with anaesthetics	36
Poisons	6

The first point of interest here is the comparison of the accidental deaths from inhalation of carbon monoxide—22 cases plus six that came to necropsy, 28 (16%) in all—with the 144 (84%) such cases due to suicide. The six poison cases were all due to alcohol. Nearly all of the 36 deaths associated with anaesthetics were due to the condition necessitating anaesthesia or to post-operative shock or haemorrhage—hence necropsy (especially in the war years) was not done. For example, 13 were cases of malignant disease and 7 were abdominal emergencies. Three special cases may be noted: one of tetanus which ended in death during a spasm while receiving chloroform; another that of a child of 23 months with a tuberculous lesion of the skull who received 40 ml. of a 0.5% solution of "novocain," followed by a nitrous oxide, oxygen, and ether anaesthesia, in which the local anaesthetic was certainly in excess of a safe dosage; and a third case, that of a man suffering from a deflected septum who had this treated locally with a swab of adrenaline dipped in cocaine crystals and died in convulsions within a few minutes, presumably from poisoning by cocaine owing to idiosyncrasy to the drug.

Infant Cases.—The 105 infant cases show the same tendency as the post-mortem cases: 55 were regarded as due to respiratory causes—50 to pneumonia and 5 to bronchitis—21 were asphyxial deaths, mostly accidental overlaying; 13 were due to convulsions, nine to prematurity, and five to acute infections—measles (three) and whooping-cough (two).

Conclusion

Exact inferences may not be drawn from the figures given, but there seem to be three points which deserve some emphasis: (1) the outstanding predominance of cases of cardiovascular degeneration as a cause of sudden death—461 (76%) out of 604 cases; (2) the frequency of coal-gas as a suicidal agent; and (3) the frequency of pneumonia (50%) as a cause of sudden death in young infants.

HEMIPLEGIA IN YOUNG ADULTS

BY

G. JOLY DIXON, M.A., M.D., M.R.C.P.

Strokes in the elderly are usually regarded as a manifestation of cerebral vascular disease, while in the young adult they are usually thought to be due to some extraneous cause. The purpose of this paper is to emphasize both the obscurity of aetiology and the frequency of strokes in young adults.

The cases selected for study are all instances of unilateral pyramidal palsy seen in Service personnel between the ages of 20 and 40 at the E.M.S. Neurological Centre, Winwick, between 1940 and 1946, and also four civilians of the same age group admitted to the Warrington General Hospital between 1944 and 1946; in all 35. Nineteen of these patients had only minor degrees of hemiparesis, and in these a convincing cause was found. Thus, seven occurred among 30 consecutive cases of cerebral tumour, six among 30 consecutive cases of disseminated sclerosis, three cases of congenital hemiparesis had been accepted by the recruitment board, and two cases of hysteria and a hemiparesis due to syringomyelia completed the total. It so happened that the onset of the palsy in all these 19 patients was insidious.

The remaining 16 patients with more severe palsies all had a sudden stroke; of these, two men suffered cerebral emboli during the course of rheumatic mitral stenosis with pulmonary infarcts (Case 20) and bacterial endocarditis (Case 21). Another in retrospect presumably had suffered a Herxheimer reaction during the treatment of secondary syphilis (Case 22); a fourth must have had a cerebral aneurysm, as his stroke occurred 10 days after a sub-arachnoid haemorrhage; and a woman (Case 24) aged 29 developed a hemiplegia whenever an overdose of insulin was administered in treatment of her diabetes mellitus. A case similar to this last was described by Fischer and Florman (1943), though their patient eventually suffered from a cerebral embolus. It is of interest that Case 21 eventually recovered from the endocarditis after treatment with penicillin, though he was left with an almost useless left hand and spastic leg and auricular fibrillation; while Case 22 recovered sufficiently to be conscripted three years after his stroke.

Eleven of the severely palsied cases were not so readily diagnosed. Of these, two convalescent Service men (Cases 25 and 26) who had no pyramidal signs were at first labelled hysteria; however, they both claimed unilateral loss of sense of position and a field defect on the same side. An almost identical Service case was described by Stewart, Randall, and Riesenman (1943) and diagnosed by them as hysteria. However, in reconsidering these three patients it appeared remarkable that all of them should have mimicked so closely the symptomatology of a vascular lesion in the posterior portion of the internal capsule, and when Case 25 was re-examined two years later the signs were unaltered, except that he had developed choreiform athetotic movements of the affected shoulder and a pneumo-encephalogram demonstrated localized cortical atrophy. Six months after his stroke Case 26 wrote that his condition was stationary. It seems possible that had the frequency of strokes in young adults been appreciated at the time both Cases 25 and 26 would have been differently labelled.

Three of the cases under review had sepsis in the jugular vascular bed on the same side as the brain was affected—namely, in Case 27 a tuberculous gland on the right side of the neck, in Case 28 a Vincent's ulcer on the left tonsil, and in Case 29 a right-sided chronic otitis media. Dowman

(1926) and Purdon Martin (1944) have described strokes in this age group due to cerebral venous thrombosis, and Symonds (1937, 1940) has stressed the importance of sepsis in the middle ear and nasopharynx as a cause of thrombophlebitis of the Rolandic veins. Another soldier (Case 30) gave a history of an unexplained femoral phlebitis six years previously, and at the time of his stroke he was under investigation for an obscure painful lesion in the left lung.

Elkington (1935) described the cases of three young adults whom he concluded to be suffering from cerebral haemorrhage because of the site of the lesion, the suddenness of the onset, and the finding of slightly xanthochromic fluid on lumbar puncture in two who had no other signs of subarachnoid haemorrhage. He compared them with a woman of 27 who died from a pontocerebellar haemorrhage resulting from telangiectasis in this region and another woman, described by Gordon Holmes (1931), who developed a left homonymous hemianopia from an arteriovenous aneurysm in the calcarine fissure. The cases here examined resembled Elkington's in the sudden onset and the site of the lesion, and resembled other vascular accidents in the fact that their condition has changed very little during the period of one to four years they have been under observation. However, abnormality of cerebrospinal fluid was a rarity. Case 25 had a slight increase in globulin and a gold curve of 1221110000, and Case 31 had seven lymphocytes per c.mm. in the cerebrospinal fluid.

It seems probable that Case 31 had a cerebral haemorrhage, as at the time of his stroke he suffered a transient glycosuria and albuminuria. This patient, a soldier aged 30, is of interest as two months previously he had suffered from infective hepatitis. Lescher (1944) and Stokes, Owen, and Holmes (1945) describe four cases of cerebral haemorrhage complicating this disease. In Lescher's case the same time interval occurred as in Case 31. The neurological signs in Case 31 were consistent with a lesion in the right internal capsule, but this lesion was presumably more extensive, as he developed epileptiform convulsions and a pneumo-encephalogram seven months after the stroke showed extensive atrophy of the right cerebral cortex. Stokes *et al.* in describing the necropsy of their Case A state: "The brain contained an area of softening lateral to the right lateral ventricle and a large haemorrhage into the right caudate nucleus." Had this patient survived he would presumably have presented an almost identical clinical picture to Case 31, except for the site of the palsy.

No other patient showed evidence that the vascular atrophy was due to haemorrhage. One patient at least, a housewife aged 26, presumably had a cerebral embolus, for on admission she was found to have a cloudy wedge-shaped area lying on either side of a pair of vessels in the upper temporal sector of the left retina. These vessels were somewhat obscured near the disk; peripherally, the artery was seen to be empty and the vein normal. It is suggested that this woman's sudden loss of consciousness the day before admission had resulted from an embolus which had broken up in the left internal carotid artery. The smaller part lodged in the retina and the larger part occluded branches of the left middle cerebral artery. The patient had prolonged unconsciousness, which after about ten days gave place to complete aphasia, right-sided spastic paralysis of face, arm, and leg, and loss of sense of position in these limbs. Later, absence of the nasal field was demonstrated in the right eye, presumably due to the local lesion in the retina.

None of these nine obscure cases had any abnormality of the urine, and the cardiovascular systems appeared healthy except that in three Service men when stress was shown during examination a high blood pressure was found

(150/100 in Case 25, 150/100 in Case 26, and 140/100 in Case 31), normal pressures being recorded at other times in these patients. Another associated finding was recent evidence of pulmonary disease; thus, Case 25 was at the time of his stroke convalescent from pneumonia, and Case 30 during the month preceding his stroke had had three attacks of left-sided pleurisy and was found to have x-ray evidence of infiltration of the left mid-lung zone. A patient not previously mentioned was found to have radiological evidence of spreading though symptomless pulmonary tuberculosis, and another stated that two years previously he had had pneumonia. French authors, such as Hartenberg (1930), Trotot (1935), and Vidal (1942), stress the occurrence of hemiplegias during artificial pneumothorax refills, but the transient nature of these catastrophes argues a different aetiology from that in the group under review.

Summary and Conclusion

It would seem that strokes resulting in permanent hemiplegias are not such a very uncommon accident in young adult life. Although many of these patients show evidence of cardiac disease likely to give rise to emboli, the majority have no such condition. It would appear that this phenomenon may be due to either a venous thrombosis in the Rolandic system of veins or a haemorrhage, thrombosis, or embolus in the cerebro-arterial system. Sepsis in the jugular vascular bed, recent pulmonary disease, and a tendency to abnormal elevation of the blood pressure are frequent associated findings.

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The first number of *Nutrition, Dietetics, Catering* is an attractive illustrated journal, to appear quarterly, issued by the British Dietetic Association and the Food Education Society (annual subscription 10s.: Newman Books, Ltd., 356-366, Oxford Street, London, W.1). It is intended to provide the latest news on dietetics for catering managers, canteen supervisors, and others responsible for the preparation of food, as well as medical men, and Sir Jack Drummond hopes, in a foreword, that it will contribute to banishing sodden cabbage and the other evils of bad cooking from our tables. As he points out, the standard of cooking in many of our hotels, hospitals, and institutions is deplorably low and we should be more ashamed of it than we are. Prof. Marrack draws attention to the fact that foods may have a nutritive value not apparent from the content of their various nutrients, and comments that the moral of recent investigations is that "animals, human or other, need food—not vitamins or other separate nutrients." An interesting observation made by Prof. Yudkin is that adolescence in the West African is delayed longer than is commonly believed. The psychological changes associated with puberty in girls occur at the age of about 16 or 17, and he thinks it likely that unsatisfactory nutrition is a possible cause. Mrs. Palmström, comparing Norwegian food with English, remarks that though it is similar it is often prepared differently and is eaten at different times of the day. There is no sign of war weariness in Norway. The people are strong and healthy—the women in fact probably working harder than they do in England. She points out that there are many factors contributing to this state of affairs, and wonders if the diet plays any part, in particular the relatively high consumption of meat and fish in conjunction with bread. The journal also includes news items, book reviews, and schemes for training in dietetics.