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# THE USE OF LIGHT HYPOTHERMIA IN ACUTE LIFE-THREATENING POLIOMYELITIS

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In the past few years the use of hypothermia has increased in the treatment of various conditions, as distinct from its use during surgical operations. As far as we are aware there have been no published reports from British sources of the use of this technique in acute poliomyelitis, though Miorner *et al.*[1] successfully treated a case of poliomyelitis with hyperpyrexia. Mihaly Janos[2] suggested that cooling, if used early, may reduce the degree of residual paralysis. In some 12 patients so treated Janos[3,4] considered that the return of muscle power in 9 was more rapid than would normally have been expected. Russell[5] considered that hypothermia would be detrimental in acute bulbo-spinal poliomyelitis, because of the adverse effect of the cold itself on nerve cells when in an already damaged condition.

Ross[6] and Gray[7] have demonstrated, on experimental animals, that severe hypothermia could only be maintained for a short period of time if a successful outcome was to be expected. Knocker[8] has presented evidence which suggests that severe hypothermia causes considerable disturbance to the liver and other intestinal organs, though Rosomoff[9] denies this. It would appear from current literature (Delorme[10]) that hypothermia may cause derangement in the normal biochemical relationships in the body; the majority of these derangements being apparently of unknown origin. It may be assumed that hypothermia, possibly even 'light' hypothermia, as defined by Delorme,[10] is not without certain inherent dangers when used over a period of some days.

Certain types of life-threatening poliomyelitis (Lassen[11]) carry a high mortality, and it was considered that, though hypothermia may be dangerous, the risks specific to certain cases of acute bulbo-spinal encephalitis were so great, and the residual degree of paralysis in those surviving so appalling, that hypothermia was justified; firstly, as a means of saving life and, secondly, as a possible means of reducing the residual paralysis.

Lassen[11] and Engstrom[12] have both reported very high mortality rates in cases of acute bulbo-spinal polio-encephalitis with hyperpyrexia, and our past experiences have been similar.

## METHODS

Cooling was effected by a water bed under the patient. Iced water was run in by gravity. Shivering was prevented by giving intravenous promethazine, 25 mg., chlorpromazine, 25 mg., and pethidine, 50 mg., which was repeated if shivering recurred. In all cases, once the temperature had been brought down to the required level, a maintenance dose was rarely required.

It was found that, provided the whole of the back was cooled, it was not necessary to apply cooling agents to the other parts of the body. The time to reach the required temperature was about 5 hours and was therefore slower than is usually convenient in an operating theatre.

The water bed was emptied when the temperature had dropped to within 2° - 3°C. (3° - 5°F.) of the required level, and iced water was again run in if the temperature showed signs of rising. In order to control ventilation and electrolyte balances in the acute phase, daily blood samples were taken for the estimations of pH., $PaCO_2$, electrolytes, and PCV. The body temperature was taken with a low-reading thermometer in the rectum. Blood pressure, pulse rates, and temperatures were taken half-hourly.

One patient was cooled down to 27·5°C. (81·5°F.) for 24 hours. This patient died at the end of 48 hours when the temperature had been allowed to rise slowly to 35°C. (95°F.). Another 5 were cooled to 32° - 34°C. (89·6° - 93·2°F.) for periods of 2 - 6 days. One was kept at this temperature for 18 days. Another patient was kept at 35·5°C. (95·9°F.) for 12 days. Six patients were maintained afterwards at a normal temperature for a week or so by keeping them on the water bed filled with tap-water.

The following are two illustrative case histories:

*Case 1*

European male, aged 24. Height 6 ft. Weight 12 st. Admitted on 18 May 1957 from a hospital 100 miles away where intermittent positive-pressure respiration had been instituted on 17 May 1957.

*On admission.* Complete paralysis of all limbs, intercostals, diaphragm, and pharyngeal muscles; and diplopia, internal strabismus and nystagmus were present. Temp. 39·5°C. (103°F.). Blood pressure 280/140 mm. Hg. Arterial $CO_2$ tension 39 mm. Hg.

*Progress.* The temperature varied between 39·5° - 40·5°C. (103° - 105°F.) for the next 5 days. The patient was cooled on the sixth day after admission to 31° - 34·5°C. (88° - 94°F.). This was continued for the next 6 days, and thereafter the temperature was maintained at 36·7°C. (98°F.) for a further 2 weeks. The hypertension was controlled during the cooling period with 'ansolysen' and 'mecamylamine'. Swallowing returned on 10 September 1957, i.e. 4 months after the onset of the disease.

*Case 2*

European female, aged 20. Height 5 ft. 6 inches. Weight 10 st. Admitted on 30 August 1957 on the third day of the illness.

*On admission.* Temp. 39·5°C. (103·2°F.). Stiffness of neck and back, considerable agitation, and weakness of biceps on both sides and of the abdominal muscles were present. Vital capacity 1,900 ml. Vomiting persistent. Respiration became paradoxical soon after admission. Cooling was begun on the day of admission and was maintained at 31° - 34·5°C. (88° - 94°F.) for 5 days from the second day after admission.

*Progress.* During the subsequent course of the illness her vital capacity fell to 500 ml. and necessitated artificial ventilation in a tank. She then developed ocular motor palsies, facial paralysis, and bulbar paralysis necessitating tracheotomy and intermittent positive-pressure respiration. She was comatose for 24 hours at this juncture. Four days after cessation of cooling, she developed acute dilatation of the stomach. Some 4 months later she was

flown to the United Kingdom. At this time both her legs were quite powerful, but the arms were severely paralysed with movements present only in the fingers, and she had no measurable vital capacity.

## RESULTS

Of the 9 patients treated by cooling 3 died. The 3 deaths occurred among the 8 who had bulbo-spinal paralysis and who were treated with intermittent positive-pressure respiration. The ninth patient was treated in a tank respirator.

Three patients showed hyperpyrexia, with sustained temperatures or rising temperatures above 40°C. (104°F.) and lasting from 3 to 10 days after the onset of paralysis, and this was also associated with hypotension or hypertension. The remaining 6 patients all had temperatures above 39°C. (102°F.) and this was associated with hypertension, paralytic ileus, and pulmonary thrombosis in 1, acute dilatation of stomach, and coma in 2, and hypotension in 1. The mortality rate for these complications, as given by Lassen,[13] is shock 65%, hyperpyrexia 91%, and hypertension 42%.

All 3 of our patients showing hyperpyrexia survived, whereas previously all our adult patients with hyperpyrexia had died. It is perhaps of significance that all 3 patients in this series who died were inadvertently allowed to cool below 31°C. (88°F.) for short periods, and certainly 1 who died was allowed to cool too low to 26·1°C. (79°F.), and 1 other patient was cooled for too long—18 days. Of the 6 who survived only 2 ever reached these figures.

*ECG Changes*

Twenty-four electrocardiograms were taken in 7 patients whose temperatures varied between 30° and 35°C. (86° - 95°F.). All showed a prolonged QT interval. Two showed a partial right bundle-branch block and 2 showed occasional ectopic beats. Large wide P waves and marked clockwise rotation of a vertical heart was observed in 1 of the patients showing partial right bundle-branch block, and the changes were present at 34·3°C. (93·7°F.) and reverted to normal when the temperature reached 37°C. (98·6°F.). The other patient with partial right bundle-branch block, whose temperature had risen to 36·4°C. (97·5°F.), showed deep S waves in Lead I, deep Q waves in Lead III, plus a secondary R wave in VR with a clockwise rotation of a vertical heart. This patient died of an auricular thrombus extending into the right ventricle and both pulmonary arteries. Dundee *et al.*[14] recorded a patient dying of pulmonary embolism 13 days after freezing.

An injury-potential was seen in 2 cases, one at 30°C. (86°F.) and the other at 32·8°C. (91·2°F.). The injury-potential was well marked in the former, and was associated with a respiratory and metabolic acidosis, pH 7·28, and $PaCO_2$ 51·8 mm. Hg. The latter patient also had a moderate degree of respiratory acidosis, pH 7·37, and $PaCO_2$ 49·7 mm. Hg.

*Pulse Rates*

All patients showed considerable tachycardia in the pyrexial phase, i.e. 100 - 160 (mean 134). With one exception all pulse rates slowed on cooling, the mean degree of fall being 40 per min.

## DISCUSSION

We have used light hypothermia in 9 patients. Of these 3 died, 1 on the 6th, 1 on the 9th, and 1 on the 26th day of illness. In the prolonged cooling, which may be necessary in the treatment of acute life-threatening poliomyelitis, there is difficulty in defining certain fundamental points, i.e. when to cool, the degree of cooling, the duration of cooling, which cases to cool, and whether the cooling should be consistently to the same degree. Reference to the literature gives little help in solving these problems, and our criteria were therefore understandably somewhat empirical.

On theoretical grounds, from the evidence presented by Rosomoff,[9] it might appear that the reduction in oxygen consumption and metabolism, the reduction in volume of the brain (and presumably the spinal cord), and possibly an effect on the virus itself would justify the use of this technique.

Although 4 of the cooled patients showed a considerable prolongation of the time during which paralysis was spreading, we do not consider that there was any startling evidence that hypothermia in any way reduced the degree of paralysis in the acute phase. We feel that the numbers are far too small to say whether hypothermia reduced the degree of residual paralysis. Certainly in our cases the evidence was not strong enough to be convincing.

It is considered that severely ill patients with acute life-threatening poliomyelitis should not be cooled below 32·2°C. (90°F.). No useful purpose seems to be served in prolonging cooling for more than 5 or 6 days. Cases of hyperpyrexia in adults should be cooled. We are unable to comment on the questions when to cool and whether the cooling should consistently be to the same degree.

*Degree of fall of temperature.* Fall of temperature varied from 3·3° to 11°C. (6° - 20°F.). Times taken to reach the lowest temperatures were 4 - 18 hours.

In only 1 patient was the degree of 'after drop' alarming. In this case the cooling mass was withdrawn at 37·2°C. (99°F.) and thereafter the temperature fell another 7·2°C. (13°F.), i.e. to 30°C. (86°F.) during the next 3½ hours. This patient was an overweight male—height 6 ft., weight 16½ stone.

## SUMMARY

The use of light hypothermia is described in 9 cases of life-threatening bulbo-spinal poliomyelitis; of these 3 died. The reasons for using this technique and some of the difficulties encountered in planning the application of hypothermia are briefly described.

It is considered that hypothermia reduced the mortality rate in severe bulbo-spinal paralysis.

There was little evidence that the degree of residual paralysis was reduced.

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