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THE COMMONWEALTH OIL REFINERIES LTD.

90 William Street,
MELBOURNE, C.I.

CONFIDENTIAL

15th July, 1947.

Mr. J. E. Batchelor,
The Associated Ethyl Co. Ltd.,
368 Collins Street,
MELBOURNE. C.I.

Dear Sir,

In compliance with your verbal request we submit herewith details of the tank cleaning operations at Canberra Airport subsequent to which two of our employees, [REDACTED] and [REDACTED] became ill.

Two 10,000 gallon underground storage tanks at Canberra aerodrome filled with water were being pumped out and cleaned internally. Pumping operations having been completed on Thursday, 19th June, the cleaning operation then commenced.

One tank had been completed and the second tank partially completed when, on ceasing work on the evening of Friday 20th June, [REDACTED] complained of feeling ill and retired without a meal. On Saturday the 21st, he consulted Dr. A. H. Hart of Queanbeyan and reported vomiting and diarrhoea. Gastritis being prevalent in the district, Dr. Hart, after consultation with Dr. A. J. Harker, had him admitted to Queanbeyan District Hospital to ensure proper night attention which could not be given at his hotel.

By this time, a second man ([REDACTED]) having developed similar symptoms, the possibility of the illness being due to the cleaning of the tank was considered, particularly as it had been since ascertained that the tanks had at some previous stage contained aviation spirit.

On the morning of Sunday, the 22nd, Dr. C. R. Bickerton Blackburn of Sydney, N.S.W. medical officer for the Associated Ethyl Co. Ltd., was notified in view of the possibility of poisoning from the tetra-ethyl-lead content of the aviation spirit, some small accumulation of which might have remained in the tank sludge.

Dr. Blackburn discussed the situation with Dr. Harker of Queanbeyan, and Kenna's condition having later in the day deteriorated, it was decided that he should be sent by ambulance immediately to the Royal Prince Alfred Hospital, Sydney, where he arrived at approximately 12.35 a.m. on Monday, the 23rd, accompanied by Dr. Harker.

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He was admitted by Dr. McDonald, resident medical officer, and was visited by Dr. Blackburn at approximately 12.45 a.m.

It is understood that treatment commenced immediately.

The tanks were purchased by us from Disposals Commission and when purchased were full of water. A test carried out on 3rd June by our Branch Engineer, Mr. N. S. Panton, gave no indication of gas. On the 17th June, after water had been pumped out with a power pump the tanks were again tested for gas with negative results.

The two operators then proceeded to clean the tanks with wire brushes. The men were not equipped with standard T.E.L. equipment but wore rubber boots and fabric gloves. Testing apparatus was kept on the job and it was arranged that if the operators found the atmosphere becoming foul, gas masks would be sent by airmail. The men were instructed to use a shovel for removing the sludge in the tank.

The Branch Engineer was misled by the fact that the tanks had been filled with water and did not check with R.A.A.F. the type of product which had been stored and overlooked that there may have been lead in the sludge which remained in the tank.

Subsequently, on unearthing vent lines from the tanks, which incidentally were carried underground for some distance, clear of the runway, it was found that they were choked with some black material. It was further discovered that there was a 44-gallon drum buried in the ground to which the vent pipes were connected. On being unearthed this drum was full of what appeared to be aviation spirit. No plans of the installation were available and the existence of the 44-gallon drum would not normally have been suspected, and more particularly the fact that it was filled with aviation spirit.

The above information is released to you on the distinct understanding that it is treated as confidential.

Yours faithfully,
THE COMMONWEALTH OIL REFINERIES LTD.

sgd. D.E. BALDWIN

Chief Engineer.

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18th July, 1947.

REPORT ON TWO MEN POISONED WITH TETRAETHYL LEAD OR ITS DERIVATIVES

Two employees of the Commonwealth Oil Refineries Ltd., we exposed to TEL in the following circumstances as narrated to me by the men concerned and verbally by officers of the COR company.

Two tanks that had not contained gasoline for upwards of two years had been found to contain water, it was therefore, proposed to remove this water and clean the tanks. The water was removed, tests apparently revealed no gasoline and the men proceeded to clean the tanks. It has been stated that the second tank alone contained TEL or its derivatives but no certain information is to hand on this point. In this tank the men proceeded to remove the sludge using no protective devices other than rubber knee boots and canvas gloves - no respirators of any kind were used. On account of the 'fumes' the men could not stay longer than 10 minutes at a time in the tank, so one man would fill a bucket with sludge (using tin cans etc to scoop up the sludge) whilst the other would haul the filled bucket out by a rope. They took turn and turn about in these operations during the day. One man, R. KENNA, acquired a number of abrasions on the backs of both hands while scooping up the sludge. The tank was cleaned on 20 JUNE 1947 and both men agree that R. KENNA had the greater exposure.

Both men developed clinical evidence of TEL poisoning and lead was found in significant quantities in their urine specimens. Their histories were complicated by an epidemic of 'food-poisoning' which was prevalent in the town (Queanbeyan) at the time. Their clinical histories and treatment are set out below, the milder case is given first.

[REDACTED] aged 34, a married man with 2 children was admitted to Royal Prince Alfred Hospital, Sydney, under my care at 10.30 p.m. 22 JUNE 47. He stated that he had a mild gastro-intestinal upset for 4 days and that his diarrhoea ceased the day before admission. His important symptoms were increasing insomnia, increasing anorexia, weakness and tiredness, and tremor involving his upper limbs more than his lower. He had consulted the local Doctor on account of his gastro-intestinal symptoms but, becoming worried over his insomnia and weakness following on the 'fumes' in the tank operations, he proceeded to Sydney on his own initiative. On arrival he was met by COR representatives and admitted to hospital.

On admission he looked tired and worried, he was very alert mentally, and had moderate tremor of both hands. His oral temperature was 96°F, his pulse rate 66 per minute, and his blood pressure 120/75 mm Hg. No other abnormal signs were found.

He was confined to bed and given alkali (potassium citrate) in doses sufficient to keep his urine alkaline, oral calcium lactate,

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barbitone sedatives, and fluids in large quantities.

2:3-dimercaptopropanol or British Anti-lewisite) as a 10% solution in oil and benzyl-benzoate was administered by intramuscular injection. He was given 4.4 mg/kg each 4 hours for 4 doses and then 2.2 mg/kg twice daily till 28 JUNE 47 when he was given 4.4 mg/kg each 6 hours; on the following day he received 2.2. mg/kg each 6 hours. From 30 JUNE to 3 JULY inclusive he received 4.4. mg/kg twice daily, from 4 JULY to 10 JULY inclusive 2.2. mg/kg twice daily, and from 11 JULY to 14 JULY 2.2. mg/kg once daily.

Though his general condition improved and he took food and fluids well he could not sleep without sedation and remained restless and bright. These latter symptoms increased from 25 JUNE to 27 JUNE when he was becoming a little fractious - quite different to his condition on 24 JUNE when he had settled down well. From 30 JUNE he was relatively quite and the made an uninterrupted recovery apart from a mild infection in his buttock following BAL injections.

The urine was examined for lead and the following results were obtained on 24 hour specimens - the specimen collected 24 JUNE contained 0.09 mg, the maximum amount of 0.79 mg was found in the specimen collected between 27 and 28 JUNE, and by 5 JULY only traces were present.

During the first five days in hospital his oral temperature ranged between 97 and 98 °F, his pulse was 52 to 60 perminute, and his blood pressure was little altered from the earlier figures quoted. An occasional stippled red blood cell was seen on two occasions. at the time of writing this man has just been discharged from hospital.

[REDACTED] aged 30, a married man, was admitted to Royal Prince Alfred Hospital, Sydney, under my care at 12.30 a.m. on 23 JUNE 1947.

His history was complicated by a mild gastro-intestinal infection that had been present for 10 days becoming worse in the last 4 days. He had been admitted to Queanbeyan Hospital under the care of Dr. Harker on 21 JUNE 47 for his gastro-intestinal upset. His alimentary symptoms had largely disappeared by 22 JUNE 47 but on this morning he was mildly confused. He had a short epileptiform fit about 1.30 pm and was brought to Sydney. He complained of insomnia and distressing dreams, anorexia, weakness of his legs, and aching of his upper and lower jaws. On admission to R.P. A.H. he was confused, could not relate the incidents of the day, and was constantly stretching his limbs and writhing on his bed. He said he had general abdominal discomfort. He was found to have tremor of his hands and many small abrasions on the knuckles of both hands, his blood pressure was 120/55 mm of Hg, his pulse rate 64 per minute, and his oral temperature 98.4°F.

He was confined to bed, given sedatives, and, as he could take nothing by mouth, was given intravenous saline with 5% glucose and, later, intravenous sodium lactate. Subsequently the urine was kept alkaline with suitable doses of potassium citrate by mouth.

BAL was given intramuscularly in doses of 4.4 mg/kg for 4 doses at 4 hour intervals and followed by 2.2. mg/kg twice daily till 27 JUNE 47. Within 14 hours of receiving therapy he was much better, he was not at all confused, was starting to take fluid and food by mouth but he was still restless and active though still in bed. His appetite returned and he was most cooperative in spite of some restlessness till the morning of 27 JUNE

ypomaniac. He escaped from the ward for a few minutes, rcibly returned, had an epileptiform fit lasting 1.5 minutes. Following this he was confused, disorientated, deluded and hellucinated, he was difficult to control, he tore up his pyjamas, and had to be restrained. He was given sedatives and hyoscine and an intravenous injection of hypertonic glucose was attempted. BAL was administered in doses of 4.4. mg/kg each four hours for 5 doses and followed by 4.4. mg/kg twice daily for 2 days, subsequently he was given 2.9 mg/kg 8 hourly from 30 JUNE to 3 JULY. By 28 JUNE he was manic and the only improvement was in the diminishing violence. However, by 30 JUNE he was much improved - he was mentally much clearer, much less active, and was resting. From this time until 3 JULY he was much improved, his appetite increased, he was mentally clear, not especially restless, but complained of increasing pains in his muscles and abdomen. By 3 JULY he had obvious pain on attempting to sit up in bed - most of his muscles were painful to move. On 3 JULY the dosage of BAL was increased to 4.4. mg/kg which was continued till 4 JULY. His muscle pains rapidly disappeared and his colic also vanished. The dosage of BAL subsequent to 4 JULY was as follows: 4 JULY - 2.9 mg/kg 4 hourly; 6-13 JULY - 2.9 mg/kg 8 hourly; 14 JULY - 1.5 mg/kg twice daily. By the evening of 14 JULY he had received 234.9 mg BAL/kg (160cc of the 10% solution in benzyl-benzoate and peanut oil). Some toxic symptoms were observed following the use of BAL. His pulse rate ranged between 48 and 60 per minute until he became maniacal but thereafter shewed considerable variation according to the dose of BAL being given. His oral temperature was usually below 98.4°F. Estimations of the lead concentration in the urine were made whenever practicable the maximum 24 hour concentration observed was 0.30 mg, only traces being found on 9 JULY. No great change was observed in the red and white blood cell counts.

In the case of KEMMA it is difficult to avoid the conclusion that BAL was the significant therapeutic agent and that it relieved his symptoms when given in sufficient dosage. It is my opinion that BAL was responsible for the very satisfactory result of the treatment of this man's lead poisoning.

COMMENT:

BAL appears to be an effective agent in the treatment of lead poisoning from the absorption of TEL or its degradation products. It needs to be given in high dosage and for a relatively prolonged time. The experience of the above patients would suggest that a regimen such as set out below might be suitable -

BAL (2:3-dimercaptopropanol) 4.4 mg/kg of body weight each 4 hours till the symptoms have disappeared, then 4.4. mg/kg each 8 hours for another 14 days. The dosage should be diminished gradually rather than suddenly ceased until experience has shewn that smaller doses are adequate.

It is suggested that the standard BAL ointment be kept in hand in all places where there may be skin contamination and that its use to be obligatory when there has been exposure to TEL on the skin. Experimental work will no doubt, indicate whether such therapy is effective.

Lastly it would appear to be most desirable to administer BAL in therapeutic doses to all personnel who have been exposed to TEL and to begin such therapy as soon as exposure is known to have occurred rather than to wait for symptoms or the demonstration of lead in the excreta.

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