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Paper

The Results of Intoxication with Orthocresyl Phosphate Absorbed from Contaminated Cooking Oil, as seen in 4,029 Patients in Morocco

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During September and October 1959 olive oil which had been adulterated with lubricating oil used in jet engines was sold in and around the city of Meknès in Central Morocco. The contaminant was a man-made lubricating oil containing 3% of mixed cresyl phosphates. After using the oil for cooking some 10,000 people became ill. Later in the year, when Government action was taken to control the sale of edible oil in the Meknès area, the merchants responsible moved to the north of the Rif Mountains to the province of Nador, and further cases of intoxication occurred in the area around the fishing port of Alhucemas on the Mediterranean coast.

At that time Meknès was full of visitors who had come to the city for the Feast of the Prophet and a number of these became ill. As they recovered they returned to their homes. Thus, during the final control of the patients in June 1961, Red Cross teams visited all regions of Morocco from Oudja on the Algerian border to Casablanca and from Tetouan in the north to Ksar-es-Souk in the Sahara.

Immediately the outbreak occurred the Moroccan Government appealed to the World Health Organization and to the League of Red Cross Societies for assistance in establishing the cause of the illness and in treating the victims. The preliminary treatment programme was planned by Professor Dennis Léroy of the University of Rennes, France, and the investigation into the cause of the outbreak was undertaken by Dr Honor Smith and Dr J M K Spalding of Oxford. They found that only Arabs were affected and that the very rich and the very poor were spared. In the crowded slum area cases occurred sporadically and while one whole family might be ill, their neighbours would not be affected. Furthermore, troops living in barracks were spared but a number of soldiers and police living out in the town became casualties. They ruled out the possibility of infection and managed to trace the contaminated oil. Analysis revealed the presence of cresyl phosphates.

Similar intoxication due to cresyl phosphate poisoning occurred in Switzerland in 1941 and in the U.S.A. in the early 1930s (the so-called 'ginger paralysis'). Recently I have received reports that American servicemen who have used jet engine lubricating oil on their skins to produce an artificial suntan have also developed signs of cresyl phosphate poisoning.

The exact mechanism by which the toxic effects are produced is not understood. Cresyl phosphate may interfere with the normal process of fat emulsification and absorption and emboli may occur in the central nervous system. The nature of the pathological lesion in man is not well documented because orthocresyl phosphate poisoning carries a very low mortality rate.

Clinical Observations

Motor changes: At the outset there was a flaccid paralysis affecting all the muscles below the knee. The paralysis was invariably bilateral and symmetrical. Early muscle testing showed all muscle groups below the knee as M=O (M.R.C. scale) in the majority of patients. If the patients had consumed more oil the small muscles of the hand were also affected. The opponens pollicis was the muscle most frequently paralysed and was the slowest to recover, but often all the intrinsic muscles were affected giving rise to the typically flat hand (*main de singe*) due to severe wasting of both the thenar and hypothenar eminences. Patients who had consumed even more oil had weakness or paralysis of the muscles of the thighs and buttocks. Amongst 4,029 patients registered at Meknès, 65% had hand involvement and 6·6% had both hand weakness and some degree of paresis of muscles to the level of the hips.

Sensory changes: A number of patients had sensory loss affecting the hands and feet, but this

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quickly recovered. No traces of loss of joint position sense or of inco-ordination were found.

Other symptoms: During the early stages of the intoxication many patients complained of impotence, but this was transitory. Later on patients became pregnant and had normal deliveries even if they were spastic. There was no impairment of bowel or bladder function. Vascular changes were rare and there were few cases of trophic ulceration even amongst patients in plaster or wearing splints. One patient who remained severely flaccid had some circulatory deficiency in his feet. In fact the clinical picture at the outset was one of a peripheral neuritis of toxic origin.

Recovery of the paralysis began during the spring of 1960 and by the end of that year all but about 700 patients at Meknès had ceased treatment. During 1960, however, the first signs of spasticity appeared in an increasing number of patients. The earliest records of this in Meknès were in April and the latest time at which spasticity was first reported was in November. Since then no fresh cases of spasticity have been seen. A few patients who developed spasticity had previously recovered from their initial flaccidity and had stopped treatment, but in most cases spasticity occurred in patients who were still under treatment with some degree of muscle weakness. Spasticity in varying degree was the main problem in treatment between February and July 1961. In February 90% of the patients under treatment in El Mers were spastic and of the 62 patients remaining under treatment at the end of June all but one were spastic.

The spasticity, which was invariably bilateral and symmetrical, was found most commonly in the gastrocnemius, soleus and long flexors of the toes, in the hamstrings, hip adductors, the quadriceps and hip flexors, in the gluteal muscles and the erector spinæ groups as far up as the lower angle of the scapula. It was never found above this level nor in any muscle group of the upper limbs. I can find no record of it in the anterior tibial muscles. Clonus could be elicited in about 65% of spastic muscles and the tendon reflexes were invariably increased. The Babinski response was positive, however, in only about 5% of spastic patients.

The spastic muscles felt 'woody' to the touch and were often tender. Muscle pain was a common symptom and persisted for several months after all motor signs had cleared up. It was also found in muscles which were otherwise normal.

The characteristic deformities of spastic patients were: (1) Equinovarus of foot. (2) Genu recurvatum or slight flexion deformity of knee – depending on whether the hamstrings or the quadriceps muscle were the more spastic. (3) Adduction, flexion and external rotation of hip. (4) Forward tilt of pelvis with increased lumbar curve.

Gait: The patient walked on his toes, throwing his hip forward at each pace. There was frequently instability of the ankle, the foot going into an acute varus position. In addition the patient hyperextended his spine sharply at each step.

The spasticity was elicited just before the patient's heel reached the ground and if the heel of the patient's shoe was raised so that the gastrocnemius was not stretched when walking the spasticity was hardly apparent. A number of patients with a very badly spastic gait showed very little sign of spasticity when examined at rest. Immediately the spasticity had been elicited in one muscle group, other groups were stimulated and spasm appeared there too.

In June, the Royal Air Force presented a diagnostic electronic stimulator to the Moroccan Red Crescent and it became possible to start a limited electrodiagnostic survey. Intensity duration curves were plotted for the gastrocnemius, the adductor magnus and the gluteus maximus in a number of spastic patients; these showed progressive distal denervation.

From March to July 1961 the degree of spasticity in a number of patients began to diminish. This was not entirely due to the effect of treatment because about 200 patients with light to moderate spasticity, who were ambulant with heel raises and simple splints, were allowed to stop treatment and to go home. They were reviewed medically once a month. It was amongst these patients that the lessening of the spasticity was first noticed. In June a number of patients who had earlier been spastic were found to have no trace of spasticity. This was a most important finding because it indicated that the more severely spastic patients would probably also recover and that the prognosis was better than had been predicted.

Classical spasticity, as seen in cerebral palsy, is due to a lesion of the upper motor neuron, and is characterized by muscle hyperactivity, increased tendon reflexes, clonus and a positive Babinski sign.

Muscle hyperactivity may also be due to changes within the muscles themselves. These changes may be at the motor end-plate (nerve gas poisoning), in the balance between intra- and extra-cellular potassium (congenital myotonia) or in the muscle spindles themselves. Spasticity of muscular origin is characterized by hyperactivity, increased tendon reflexes and clonus, but the Babinski response is negative.

The prognosis in upper motor neuron spasticity is poor and spontaneous recovery does not occur, whereas spasticity due to causes within the muscles themselves carries a very much better prognosis. The fact that a number of our patients at Meknès have already shown signs of a spon-

taneous remission is in favour of the hypothesis that the spasticity is due to changes within the muscles themselves. This is borne out by the intensity/duration curves, and also by the fact that only 6% of our patients had positive Babinski responses. The muscle pain and tenderness also favour this theory. It is to be hoped that specimens for muscle biopsy may be obtained when any reconstructive surgery is being done.

Professor K M Walthard of Geneva, who was responsible for the care of patients who suffered intoxication with triorthocresyl phosphate in Switzerland, lectured to the staff in Meknès in May. He claimed that the spasticity was due to changes in the muscle spindles and not to a lesion of the central nervous system, and that this had been recently confirmed by muscle biopsy. He suggested that 'pseudo-spasticity' would be a better name for the muscle hyperactivity. I believe our findings in Meknès lend support to this view.

The development of the oil intoxication may be divided into three phases: the stage of flaccid paralysis (0–6 months), the stage of recovery of flaccid paralysis and the onset of spasticity (6–15 months), and the stage of recovery of spasticity (18–? months).

Case distribution by age and sex: Of our patients in Meknès 31·1% were men, 49·6% women, 12·4% children between the ages of 7 and 15 years and 6·9% infants of less than 6 years.

The preponderance of women was even greater amongst the more seriously affected patients. This might be due to two factors, both social: It is likely that women would taste the food during preparation and would therefore consume more oil. In Morocco it is customary for men to eat alone first; the food remaining is then passed to their families who finish it up and usually mop up the dish with bread; this residue would contain large quantities of the oil in which the food had been cooked.

Treatment

Physiotherapy: During the early part of the treatment programme routine physiotherapy consisted of active and passive movements, but in March 1961 more modern techniques were introduced. These depended on working the patient in a relaxed position, thus preventing the onset of spasm whilst the weak muscles were exercised. Reinforcement and proprioceptive techniques were also used.

Although many thought that it would be difficult to teach these exercises to the Moroccan staff, they proved extremely effective and were responsible for the return of many patients to full activity. They were, however, time consuming and it was necessary to reorganize the treatment programme and to use group therapy and remedial games to occupy those patients who were not having individual treatment from the physiotherapists or Moroccan kinesiotherapists.

Hydrotherapy: Two hydrotherapy tanks had been installed during the autumn of 1960 and during April an appraisal was made of the value of hydrotherapy at this stage of the disease. The weather was then quite hot and the atmosphere in the rooms housing the two hydrotherapy tanks was intolerable. There had always been the difficulty of persuading some patients (particularly women) to undress and go into the pool. Only very limited forms of treatment coud be given as it was impracticable for the physiotherapist to enter the water with the patients. The cost of running the pools was considerable and it seemed well worth while to make sure that this cost was justified by the results obtained.

It was therefore decided to close the pools for an experimental period of two weeks. During this period the physiotherapists and doctors kept a very close watch to see if there was any deterioration in the condition of the patients who had been receiving hydrotherapy treatment. No subjective or objective difference was noticed and the period of closure of the pools was extended to one month. As this confirmed that hydrotherapy *at this stage* had no value at all, the pools were not reopened.

There can be no doubt that hydrotherapy had been an extremely valuable form of treatment earlier in the disease and particularly during the winter. The expense of providing these facilities was therefore justified. However, careful assessment showed that by April 1961 the patients treated at Meknès had progressed beyond the stage at which hydrotherapy could give any useful help and I considered that the expense of operating the pools was no longer justified.

Other methods: The spasticity was not relieved by destruction of the proprioceptive nerve endings in the Achilles tendon with absolute alcohol, nor was it in any way affected by the use of relaxant drugs. It was, however, abolished under deep anæsthesia. Rest, either in a below-knee walking plaster cast or in bed for four or five weeks, would diminish or abolish the spasticity, but, unless the patient was subsequently given heel raises, in a number of cases the spasticity returned in three to four weeks. When the heel of the patient's shoe was raised this did not occur. The application of corrective plasters in 67 cases resulted in spasm being abolished in 14 and reduced in 46; it was unchanged in 7.

Plaster stabilization of the ankles was also used, in 21 patients, to assist in gait training where the degree of deformity at the ankle prevented the patient from walking at all. All these patients were given splints, usually with high heel

raises on removal of the plaster; the gait was improved in 12, unchanged in 8 and made worse in 1. The patient whose gait was made worse had been spastic for over a year; when the plaster was removed it was found that his spasticity had been considerably reduced and he then found it very difficult to maintain his balance.

Despite the heat the plasters were well tolerated and by the use of adequate padding pressure sores presented very little problem.

Resettlement

The most serious problem was that of resettlement. In Morocco the overall unemployment rate is 20% and in Meknès it rises to 35%. Indeed Meknès is one of the areas for which special Government help has been given. Mechanization in the vineyards has cut the labour force to one-third of what it was and has augmented the unemployment problem; and in the fields the combined harvester has supplanted the hand reapers. As a result of the Red Cross action, on April 17 an inter-ministerial committee sat in Rabat to discuss the problem and to make recommendations for government intervention to help in the resettlement of the disabled. The first result of this action was the starting of preliminary schooling for 35 male patients at El Mers prior to apprentice training. The first course began in June and was due to last for three months; at the end of that time these patients would move on to practical training and it is hoped that a further preliminary course will begin. However, even after apprentice training, government action will still be required to place these patients in industry.

Training

Besides the treatment of our patients, we were also responsible for training the Moroccan staff.

Workshop technicians were trained by the European splint makers. The training was entirely practical and included the measurement of the patient, manufacture and fitting of appliances and the necessary book-keeping; despite language difficulties it was thorough and a very high standard was reached. Six hundred splints were made in our workshop between March and July.

Aides sanitaires: On recruitment all *aides sanitaires* were given a two-week theoretical and practical course and then had to pass an examination before being accepted on a month's probation. During this month they received further practical instruction and thereafter had lectures at least once a week. This training and the interest shown in the *aides* by the Red Cross staff produced excellent results and in the months of May and June all our *aides* gained very high monthly assessments; several also passed their examinations into further training schools.

Kinesiotherapists: This very important commitment caused much anxiety. The kinesiotherapists were recruited from nurses under training (male and female) and in Rabat were given an eight months' intensive course of anatomy, physiology, pathology and the principles of human kinesiology. In Meknès most of them were Spanish-speaking and had an appreciable difficulty in speaking French fluently; they had all come from other parts of the country and most had had to leave their families behind. They had many problems to contend with.

Conclusions

The final results in 4,029 patients registered at Meknès are given in Table 1. I am certain that the Moroccan authorities will be able to supervise and treat the relatively few patients requiring further assistance.

Table 1

The results of treatment at El Mers

Results of hand paralysis in 1,301 patients		
Hands normal	1,278	
Weakness of opponens only	4	
General intrinsic weakness	19	
Residual weakness or spasticity of legs in 3,186 patients		
Minimal weakness (M = 4)	229	(7·2%)
Weakness requiring splint	47	(1·5%)
Minimal spasticity	116	(3·6%)
Moderate spasticity requiring splint	33	(1·0%)
Severe spasticity with deformity requiring corrective surgery	31	(1·0%)
Legs normal	2,730	(85·7%)
Final results as at July 1 1961		
Total number of patients registered	4,029	
Patients remaining under treatment		
(*a*) As in-patients 8 } 62		
(*b*) As out-patients 54 }		
Patients requiring medical review	205	

When I left Meknès on July 1 the position regarding resettlement was still unsatisfactory. Admittedly a start has been made with the preliminary schooling course in El Mers. However, when 20% of fit men are unemployed, special legislation and provisions will be required if those who are disabled are to find work.

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