Summary

We are now very near to being able to predict from tests *in vitro* whether a given sample of red cells will undergo rapid destruction if transfused to a particular patient and, if so, how rapid destruction will be, whether it will involve all or only some of the transfused red cells, whether it will be predominantly intravascular or extravascular, and, if extravascular, whether it will occur predominantly in the liver or the spleen.

Further observations, particularly in patients whose serum contains "cold" specific iso-agglutinins, are needed to define still more clearly the relationship between the thermal range of an antibody *in vitro* and its power to bring about red-cell destruction *in vivo*. The more academic problem of the relatively slow destruction, in particular subjects, of transfused red cells in the absence of demonstrable antibodies still awaits solution.

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CASE OF CARBON-MONOXIDE POISONING WITH COMPLICATIONS SUCCESSFUL TREATMENT WITH AN ARTIFICIAL KIDNEY

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The inhalation of carbon monoxide is a common method of attempted suicide, but nerve damage and muscle necrosis with acute renal failure are a rare complication. Reports have been published of mental and neurological sequelae from carbon-monoxide poisoning; among records of cases that survive the usual fatal long exposure to the gas (Shillito *et al.*, 1936; Adams *et al.*, 1953) we have been unable to find any mention of myonecrosis and wasting due to neuropathy occurring in the same patient.

Table I gives a summary of the cases with muscle necrosis and renal failure we have traced in the literature.

The following case is published in view of its rarity and because, in spite of many serious hazards, treatment with an artificial kidney (Kolff twin-coil) was successful. As a result of the carbon-monoxide poisoning the patient had muscle necrosis, acute renal failure, mental changes, a radiculomyelitis of L 2 to 5, and probably myocardial damage.

TABLE I.—*Muscle Necrosis and Renal Failure*

Authors	Cases	Causes	Renal Failure	Results
Günther (1921)	1	Carbon monoxide	Yes	Died
Mautner (1955)	1	" "	Oliguria 4 days. Blood urea up to 180 mg./100 ml.	Recovered
Fahlgren <i>et al.</i> (1957)	5	Alcoholism and barbiturates	Yes (2 cases)	Both died (one on way for treatment by artificial kidney)
Loughridge <i>et al.</i> (1958)	1	Carbon monoxide	Yes	Died (just prior to haemodialysis)

Case Report

A married woman aged 35 attempted suicide by inhalation of carbon monoxide in the early hours of September 19, 1958. She sealed herself in her kitchen, turned on all the gas-cooker taps, and sat on a chair with her head resting on her arms on a table. When found by her husband at 7.30 a.m. on the 19th exposure was estimated to have lasted 6½ hours. It took three hours for her to regain consciousness. When she was found her position on the chair was as shown in Fig. 1. She complained of severe pain in her back and lower limbs on recovering consciousness and was continually crying out.

On September 20 (2nd day) her doctor referred her to the local hospital for x-ray examination of her spine on account of persistent severe back pain. As the findings were doubtful she was admitted the same day to the Wingfield-Morris Hospital, Oxford, with a suspected spinal fracture. The next day she passed no urine. X-ray examination of her spine showed it to be normal.

On September 22 (4th day) she was transferred to the Radcliffe Infirmary, where her urinary output was only about 120 ml. in 24 hours and the blood urea 200 mg./100 ml. No myoglobinuria was found. Muscle necrosis was suspected because of the continued back pain and tenderness.

She was transferred to the renal unit at Halton on the 23rd.

On admission she was fully conscious, and was groaning and shouting because of headaches and pain, with extreme tenderness in the muscles of her back and lower limbs. She was very emotional. Examination revealed acute tenderness of all muscles, particularly over the lower thorax, thighs, and calves. There was a swelling (5 by 4 in.: 12.5 by 10 cm.) of the right paravertebral muscles over the line

FIG. 1.—Position of patient on chair when found. Note pressure line across lower thorax posteriorly.

of the 10th rib. This was exceptionally tender, there being superficial pitting oedema. Reflexes were absent in both knees, diminished in the ankles, and equivocal in the plantars. There was gross weakness of the back and lower-limb muscles. Urine volume was 100 ml./24 hours, blood urea 238 mg./100 ml., and serum potassium 5.3 mEq/l. During the next 48 hours she had intermittent generalized epileptiform fits.

Conservative treatment for acute renal failure was begun and, in view of the known dangers of severe hyperkalaemia, included "resonium-A" (an ion-exchange resin) by rectum. Fluid was administered orally as water and glucose most of the time, with only occasional superficial venous infusions of 5% glucose.

Fig. 2 shows the progress with regard to urine volume, blood urea, and dialyses. The patient was difficult to manage because of emotional instability and bouts of overbreathing, twitching, and screaming. Fig. 3 depicts the serum-potassium levels and the effect of large amounts of resonium-A per rectum. It will be seen that haemodialysis

FIG. 2.—Progress during period of oliguria and diuresis, showing blood urea falling during each dialysis.

was carried out on September 27 (9th day) for six hours, because of anticipation of a sudden hyperkalaemia.

Muscle biopsy was performed on September 29 (11th day) at the site of the tender swelling on the back (a second such area had occurred by this time, adjacent to the one described).

Histological examination (Wing Commander R. M. Cross) revealed sections of striated muscle with considerable change in normal appearance. Some of the nuclei showed degenerative changes and the majority of them had died. There was variable loss of striation and many had a granular appearance. There was little chronic inflammatory-cell response present. The appearances confirmed the diagnosis of muscle necrosis.

Fig. 4 shows the muscle necrosis. On October 2 the second dialysis was performed for six hours mainly because

FIG. 3.—Serum-potassium levels during oliguric phase and effect of rectal resonium-A and dialysis.

of clinical deterioration, and during this dialysis the patient developed hypotension, requiring noradrenaline, which we were unable to withdraw for 18 hours. Prior to this, E.C.G.s had shown some depression of the ST segments in the limb leads and leads V3 to 7. We thought that there was some myocardial damage and that the profound hypotension might have been associated with this.

On October 8 (20th day) a diuresis began (a urine volume of over 1 litre in 24 hours), and during a period of six days supplements of potassium totalling 378 mEq were required because of the excessive loss of this ion in the urine. In

FIG. 4.—Low-power photomicrograph of necrosed muscle.

the oliguric phase (20 days) she was given resonium-A to a total of 600 g. by rectum to reduce the hyperkalaemia, as shown in Fig. 3. Her blood urea reached the normal figure of 25 mg./100 ml. on October 27 (39th day).

Neurological Examination

On October 13 (25th day) a full neurological examination was made; the findings were as follows:

The patient was correctly orientated, but felt guilty about her behaviour and wept easily. She had a poor memory for recent events, a digit span of seven numbers, and a good knowledge of current affairs; she performed the serial seven test in two minutes with one error, and repeated the Babcock test sentence with one error. The cranial nerves and upper limbs were normal. The abdominal movements were normal; abdominal reflexes were absent in the lower quadrants. There was marked weakness and tenderness of the erector spinae muscles. The lower limbs were symmetrically affected. Power was grossly reduced in the quadriceps, which were wasted, and to a less extent power was reduced in the antero-lateral muscle group and the calf muscles. Tone was normal at the hips and knees; the knee-jerks were absent; the ankle-jerks were increased, with ankle clonus and extensor plantar responses. There was bilateral symmetrical blunting of sensation in all modalities in the dermatomes supplied by L 2-5 roots. There was also blunting of sensation over the sacrum where she had been lying across the chair during coma. The lower dermatomes and cauda equina were spared. The electromyogram and strength/duration curves confirmed that the lower-limb weaknesses were due to lower-motor-neurone lesions. There was transient urinary and rectal incontinence. The cerebrospinal fluid on November 25 showed 90 mg. of protein per 100 ml.

There had been a gradual recovery by mid-December. Sensation was still blunted over a small area on the front of the thighs; the knee-jerks were absent and the plantar responses extensor. Power in the lower limbs and spine had greatly improved, so that she could walk unaided for half a mile. Serial examination of the strength/duration curves showed recovery to normal within two months (Figs. 5 and 6). Sphincters had recovered.

Owing to the neurological sequelae it was not until December 6 (79th day) that she was fit to be discharged home.

When seen as an out-patient at Halton on February 4, 1959, her physical condition had greatly improved. The knee-jerks had returned and the plantar responses were flexor. Power and muscle bulk were 90% recovered. Sensation was only slightly impaired over the front of the thighs. Her mental state was normal. The Hb was 96%, x-ray examination of her back muscles showed no calcification. Renal-function tests will be performed at a later date, but her blood urea was 20 mg./100 ml., urinary urea 1.8 g./100 ml., and output about 1,500 to 2,000 ml. in 24 hours.

We consider the mental symptoms were a mild toxic psychosis. The strength/duration curves in the lower-limb muscles, the reflex changes, and the loss of sensation in the associated nerve roots all point to a radiculomyelitis as the cause of the muscle-wasting in the lower limbs.

Discussion

Muscle Necrosis

Apart from trauma, muscle necrosis occurs from anoxia due to carbon monoxide, barbiturates (Touzé and Thierry, 1952), barbiturates and alcoholism (Fahlgren *et al.*, 1957), severe heart failure (Adams *et al.*, 1953), and general anaesthesia (Gordon and Newman, 1953). Of the acute myonecrosis following carbon-monoxide poisoning Adams *et al.* (1953) write: "Anoxaemia, if sufficiently severe, may cause necrosis of muscle. We have observed lesions of this kind in the heart and skeletal limb muscles of humans who have survived for 24-48 hours after being subjected to prolonged anoxia in circulatory collapse." These authorities imply that carbon monoxide myonecrosis is secondary to prolonged pressure and is confined to muscles laid upon during anoxia. We would stress that pressure is important only in causing local increase of anoxia and thereby the actual site of necrosis. The most sensitive part of a limb to oxygen lack is the extremities, and carbon-monoxide myonecrosis has been reported symmetrically in the hands and feet (Laignel-

FIG. 5.—Left vastus medialis muscle: strength/duration curve showing a normal response on November 1, 1958.

Lavastine and Alajouanine, 1921). Heart muscle can also be damaged by carbon-monoxide poisoning (Beck and Suter, 1938; Hadley, 1952), as we believe occurred to a small extent in this case.

It is important to remember that necrosis of skeletal muscle can occur with alcoholism. One of the patients of Fahlgren *et al.* (1957) in renal failure developed a swelling of his right arm and shoulder with superficial oedema. Muscle necrosis was found at necropsy. In retrospect we believe necrosis was probably present in a man aged 26 admitted to our renal unit in 1958 in acute renal failure following acute alcoholism (he had infantile hemiplegia and was an epileptic). He had been thrown out of a London public-house and was found lying on his right side and arm in the gutter. He developed a swelling of his right arm from shoulder to elbow which was very tender and took three weeks to subside. There was no bruising. His blood urea rose rapidly to 580 mg./100 ml. in eight days, he was dialysed once, and recovered; muscle biopsy was not done.

Neurological Sequelae

The incidence of nerve damage in carbon-monoxide poisoning is difficult to calculate. If the transient Babinski sign and minimal nystagmus are taken as evidence of nerve damage, then the condition is common; thus Meigs and Hughes (1952) found nerve damage in 79% of 105 hospital cases. Shillito *et al.* (1936) found among the records of 21,000 cases only 43 neuropathies (0.2%), but he listed only well-established lesions.

The histological changes in nerve tissue poisoned by carbon monoxide are those of anoxia (Kinnier Wilson, 1954). Whether the oxygen lack is anaemic, anoxic, or stagnant, the damage wrought in the cells is the same. Direct histotoxic damage to nerve by carbon monoxide was disproved by culturing neuroblasts in 70% carbon monoxide (Haggard, 1922). The concentration of carbon monoxide necessary to inhibit oxidase systems is much higher than the toxic dose (Crisler, 1935). The neuro-histology of "delayed" cases is different and

merits comment. Courville (1957) reviewed 22 necropsies. The average time between poisoning and death was 48 days. The pathology was similar in all cases. Extensive confluent zones of perivascular demyelination occurred in the central white matter of the hemispheres. The immediately subcortical white matter was spared. In the demyelinated zones capillaries were reduced in number, arterioles could no longer be identified, and oligodendrocytes were sparse.

Damage to nerve tissue may result in psychiatric, neurological, or mixed syndromes. Cases may be classified as immediate or delayed according to whether symptoms arise within a day or two of return to consciousness or some weeks later. Lesions have been recorded in all parts of the nervous system in carbon-monoxide poisoning. Cortical damage has resulted in hemiplegia, monoplegia, aphasia, hemianopsia, cortical blindness, etc. Commonly, these symptoms are transient. Diffuse cortical damage gives rise to dementia: all gradations are seen, from a minor constrictional apraxia to complete mindlessness. Gerstmann's syndrome is common during recovery from confusion (Allison, 1956).

Traditionally the basal ganglia are considered a locus minoris resistentiae to carbon monoxide, and Parkinsonism the commonest sequela. Analysis of the cases of Shillito *et al.*, one of the largest groups in the literature, makes this view dubious. Table II shows that

TABLE II.—*Sequelae in Carbon-monoxide Poisoning Among 21,143 Cases (Shillito et al., 1936)*

No. showing muscular hypertonia (increased reflexes, perhaps clonus and positive Babinski sign)	9
No. showing hypertonia and peripheral neuritis	5
" " Parkinsonism	4
" " and some degree of peripheral neuritis	1
" " peripheral neuritis only	4
" " no neurological sign	10
" dying before complete neurological examination	10
	43

of 43 survivors from carbon-monoxide poisoning with neurological damage, five had Parkinsonism. But the chance of extrapyramidal symptoms getting better is slight. Only one case of Parkinsonism cleared up, while all but four of 28 other neurological cases lost their symptoms. Because anoxic Parkinsonism rarely shortens life, is rarely cured, and is recognizable at a glance, it may be reported too often. Other clinical pictures resulting from anoxic damage to the basal ganglia are rare. Pupillary changes and nystagmus often tell of brain-stem lesions (Meigs and Hughes, 1952). In the cord, paraplegia has been noted (Zipf, 1937). Radicular lesions are less common than neuropathies (Renfert and Drew, 1955). Peripheral neuropathies are sometimes complicated by burning pain. Epilepsy is rare but may be intractable.

The mildest psychic changes in carbon-monoxide poisoning may be confined to a transient mood change, often in keeping with the life situation, as in the case above. Temporary uncovering of anti-social traits such as aggressiveness may last some months. The severe psychiatric syndrome is an organic toxic-confusional state: confusion, amnesia, disorientation, lack of judgment, and mood changes are evident, while hallucinations and other schizophrenic symptoms are uncommon.

The neurological deficit after carbon-monoxide poisoning is usually apparent within a few days of exposure. The delayed cases may occur weeks later: usually, recovery has not been complete when the

FIG. 6.—Left gastrocnemius muscle: strength/duration curves showing a normal response on November 1, 1958.

relapse occurs. The neurological lesions are in no way distinctive, and may affect any part of the nervous system. There is nothing to distinguish the toxic psychosis of carbon monoxide from that due to any other noxious process unless it be the history of exposure and the coincidental neurological damage.

Carbon monoxide has long enjoyed an evil reputation for causing irreparable mental and nervous syndromes. Yet of 43 survivors with severe neurological or psychiatric disorders all but 10 recovered (Shillito *et al.*, 1936). Recovery usually ceases within two years but may continue up to this time, so that there is no reason to give a gloomy outlook in the early acute case. The delayed case is a different matter. In two series "pseudorecovery," as this is sometimes called, has been followed by death in half the cases. Treatment of the neurological damage is confined to reassurance, rest, passive movement, and prevention of stretching the paretic muscles. Passive full-range movement of necrosed muscles is essential to prevent Volkmann's contracture and calcification. The treatment of psychiatric syndromes should be supportive psychotherapy.

Renal Failure

When acute renal failure has occurred with muscle necrosis the constant danger is the hyperkalaemia with a sudden rise to fatal levels. It can be seen from Fig. 3 that, in the case reported, large amounts of resonium-A only just controlled the hyperkalaemia temporarily. Dialysis in these cases should be carried out earlier than the clinical state would indicate and before the serum potassium has reached 7 mEq/l., or the serum bicarbonate 12 mEq/l. Serum-potassium levels were estimated twice daily together with the occasional E.C.G., though the latter we have found to be of less assistance in the assessment.

With this type of case we would go so far as to recommend transfer to a unit where the technique for haemodialysis is available, as soon as the possibility of muscle necrosis is suspected.

No myoglobinuria was found in this patient, as it was only on the third day that a search was made. The renal lesion was acute tubular necrosis, and it is anticipated that the patient will regain normal renal function within 12 months. The management of a difficult case of this nature is the same as with any acute renal failure with much tissue breakdown. Our experience has shown the necessity for the most careful and frequent assessment at the bedside, with all the facts assembled at least twice a day. Otherwise these patients can slip out of control rapidly. One should be prepared to dialyse early even though this may mean an extra dialysis. This is made evident by the reports of Fahlgren *et al.* (1957) and of Loughridge *et al.* (1958).

Summary

A case is described of carbon-monoxide poisoning with the following complications — skeletal-muscle necrosis, probable myocardial necrosis, acute renal failure, mental changes, and a radiculomyelitis of L 2-5.

The details of conservative management and haemodialysis (twice) by a Kolff twin-coil artificial kidney are given. The literature concerning muscle necrosis and renal failure, with the neurological sequelae, is reviewed.

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ARTIFICIAL RESPIRATION AND AN ARTIFICIAL KIDNEY FOR SEVERE BARBITURATE POISONING

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Most patients admitted to hospital suffering from barbiturate intoxication will recover provided that steps are taken to keep the airway clear, to maintain fluid balance, and to prevent hypostatic pneumonia (Nilsson, 1951; Locket and Angus, 1952). Some authors favour the use of analeptics such as picrotoxin and bemegride ("megimide") (Shulman *et al.*, 1955; Koppanyi and Richards, 1958), but there is little evidence that these drugs play any major part in the recovery of the patients (Eckenhoff and Dam, 1956). The essential of treatment, however, should be to maintain adequate respiration of the patient and an adequate circulation. The problem is similar to that of managing a deeply anaesthetized patient; the only difference is that the person with barbiturate intoxication usually remains in coma for a longer time. It may be possible to shorten the duration of coma substantially by dialysing