Three weeks after discontinuation the eye-grounds were normal and x-ray examination of the skull showed normal sutures. There were no sequelae and the boy was doing very well.

DISCUSSION

The first two episodes of increased intracranial pressure occurred in close connexion with nalidixic acid medication. No other drugs were involved. The latter patients (Millichap, 1959; Fields, 1961; Opfer, 1963; O'Doherty, 1965), with corticosteroids (Doez and McKay, 1959; Valentine, 1959; Laurence et al., 1960; Greer, 1963; Walker and Adamkiewicz, 1964), and with toxic doses of vitamin A (Josephs, 1944; Oliver, 1958; Marie et al., 1963; Persson et al., 1965). A bulging fontanelle as a side-effect of nalidixic acid, however, has not previously been described in the literature, though Walker et al. (1966) reported that one of their patients, a 9-year-old girl admitted to hospital after a car injury, developed a slight papilloedema while receiving nalidixic acid for a urinary tract infection. Details were not given, but this suggests a drug effect, since the papilloedema disappeared when the drug was discontinued. Furthermore, the manufacturers (Winthrop Company) have informed us that in their files they have four recent reports on intracranial hypertension during nalidixic acid therapy in children. However, in none of these cases was a connexion with the medication proved and none of them has been published.

The real incidence of intracranial hypertension after nalidixic acid is not yet known. It is recommended that the possibility of this side-effect be kept in mind during treatment with the drug.

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REFERENCES


Mendelson's Syndrome: Its Treatment by Tracheostomy and Hydrocortisone


Hall (1940) described the cases of 15 patients who had inhaled vomitus during or after anaesthesia for childbirth. He described two types of case: (1) those patients in whom the inhaled vomitus consisted of solid material; this caused mechanical obstruction of the airways; and (2) those in whom the inhaled vomitus was entirely liquid; these latter patients developed marked cyanosis, tachycardia, and tachypnoea several hours later.

Mendelson (1946) also noted the two types of reaction, depending on the quality of the vomitus, which had been noted by Hall. When the vomitus contained solid material there was an acute airway obstruction with massive collapse of the lung. When the vomitus was liquid there developed, after a latent period of one to two hours, a syndrome of dyspnoea, cyanosis, tachycardia, and in Mendelson's cases generalized bronchospasm. Mendelson was able to cause a similar syndrome in rabbits by instilling either N/10 hydrochloric acid or acid gastric juice into the tracheobronchial tree. When distilled water, neutralized gastric juice, or 0.5% sodium bicarbonate solution was used the syndrome did not develop (Mendelson, 1946).

Others have confirmed that pulmonary oedema may develop after the inhalation of acid gastric contents (Hartzell and Mininger, 1946; Parker, 1954; Hausmann and Lunt, 1955).

The presence of oedema at the blood/gas interface in the lungs may upset gaseous exchange with the atmosphere, and if extensive it may be fatal. It is now recognized that tracheo-

stomy by reducing the respiratory dead space will improve the efficiency of alveolar ventilation (Shackleton, 1959). Though this operation has been employed in the management of patients who have inhaled vomitus, it has been for the relief of acute airway obstruction due to the impaction of solid undigested food in the larynx and upper trachea (Merrill and Hingson, 1951). There is a dearth of case reports in which a tracheostomy has been employed to reduce the respiratory dead space in patients with Mendelson's syndrome. One such case is reported.

CASE REPORT

A 31-year-old primigravida was admitted in labour at 42 weeks. Owing to incoordinate uterine action labour was prolonged, and after 32 hours she was delivered by caesarean section under endotracheal general anaesthesia. Operation and anaesthetic were uneventful.

About 30 minutes after returning to the ward she had a bout of vomiting with transient cyanosis. The mouth and pharynx were sucked clear of vomit, the colour improved, and her condition appeared satisfactory. Two and a half hours later she became very cyanosed with stertorous breathing. The blood pressure was 130/80, but the pulse rate had risen to 160. There were crepitations over both lung fields. The patient was unconscious. The pupils were equal and widely dilated.

A further bout of faecal vomiting occurred at this stage. The airway was sucked clear above the glottic opening and oxygen given under pressure from an anaesthetic machine, but without any material improvement in the patient's colour. She was transferred to the theatre, where oxygen was given, the trachea was intubated, and tracheal suction was performed, some vomitus being obtained from the trachea. A further bout of faecal vomiting occurred as these procedures were being carried out.

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It was then three and a half hours after the caesarean section and approximately three hours after the first episode of vomiting. She was given 100 mg. hydrocortisone intravenously and tracheal suction was repeated, but no further vomiting was obtained from the time the catheter was withdrawn. The next morning, peritoneal oedema fluid welled up from the endotracheal tube. Oxygen was given under pressure through the endotracheal tube, with a rapid improvement in the patient's condition. She became very active, the muscle tone increased, the jaw and pharyngeal muscles tightened, and she resisted laryngoscopy. She appeared to regain consciousness, so the endotracheal tube was removed.

Though conscious, she continued to be very distressed. She was still very cyanosed, her respiration was mainly diaphragmatic, and there was a tracheal "tug" present on inspiration. It was thought that in addition to the pulmonary oedema recurarization might have occurred. An injection of 0.6 mg. of atropine sulphate followed by 2.5 mg. of neostigmine given intravenously brought an improvement in respiratory movements.

Her blood pressure was 110/85, and the pulse was regular between 160 and 170 beats a minute. She was very restless and orthopnoeic, insisting on sitting bolt upright on the operating-table, as in this position she felt least distressed. Oxygen was given by mask from an anaesthetic machine, with slight diminution in her cyanosis. One hour after the first injection of hydrocortisone a second 100-mg. dose of this hormone was given intravenously, followed by an intramuscular injection of 2 ml. of Crystacyclin (benzylpenicillin and streptomycin). For the next hour there was no change in her condition. As the suction catheter was withdrawn pink frothy oedema fluid welled up from the endotracheal tube. Oxygen was given under pressure through the endotracheal tube, with a rapid improvement in the patient's condition. She became very active, the muscle tone increased, the jaw and pharyngeal muscles tightened, and she resisted laryngoscopy. She appeared to regain consciousness, so the endotracheal tube was removed.

After premedication with atropine sulphate 0.3 mg., under general anaesthesia (nitrous oxide, oxygen, and halothane) given through a No. 9 Magill endotracheal tube, tracheostomy was performed after dividing the thyroid isthmus. A Magill size 8 Oxford tracheostomy tube was inserted into the trachea.

At the end of this procedure her systolic blood pressure had dropped to 80 mm. Hg. Her colour was much improved and she no longer required an oxygen-enriched atmosphere to breathe, to avoid becoming cyanosed. Her lungs were drier.

A third injection of 100 mg. of hydrocortisone was given intravenously one hour after the second, and this was followed by a similar dose given intramuscularly, so that in a period of three and a half hours since the onset of pulmonary oedema she had had 400 mg. of hydrocortisone parenterally.

During the next 30 minutes her systolic blood pressure gradually rose to 100 mm. Hg and the pulse rate slowed to 136. She was transferred to the ward to be nursed in a steam tent. During this phase the trachea was sucked out each hour routinely, and more often if there appeared to be mucus in the airway. Crystacycin 1 ml. was continued 12-hourly and hydrocortisone 100 mg. six-hourly.

On the following day the intramuscular dosage of hydrocortisone was reduced to 75 mg. six-hourly. On the second day it was reduced to 50 mg. six-hourly, and the Oxford tube, which had been inserted when the tracheostomy was performed, was replaced by a metal tube. On the third day the dosage of hydrocortisone was reduced to 50 mg. 12-hourly, and it was stopped altogether on the fourth day.

The tracheostomy tube was removed on the fifth day, and convalescence thereafter was uneventful. The patient went home on the 17th day after caesarean section.

**DISCUSSION**

Many different treatments have been employed in the management of established Mendelson's syndrome; among them the use of cortisone was suggested by Hausmann and Lunt (1955).

They thought that the obstetric patient is liable to have adrenal insufficiency immediately after delivery, because of the loss of the placenta as a source of cortisone-like substances. They gave two patients with aspiration pneumonitis cortisone, which resulted in marked improvement. It is possible that corticosteroids will benefit these patients even in the absence of adrenal insufficiency, by inhibiting the inflammatory reaction in the bronchial and bronchiolar walls which is evoked by the acid gastric juice. The outpouring of tissue fluid which accompanies inflammation (MacCallum, 1942) will only aggravate the airway obstruction at bronchiolar and alveolar level. Further, the use of cortisone may be expected to reduce the bronchoospasm, which was stated by Mendelson (1946) and by Parker (1954) to be a feature of these cases.

Several authors, notably Hall (1940) and Parker (1954), have described the pathological changes in the lungs in severe cases of Mendelson's syndrome. In their papers one reads of "diffuse wet consolidation with air present only at the extreme apices" and "massive oedema of all lobes of the lungs." It is evident that such extensive changes will occur only in the fatal cases. Less severe cases will presumably have similar but less extensive lesions.

Changes such as these may be associated with certain alterations in pulmonary function. (1) The presence of oedema in the alveoli will cause impairment of diffusion capacity at the gas/blood interface. (2) If the circulation continues in the oedematous lung there will be a venoarterial shunt due to the perfusion of nonventilated alveoli. (3) The waterlogged lung will be less compliant than the healthy lung tissue, so that the work of breathing (and the oxygen consumption of the respiratory muscles) will be increased.

The administration of oxygen, by increasing the alveolar-capillary oxygen gradient, may improve the patient's condition, but because of the shunt effect mentioned above cyanosis may persist.

Alveolar ventilation is given by the formula:

\[ V_a = (V_T - V_D) \cdot f \]

Alveolar ventilation = (tidal volume – dead space) × respiratory rate.

As tachyphoea develops the alveolar ventilation will also increase. This is achieved at the cost of an increased oxygen consumption in the respiratory muscles, which are already working in a hypoxic environment. It seems undesirable that part of the limited amount of oxygen available to the respiratory muscles should be expended in unnecessarily moving a volume of air equivalent to the dead space during each respiratory cycle.

Tracheostomy causes a reduction by one-half in the respiratory dead space, and the improvement in alveolar ventilation which occurs after this procedure appears to make it worthwhile in a severe case of respiratory failure due to pulmonary oedema after inhalation of vomitus.

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