serotonin and acted as antimetabolites. The subsequent discovery of a variety of psychosis-inducing chemical agents, each of which was clearly linked to serotonin, has added support to this hypothesis. Some of these agents have been found in plant sources and others have been synthesized. Excitement may be caused by drugs having a serotonin-like action: normal men have been made to develop a transient condition resembling schizophrenia and the increase in serotonin content of the brains of a few schizophrenic subjects induced by administration of the serotonin precursor, 5-hydroxytryptophan, has resulted in a worsening of their condition. (Woolley, 1958). Such findings are compatible with the hypothesis that an excess of serotonin in the brain leads to hallucinations and schizophrenic manifestations.

That depression can result from a deficiency of serotonin in the brain is supported by much clinical work which has been done with drugs which inhibit mono-amine oxidase, the enzyme chiefly responsible for destroying serotonin. These inhibitors increase brain serotonin and relieve simple depressions, and have usually been found to worsen the condition of schizophrenia.

When serotonin is injected it is rapidly destroyed by specific enzymes and no increase in its concentration in the brain can be demonstrated unless a mono-amine oxidase inhibitor is given simultaneously (Costa and Aprison, 1958). The location of serotonin-destroying enzymes in the walls of cerebral capillaries may therefore act as a blood-brain barrier (Woolley, 1958). Presumably this is why patients with the carcinoid syndrome do not develop mental changes. This barrier was possibly defective in this patient, and the carcinoid tumour was responsible for the psychosis. The fact that she started drinking heavily as a result of the psychosis, and that apart from fatty degeneration of the liver there were no other manifestations of chronic

alcoholism, is evidence against the latter as a cause.

There were no signs of a deficiency of nicotinic acid which sometimes accounts for mental changes. There were no major cerebral arteriosclerotic changes. In a manic-depressive state, intelligence and memory are usually unimpaired; only when manic excitement is severe are clouding of consciousness and confusion observed. Generally speaking, depression and mania occur in a setting of clear consciousness. This serves to differentiate affective disorders from similar psychoses. (Mayer-Gross, 1960).

Summary

A case of carcinoid tumour is reported that presented with psychosis fourteen years before diagnosis at necropsy. This is the first case to be recorded with such an association.

REFERENCES

- BIORCK, G., AXEN, O., and THORSON, A. (1952): Unusual Cyanosis in a Boy with Congenital Pulmonary Stenosis and Tricuspid Valve Insufficiency, Fatal Outcome, *Amer. Heart J.*, **44**, 143.
- COSTA, E., and APRISON, M. H. (1958): Distribution of Intracarotidly Injected Serotonin in the Brain, *Amer. J. Physiol.*, **192**, 95.
- HOSSAIN, M. A. (1962): Carcinoid Syndrome with Unusual Features, *Postgrad. med. J.*, **38**, 204.
- MAYER-GROSS (1960): Clinical Psychiatry, 2nd Edition, London: Cassell.
- THORSON, A., BIORCK, A., BJORKMAN, G., and WALDENSTROM, J. (1954): Malignant Carcinoid of the Small Intestine with Metastases to the Liver, Valvular Disease of the Right Side of the Heart, Peripheral Vasomotor Symptoms, Broncho Constriction, and an Unusual Type of Cyanosis: A Clinical and Pathological Syndrome, *Amer. Heart J.*, **47**, 795.
- WOOLLEY, D. W., and SHAW, E. (1954): Some Neurophysiological Aspects of Serotonin, *Brit. med. J.*, **ii**, 122.
- WOOLLEY, D. W. (1958): Serotonin in Mental Disorders, *Ass. Res. nerv. Dis. Proc. Res. Pub.*, **36**, 381.

AKINETIC MUTISM FOLLOWING COAL-GAS POISONING WITH SUBSEQUENT RECOVERY

T. F. MACKINTOSH

Poisons Treatment Centre, The Royal Infirmary, Edinburgh, 3.

THERE ARE many sequelae to carbon monoxide poisoning and one of the most unusual is the development of akinetic mutism; the patient to be described developed this state after a suicidal attempt by gassing. She eventually recovered spontaneous speech and movement and only then did she show evidence of an upper motor neurone lesion.

Akinetic mutism is rarely encountered in general medical practice and most patients in whom this state occurs are suffering from severe disease from which recovery does not take place. However, occasionally it can occur in a patient whose illness is in itself not fatal; the patient to be described falls into this category. Recovery from akinetic mutism has rarely been reported and

this case illustrates that it can occur. It demonstrates the importance of reaching the correct diagnosis and the value and interest of the ancillary investigations.

Akinetic mutism is not a diagnosis; it is a state of altered consciousness, a step on the ladder from full consciousness to coma and death. It rarely persists for any great length of time but short periods of akinetic mutism may be more common and go unrecognised.

Consciousness is best defined as "awareness of environment and self" (Cairns, 1952), and according to Cravioto, Silberman and Feigin (1960) patients with akinetic mutism are probably not aware of their surroundings. A functioning cortex is necessary for "full" consciousness to be evident (French, 1952; Jefferson, 1959; Williams and Parsons-Smith, 1951) and this probably depends on the synthesis of impressions from the outside world with those from the interior of the body and their passage through the reticular formation which acts as an arousal centre. (Cairns, 1952; Ranson, 1959; Williams and Parsons-Smith, 1951).

Cairns, Oldfield, Pennybacker and Whitteridge (1941) provided the classic description of akinetic mutism:

"The state was described by the child's parents and nurses as one of drowsiness but drowsiness was not a feature of it. The most profound manifestations were mutism, loss of feeling tone, loss of emotional expression, of spontaneous and most other voluntary movements, and total incontinence of urine and faeces of a degree and persistence rarely met with in brain lesions not associated with coma. She was incapable of originating active manifestations of any kind with the notable exception that ocular fixation and movements occurred in response to movements of external objects and to sounds. It was this that gave the child the appearance of alertness so incongruous with her silence and general immobility. All feeling appeared to be in abeyance. To painful stimuli there was reflex withdrawal but never any signs or sound of displeasure. Pleasant and unpleasant substances were all swallowed promptly without any expression of pleasure, distaste or discomfort."

This description is strikingly similar to that for depressive stupor by Kraepelin (1921) and emphasises the difficulty in the differential diagnosis.

"In the most severe stuporous forms every volitional expression of the patient may be arrested so that he is only able to lie still and can scarcely open his eyes. He is unable to show his tongue, to take meals, to give his hand or even to leave his bed to relieve nature. Although he perhaps understands quite well what he is told to do, yet at most a few weak, trembling attempts at the required movements follow. The patient retains uncomfortable attitudes because it is not possible for him to change his position; all objects which are placed in his hand one after the other he spasmodically tries to hold as he is incapable of letting them go . . . Volitional utterances are extremely scanty. As a rule the patients lie mute in bed, give no answer of any sort, at most withdraw themselves timidly from approaches but often do not defend themselves

from pin-pricks. Sometimes they display catalepsy and lack of will-power, sometimes aimless resistance to external interference. They sit helpless before their food; perhaps, however, they let themselves be spoon-fed without making any difficulty. They hold fast what is pressed into their hands, turn it slowly about without knowing how to get rid of it. They are, therefore, wholly unable to care for their bodily needs and not infrequently they become dirty. After the return of consciousness which usually appears rather abruptly, memory is very much clouded and often quite extinguished."

The case described by Cairns and others (1941) was of a patient with an epidermoid cyst of the third ventricle; her level of consciousness varied with the size of the cyst; following aspiration the patient was normal, but as the cyst increased in size her level of consciousness fell until she was in the state of akinetic mutism with bilateral pyramidal tract signs. This abnormal clinical state had been noticed prior to Cairns' description but no special name had been given to it (Traut, 1935). The largest series of cases is that of Cravioto and others (1960). Seven of their eight cases developed akinetic mutism, following cerebral vascular accidents and one followed coal-gas poisoning. All these patients died.

Akinetic mutism has been reported when various areas of the brain have been damaged and with varying pathologies, such as craniopharyngioma, pineal tumours, thalamic haemorrhages, posterior fossa tumours, with bilateral cingulate gyrus destruction and following ischaemic brain-stem lesions. The commonest areas of destruction have been in the brain-stem with involvement of the reticular formation. (Cairns and others, 1941; Cravioto and others, 1960; Williams and Parsons-Smith, 1951). Only two cases following coal-gas poisoning have been described and both were fatal. However, Denny-Brown states that "in many cases of coal-gas poisoning an akinetic mutism state remains following initial coma" and he apparently considers that all such cases are fatal (Denny-Brown, 1962).

In carbon monoxide poisoning the pathological lesions in the brain may be widespread but they characteristically occur in certain areas; the basal ganglia, the reticular zone of the substantia nigra, the deeper layers of the cerebral cortex and the Purkinje cells of the cerebellum (Greenfield, 1963; Bokojnic, 1963). In a severe case of poisoning probably most of these areas will be affected in varying degrees.

Case Report

Mrs. H.M. is a 42-year-old white South African who came to this country in August 1963. She has been in good physical health previously but on several occasions she had required treatment for attacks of depression and in May 1963 she received a course of electroconvulsive therapy with good effect. Shortly after arrival in the United Kingdom she again became depressed and she was treated by her family doctor. Under his care she apparently recovered and was able to resume her job as a packer in a biscuit factory.

Two days prior to admission she again appeared

depressed with some paranoid features. On the 28th January, 1964 (day 1) her husband left for his work at 7.30 a.m. at which time she appeared "perfectly well." When he returned home at 5.30 p.m. he found her unconscious in a gas-filled room. The time at which she had turned the gas on and the duration of unconsciousness are unknown.

She was admitted to the Poisoning Treatment Centre at the Royal Infirmary.

On Examination she was unconscious with no response to painful stimuli. There was no neck stiffness, the pulse rate was 108 per minute, systolic blood pressure 95 mm. Hg., and there was a cherry red hue to the skin and mucous membranes. Pupils were equal, of normal size and reacted to light. Optic fundi were normal. The tone in the limbs was increased, tendon reflexes were present and equal and the plantar responses were flexor. No lateralising neurological signs were found. Treatment with $\text{CO}_2\text{-O}_2$ mixture was given with little effect. For four days there was no change in the neurological signs or level of consciousness. A lumbar puncture was performed on the third day but no biochemical or serological abnormalities were found.

Progress. On the fifth day after admission her level of consciousness had lightened, she still responded only minimally to painful stimuli and made no response whatsoever to spoken or shouted command, but on occasions she would open her eyes and follow other patient's movements around the ward, but no consistent response was obtained to visual stimulation by the examining physicians. She would occasionally blink in response to a visual threat but would not follow moving objects presented to her or show any signs of recognition of her husband or the nurses. The visual fields appeared full to threat but could not be otherwise determined. The eye movements appeared normal and no ocular palsies were noted. Although she would not look at objects presented to her she could move her eyes voluntarily and the nurses frequently observed that she was watching them as they performed their tasks in the ward. Apart from the infrequent voluntary eye movements no other spontaneous activity of any sort was observed. For most of the day she lay in whatever position she had been placed with her eyes shut. There were no lateralising neurological signs and the tone in the limbs had become normal. She was incontinent of both urine and faeces. She expressed no emotion, did not speak and showed no reaction to unpleasant stimuli, such as the passing of a gastric tube. In attempt to differentiate akinetic mutism and depressive stupor intravenous sodium amytal was given but she showed no response to questioning or commands under the influence of the barbiturate and there was no change in the level of "awareness."

On the eighth day an electroencephalogram was recorded and Dr. J. Laidlaw reported that it showed an irregular background activity with a marked polyrhythmia within the theta range, with waves of amplitude of 100 microvolts, indicating a moderately severe generalised abnormality. A further EEG (Fig. 1) on the eleventh day (when there has been little or no change in the patient's clinical condition) differed considerably from the previous one, first the background activity was more rhythmic, reaching an amplitude of 70-80 microvolts and was more plentiful. Secondly, the report was interrupted by a number of paroxysms lasting some five to eight seconds of high amplitude slow waves (up to 120 microvolts, period up to 0.5 seconds), which were of

greatest amplitude anteriorly. This record indicated a severe generalised abnormality and the runs of high amplitude slow waves suggested diencephalic dysfunction. However, the presence of such a well-developed, though abnormal background activity was thought to show a possible improvement on the previous record.

Over the next six weeks she made very slow, spontaneous improvement although her general progress was retarded by the development of infections of the urinary tract, right middle ear and buttock. These infections responded to antibiotic therapy and other measures. Her level of consciousness remained unaltered but she was "awake" for longer periods and her eyes followed the activities going on in the ward at these times. She remained absolutely passive with no spontaneous movements, speech or emotional response to stimuli. Two weeks after admission she began to take liquids by mouth but without any signs of preference or pleasure and a week later she began to open and close her eyes when subjected to painful stimuli. Electroencephalographs recorded on the 16th and 26th days showed no significant improvement. Pneumoencephalography was performed eight weeks after admission and showed marked pooling of the air over the left fronto-parietal cortex and the right temporal region. There was also a diverticulum of the left lateral ventricle. (Fig. 3-5).

Subsequently the patient began to improve more rapidly and as her level of consciousness lightened she began to move her left arm a little and the power of speech started to return. Her limbs were now spastic. Nine-and-a-half weeks after admission spontaneous movement was confined to the extra-ocular muscles but occasionally when commanded she would put out her tongue, open her mouth and shut her eyes. Spontaneous speech was absent but if subjected to pain she would speak; she had developed slight contractures of both elbows and straightening these joints caused sufficient pain for her to cry, "It is sore," and when asked where it was sore, to say, "In the joint." Her speech returned over the next month in such a way that almost from the first she would give sensible and appropriate answers to questions although initially the stimuli required to get the answers needed to be strong. There was no evidence of dysphasia at any time.

An electroencephalograph at this time showed persistence of the large, slow waves with evidence of a generalised disturbance of cerebral function suggesting involvements of the deep central structures.

The return of speech was accompanied by the return of emotional expression and up to the time of discharge she remained emotionally labile. The ability to move her limbs returned over the three months; the initial slight voluntary movements were accompanied by a very coarse tremor which was not present at rest. Tone was markedly increased in all limbs, tendon reflexes were increased symmetrically and an extensor plantar response was present on the right side for the first time. Sensation could not be tested accurately but pain was obviously felt. No abnormality of the cranial nerves was detected. Return of motor function on the left side was always in advance of that on the right and after three months progress was rapid. By the time of discharge fourteen weeks after admission the patient was able to walk well with the aid of sticks, tremor was minimal, tone was just greater than normal, the plantar responses were flexor and there were no cranial or sensory deficits. An electroencephalograph was

FIG. 1.—4th February, 1964. E.E.G. taken whilst the patient was in the akinetic mute state. Showing marked polyrhythmia with irregular background activity.

Guide to positions of leads of E.E.G.

completely normal. (Fig. 2). She was able to carry on a normal conversation although it became apparent that there were defects in her memory, for instance, she did not know the Prime Minister's name but once she had been told it she readily recalled it the next day.

We have no previous record of her intellectual level but her account of her school and work record and interest patterns suggest that her intellectual level prior to admission was only low average, i.e., better than only 25% of people of her age. Intelligence tests were performed twelve weeks after admission. (Mr. McPherson). These showed that her verbal intelligence was not impaired. However, her non-verbal intellectual test score was better than only 10% of people of her age. She also showed a severe defect in the learning phase of memory affecting both verbal and non-verbal learning tasks; some learning did occur but it was very much slower and less efficient than normal. Computation and tests of spatial ability were also impaired. These results are characteristic of patients with cerebral damage.

FIG. 2.—4th May, 1964. Prior to discharge. Normal E.E.G.

Discussion

The differentiation between depressive stupor and akinetic mutism is not easy when a patient is in this state in which she receives and apparently partially understands some information but is incapable of making any response. The importance of arriving at the correct diagnosis lies in the fact that electroconvulsive therapy is frequently prescribed for depressive stupor and the accompanying cerebral insult can aggravate underlying cerebral damage. As can be seen from the descriptions given of depressive stupor and akinetic mutism the clinical picture is very much the same in both states and as methods of resuscitation improve the problem of differentiating them will become more common. Most authorities are agreed that the principle distinguishing features in the absence of neurological signs are:—

1. Incontinence is very rare in depressive stupor and is never of the degree of severity found in akinetic mutism.
2. In akinetic mutism spontaneous movement is limited to the eyes whereas patients in depressive stupor are generally capable of initiating some other activity.

3. Sometimes in depressive stupor a short remission can be induced with intravenous sodium amytal.

At first, depressive stupor was suspected in this patient in view of the previous psychiatric history, the lack of positive neurological signs and the appearance of the patient. However, the diagnosis was discarded for various reasons: patients who are in depressive stupor usually have a history of sliding into their depression but this patient had not (any depressive precipitant must have been acute), the patient was incontinent for a time, and there was no response to an intravenous sodium amytal test. The most important factors which led us to the view that this patient suffered from akinetic mutism, however, were the grossly abnormal electroencephalograph and pneumoencephalograph.

The electroencephalograph in akinetic mutism has not been investigated thoroughly but most reported cases appear to conform with our patient in that there is an abnormal electroencephalograph with the production of generalised high amplitude slow waves (Cairns and others, 1941; Cairns, 1952; Walter, Griffiths and Nevin, 1939; Williams and Parsons-Smith, 1951). The

FIG. 3.

FIG. 5.

electroencephalograph is of great help in differentiating between an organic and functional state, and in this case the finding of generalised, synchronous, symmetrical, slow waves, indicated organic damage to the deep central structures.

The differential diagnosis is thus difficult but very important. To illustrate this point the only case of akinetic mutism from coal-gas poisoning in Cravioto's series had received several electroconvulsive treatments, and at post mortem bilateral lesions were found in the globus pallidus with cystic destruction of the right middle frontal gyrus and with neuronal loss in cortical regions. This may have been due to the coal-gas itself, but the electroconvulsive therapy may have aggravated any lesions that were present.

FIG. 4.

FIGS. 3-5.—Air encephalograms 8 weeks after admission.

Due to the widespread brain damage in our patient one cannot accurately localise the area of brain involved which caused the akinetic mutism state. It is interesting to note that the pyramidal tract signs were not evident for some time. The explanation in physiological terms of this masking effect is difficult; probably the cells of the cerebral cortex were damaged so that only on their recovery did positive pyramidal tract signs appear.

The importance of a high degree of nursing care, the prevention and treatment of infection and adequate physiotherapy to the limbs, cannot be stressed too highly and are part of a therapy of hope in the realisation that a fatal outcome is not inevitable. These patients should not be left with permanent orthopaedic and other problems in addition to their residual neurological damage.

Summary

A case report is presented of akinetic mutism following coal-gas poisoning. Recovery took place. Spastic hemiparesis was only manifest after the akinetic mutism disappeared. The differential diagnosis of akinetic mutism from depressive stupor is discussed. The reason for the later appearance of signs of upper motor neurone damage is discussed.

I wish to thank Dr. Henry Matthew for permission to study patients under his care and both Dr. Matthew and Dr. Neil Kessel for their help and criticism in the preparation of this paper. Dr. John Laidlaw kindly performed and interpreted the E.E.Gs.

REFERENCES

- BOKOJNIC, N. (1963): Stagnant Anoxia and Carbon Monoxide Poisoning, *Electroenceph. clin. Neurophysiol. Suppl.*
- CAIRNS, H. (1952): Disturbances of Consciousness with Lesions of the Brain-Stem and Diencephalon, *Brain*, **75**, 109.
- CAIRNS, H., OLDFIELD, R., PENNYBACKER, J. B. and WHITTERIDGE, D. (1941): Akinetic Mutism.

- with an Epidermoid Cyst of the 3rd Ventricle, *Brain*, **61**, 273.
- CRAVIOTO, H., SILBERMAN, J., and FEIGIN, I. (1960): A Clinical and Pathologic Study of Akinetic Mutism, *Neurology*, **10**, 10.
- DENNY BROWN, D. (1962): *The Basal Ganglia*, p. 130, London: Oxford University Press.
- FRENCH, J. D. (1952): Brain Lesions Associated with Prolonged Unconsciousness, *Arch. Neurol. Psychiat. (Chic.)*, **68**, 727.
- GREENFIELD, J. G. (1963): *Greenfield's Neuropathology*, 2nd Ed., London: Edward Arnold.
- JEFFERSON, G. (1960): *The Selected Papers of Sir Geoffrey Jefferson*, p. 526, London: Pitman Medical.

- KRAEPELIN, E. (1921): *Manic Depressive Insanity and Paranoia*, Trans. by R. M. Barclay, p. 80, Edinburgh and London: E. & S. Livingstone.
- RANSON, S. W. (1939): Somnolence caused by Hypothalamic Lesions in the Monkey, *Arch. Neurol. Psychiat. (Chic.)*, **41**, 1.
- TRAUT, E. F. (1935): The Case of Patricia Maguire, *J. Amer. med. Ass.*, **104**(2), 1210.
- WALTER, W. G., GRIFFITHS, G. M., and NEVIN, S. (1939): The Electro-encephalogram in a Case of Pathological Sleep due to Hypothalamic Tumour, *Brit. med. J.*, **i**, 107.
- WILLIAMS, D., and PARSONS-SMITH, G. (1951): Thalamic Activity in Stupor, *Brain*, **74**, 377.

LINER DYSFUNCTION WITH JAUNDICE IN A CASE OF SEVERE MALNUTRITION AND MALABSORPTION

K. W. HEATON, M.B. Cantab., M.R.C.P.

Medical Registrar, Royal Free Hospital, North Western Branch, London, N.W.3.*

ALTHOUGH malnutrition is accepted as a cause of liver disease in experimental animals, its role in the genesis of liver disease in man is controversial (Fernando, Medonza and Rajasuriya, 1948; Patwardhan, 1955; Higginson, Grobelaar and Walker, 1957). Protein malnutrition in children (kwashiorkor) is often associated with fatty liver, but when hepatocellular failure and jaundice occur they are usually blamed on coincidental viral hepatitis. In adults overt liver disease has seldom been convincingly attributed to malnutrition. For this reason the case is reported here of a middle-aged woman who developed jaundice and other evidence of liver-cell dysfunction in addition to fatty liver, while suffering from severe malnutrition and malabsorption.

Case Report

Miss L.M. was diagnosed in 1941, at the age of 19, as having Crohn's disease (regional enteritis). This was established at laparotomy after a two-year history of intermittent abdominal pain, vomiting and diarrhoea. Extensive bowel resection was carried out then, and by 1957 five further resections had been done for recurrences of the disease. These left her with an estimated four feet of small intestine. Steatorrhoea became apparent in 1957 and soon afterwards other symptoms of malabsorption occurred such as glossitis, oedema, paraesthesiae and a bleeding tendency. Replacement therapy was only taken spasmodically. During the first six months of 1963 her condition gradually worsened with severe anorexia, continuous diarrhoea and loss of 3 stone in weight. Shortly before admission to the Royal Free Hospital on the 19th June 1963 she began to have painful cramps of her hands and feet.

Examination at this time revealed a pale, ill and wasted woman of 41 years with a pyrexia of 99-100°F. She was faintly jaundiced and vomiting occasionally. Her stools were frequent, pale and watery, with no visible blood. Chvostek's and

Trousseau's signs were positive and at times spontaneous tetany occurred with typical carpo-pedal spasm. Pulse 120/min., regular. BP 110/65 mm. Hg. Abdomen distended, with numerous laparotomy scars, tenderness on the right side and an ill-defined mass below the right costal margin.

Investigations. Hb 11.0 g.%, film—moderate hypochromia. WBC 4,500/cu. mm. (normal differential). Prothrombin time 18 seconds (control 14 seconds). Blood urea 13 mg./100 ml. Serum potassium 1.4, sodium 140, chloride 91, bicarbonate 30 mEq/l., calcium 5.5 mg./100 ml. Plasma bilirubin 3.6 mg./100 ml., alkaline phosphatase 13 K.A. units/100 ml. Albumin 2.3, globulin 2.4 g./100 ml. ECG—generalised flattening or inversion of T waves, Q-T interval prolonged (Q-Tc = 0.65 secs.).

Progress. During the following two weeks jaundice deepened (serum bilirubin 6.5 mg./100 ml.) and features of liver-cell dysfunction appeared. These included drowsiness and euphoria, ascites, raised serum enzymes (aspartate transaminase 122 i.u./l., isocitrate dehydrogenase 11.2 i.u./l.), raised serum amino-acids (9.1 mg./100 ml.) lowered blood urea (9 mg./100 ml.) and abnormal thymol and zinc sulphate turbidities. Profuse rectal bleeding also occurred, an estimated six pints being lost and replaced. Treatment with a high-carbohydrate, protein-free diet, oral neomycin and parenteral B vitamins was ineffective, but marked improvement occurred when high-dosage corticosteroid therapy was instituted (prednisone 30 mg. 8 hourly). After a further six weeks all routine liver function tests were normal and bromsulphthalein retention was only 8% at 45 minutes. Needle liver biopsy however showed extreme fatty infiltration (also mild inflammatory cell infiltration of the portal tracts and slight periportal fibrosis not amounting to nodular cirrhosis).

Discussion

This patient presented with severe malnutrition and an exceptional degree of electrolyte depletion and was found to have increasing jaundice and evidence of liver cell dysfunction amounting almost to hepatic precoma. No cause could be found for her hepatocellular failure apart from

* Present address: Department of Medicine, Royal Infirmary, Bristol, 2.