and air sampling and dust sampling at the factory followed by culturing, was negative in all respects.

2 The investigation of the wire covering revealed that this material was a polyurethane and that when it is heated to destruction, isocyanate vapours are released to atmosphere. Subsequently, enquiries of the manufacturer of the wire disclosed that the covering was polyurethane based on toluene diisocyanate.

Nitriles and isocyanates produce absorption bands at $4.4\ \mu$ when scanned on an infra-red spectrophotometer. This position is relatively free from other well-defined absorption bands. Absorption bands at $4.4\ \mu$ were found when the vapours evolved during soldering were absorbed in wash bottles of petroleum ether and the resulting solutions scanned. Decomposition of polyurethanes occurs at $220^{\circ}\text{C}.$ – $275^{\circ}\text{C}.$, producing isocyanates and N-oxides (Figs 1 and 2). The temperature of a soldering iron in normal soldering operations is $300^{\circ}\text{C}.$

The picture then appeared to be complete, and some of the more puzzling aspects were explained. The onset of symptoms coincided with the introduction of the polyurethane-covered wire. The wire was not in constant use but was used intermittently for winding a particular type of coil. The fact that all the cases which occurred in the terminal dipping section were among those working at the ventilated benches appeared also to be explained because these benches were adjacent to the coil winders who, by applying hot soldering irons to polyurethane, were releasing small quantities of isocyanate vapour to atmosphere at frequent intervals.

Conclusions

This finding appears to be important in a number of respects. There is the immediate hazard in relation to the work of coil winding using polyurethane-covered wire. There are also remote hazards, indicated by this finding, related to the ever widening use of these polyurethane materials. When buildings in which are incorporated large quantities of polyurethane foams, surface applications, etc. are set alight, there might be a serious hazard to firemen and others engaged in combating the fire. The position appears to be even more serious in the case of fire at sea when refrigerated compartments in ships become involved or in engine rooms where polyurethane cladding of engines has been used for noise suppression, because in these circumstances those engaged in putting out the fires would probably be working in confined spaces and might be exposed to high concentrations of isocyanate vapours.

My thanks are due to Professor K. B. Fraser, Professor of Microbiology, Queen's University, Belfast for the 'farmer's lung' investigation, to Dr. A. J. Howard, M.A., Ph.D., F.R.I.C., Director, Department of Industrial and Forensic Science, Ministry of Commerce, Northern Ireland, to Mr. T. J. McCullins, M.Sc., A.R.I.C., C.Eng., Principal Scientific Officer, Department of Industrial and Forensic Science who carried out the chemical investigation, to Dr. J. E. Sutcliffe, Works M.O., to Dr. F. F. Main, Chief Medical Officer, Ministry of Health and Social Services Northern Ireland for permission to publish this report, and to Dr. T. A. Lloyd Davies for valuable advice.

Received for publication April 19, 1968.

Munchausen's syndrome simulating caisson disease

J. H. KEMP AND J. G. MUNRO
H.M. Dockyard, Chatham, Kent

We report a patient who was transferred from a London hospital with apparent caisson asphyxia and was treated in the compression chamber of H.M. Dockyard, Chatham which, in addition to its naval uses, treats civilian cases from south-east

England. He was found to have Munchausen's syndrome which has not been reported with this presentation before.

At 7.0 p.m. on January 8, 1968 a small, one-eyed, elderly man smelling of alcohol arrived from hospital

with his face, hands, and chest clothing covered with clotted blood. He said he was a tunnel worker on the new Victoria Line in London where, during the previous 12 hours he had done four two-hour shifts at 30 lb./sq. in., with 20 minutes decompression each shift. He complained that one hour previously he had been seized with retrosternal pain and had coughed up half a pint of blood. He said he knew he had 'the bends' as he had once been a naval diver and had had them before.

As we began to examine him, he complained of chest pain again and said he felt faint and giddy; then he collapsed, his pulse disappeared, and he stopped breathing. So he was put straight into the chamber with a doctor (J.H.K.) and a diver, and compressed to the equivalent of 165 ft. depth. He remained unconscious, but a weak pulse and breathing returned.

As the metal poles of the patient's stretcher could not be removed from the closed chamber they had to be sawn in half before being passed out of the lock. While sawing, the attendants became distressingly short of breath, after which they suffered a brief period of amnesia during which outside observers noticed they were behaving oddly; possibly this was a nitrogen narcosis due to excess CO₂ from the strenuous work under pressure in a small confined space occupied by three people.

The patient then came round and said that he had been working at 30 lb./sq. in., not for 12 hours, as he first said, but for 24 hours. He had worked overtime because there had been a 'cave-in' in which two of his friends had been killed. He had volunteered for the extra work in an effort to reach these friends. The patient also stated that he was 72 years old and gave the address of his wife, his next of kin. These details were passed to the attendants outside the chamber.

He then had the first of many attacks which were to become a feature of this therapeutic recompression. The attack started with the whole body becoming rigid, the legs fully extended, the arms flexed, the feet hammered on the deck, the patient lost consciousness, respiration stopped, but the pulse remained strong and regular. Respiration gradually returned, generalized convulsions occurred for a few seconds, and the patient relaxed and recovered consciousness after about one minute.

These epileptiform seizures occurred at approximately five-minute intervals. Between the attacks the patient was coherent and co-ordinated. No abnormality of his cardiovascular or respiratory systems was detected. During discussion in the patient's lucid intervals the following further facts were elicited:

The patient had been invalided from the Royal Navy as a Lieutenant Commander (E) with pulmonary tuberculosis.

He had had four periods as an in-patient in different sanatoria.

He was receiving P.A.S. and daily streptomycin from a District Nurse.

He had had a number of attacks of 'bends'.

He had lost his left eye in a Japanese P.O.W. Camp on the Burma Road after surviving the sinking of H.M.S. *Repulse*. He had been the Engineer Officer of H.M.S. *Repulse*.

He had sustained multiple injuries in a mine accident in Yorkshire in 1958 which explained his numerous abdominal scars.

He had injured his left hand in a mine in 1962 and had lost his left little finger and the end of his ring finger.

He also gave several addresses to confirm his recent occupational history.

Meanwhile the attendants outside the chamber had been busy trying to contact his wife at the address the patient had given in Birmingham but had been unsuccessful. They had also been unsuccessful in their attempts to find any contractor on the Victoria Line who would admit that he employed him. New Scotland Yard's services had been engaged and they had ascertained that no accident had occurred in the construction of the Victoria Line for at least 14 days. Furthermore, they had been unable to trace the man at any of the addresses he had given.

The attendants outside the chamber therefore searched his pockets and effects. They discovered letters addressed to him in four different names. Through these names it was possible to establish his identity and present address from the details elicited.

When he was taxed with these facts he expressed anger that his personal effects had been searched. He admitted the names but explained that he had done this to obtain work as employers were reluctant to employ old men with tuberculosis. He volunteered further addresses for the attendants to investigate.

The patient had been kept for two hours at 165 ft. with little or no change in his condition and therefore therapeutic decompression from this depth had been started.

Gradually the convulsions changed in character and became less frequent. They started with very severe pain in the right loin radiating to the right groin. He clutched his right side, became rigid, lost consciousness, suffered rigors, relaxed, and regained consciousness. These attacks occurred in rapid succession. They were relieved by pethidine, 100 mg. intramuscularly for a period of approximately four hours at the beginning of the decompression, until towards the end of the decompression pethidine, 100 mg., relieved his symptoms for eight hours.

The patient also had several haemoptyses. He vomited all the food and drink he took in the chamber. He complained of dysuria.

He volunteered the information that he had developed renal tuberculosis whilst under sanatorium treatment and had had a right heminephrectomy after his accident in 1958. This history was related to the attacks which were by this time developing clinically into episodes of renal colic as described above. He was tender in the right loin. He gave a history of nocturnal frequency for several weeks. He gave a history of vomiting all food and drink for three weeks prior to entering the chamber.

His position after being in the chamber for 12 hours may be summarized as follows.

His history of exposure to increased air pressure was doubtful and had not been confirmed.

Everything that he had told us was suspect and one could not believe anything about his social, medical, and occupational background.

It did appear that there was genuine right renal pathology and possibly a secondary uraemia.

It was very doubtful that he had had a bend at all but unfortunately he had been submitted to two hours' compression at 165 ft. and slow therapeutic decompression had to be continued.

Decompression was continued but punctuated by vomiting, haemoptyses, and attacks as described above. The tedium was relieved by lurid descriptions of Japanese atrocities on the Burma Road (even this was suspect as one usually associates the Burma Railway with prisoners-of-war and not the Burma Road); stories of his unsatisfactory family background with nine children; anecdotes of his childhood with his father who was still alive aged 99 in Doncaster; tales of his adventures in the diamond and gold mines of Zambia and Rhodesia; and accounts of lucky escapes from the Congo during the troubles in 1962.

These stories were told as he polished his glass eye frequently and furiously, lamenting the while that he had left his spares in his tobacco tin outside the chamber.

At 30 ft. the Medical Officer developed a bend in the left third metacarpophalangeal joint which was relieved a little by descent to 50 ft. But in view of the suspected uraemia of the patient with his developing dehydration it was considered essential to surface in the earliest possible time. Decompression was therefore continued and the chamber 'reached surface' at about 2 p.m. on January 10, 1968.

After transfer to a local hospital he collapsed again but recovered quickly when resuscitation was carried out.

He was noted to be picking his nose by the nursing staff who minutes later were summoned urgently to an impressive epistaxis. He had to be restrained from attacking the house physician.

He took his own discharge from hospital the following day.

Investigations

The following investigations were undertaken.

Haemoglobin 98%; W.C.C. 4,000 per cu. mm., polymorphs 73%, eosinophils 2%, lymphocytes 25%; E.S.R. 12 mm. in one hour; P.C.V. 43%; M.C.H.C. 33%. Blood urea 22 mg./100 ml.; serum sodium 141 mEq./litre, potassium 4.6 mEq./litre, chlorides 100 mEq./litre. Blood group A positive.

The urine showed no abnormality.

Chest radiographs showed a fracture of the sixth right rib anteriorly and the seventh right rib in the axillary line. The lung fields and cardiac silhouette were normal.

A radiograph of the abdomen showed multiple metallic sutures to the right of 12DV but otherwise no abnormality.

On January 15, 1968, *i.e.*, exactly one week later, a request was received for facilities to treat a case of 'bends'. Enquiry revealed that it was the same patient again. Recompression was not carried out.

Discussion

The three modes of presentation described by Asher, acute abdominal pain (laparotomaphilia migrans), visible blood loss (haemorrhagica histrionica), and dramatic central nervous system symptoms (neurologica diabolica), were all portrayed by this patient. The history was suitably dramatic and held water enough to mislead experienced divers; the technical details of his times and 'depths' under pressure were not challenged. The evidence of a multiplicity of injuries was shown in this case and, however they arose, bore tribute to a desire for self-mutilation attributed by some authors to these patients. It is also hard to imagine a more spartan choice than spending two days and nights with two others in a sealed metal cylinder 9 ft. long and only 5 ft. in diameter!

He discharged himself from hospital when the normal results of all clinical investigations were about to confirm a diagnosis consistent with Munchausen's syndrome.

It is interesting to speculate how long this man will be successful with his compression illness presentation. For, although he can choose between many hospitals in the country, recompression chambers are fairly limited and it should not be long before his description is known.

The authors wish to express their thanks to Vice Admiral W. J. Parker, C.B., O.B.E., D.S.C., for permission to publish this paper, and to Dr. J. D. Craig, Consultant Physician, Medway Hospital, for the results of investigations on this patient while under his care.

Received for publication April 26, 1968.