following an attack of tonsillitis. With appropriate treatment recovery was complete in 48 hours.

**Premedication.** Adequate pre-operative medication is a *sine qua non* for a successful anaesthetic. This applies particularly to pre-operative sedation for children. Children from the ages of 2-13 years are, as a group, more difficult to control than adults, and those from 7-13 years in particular, if normally well developed and in good health, are usually very difficult subjects to induce smoothly and to maintain on an even anaesthetic plane. It has been our endeavour, therefore, to find a suitable drug for use in these cases, one which will adequately quieten the child before the operation and yet at the same time not interfere with the subsequent anaesthesia and post-operative recovery. It is, of course, not necessary to emphasize the importance of preventing or diminishing in the child’s mind the psychic trauma associated with any operation. For this reason, too, no pre-operative enema is given to our cases.

Of the drugs employed nembutal and seconal sodium were found most useful and of these the former is used as a routine. Here it must be emphasized that the dosage must be high. In the majority of cases, nembutal causes an initial phase of restlessness, when the child tosses to and fro and would seem to be fighting sleep. If the dosage is correct, this phase passes over rapidly and sleep or marked drowsiness supervenes; but if insufficient, the restlessness persists and no benefit at all will be derived.

Children tolerate large doses of nembutal orally very well, this being due to their relatively high metabolic rate and so far the following dosage has proved to be most satisfactory:

<table>
<thead>
<tr>
<th>Age (in Years)</th>
<th>Dose (in Grains)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>1/-11</td>
</tr>
<tr>
<td>3</td>
<td>11/2-2</td>
</tr>
<tr>
<td>4</td>
<td>11/2-2</td>
</tr>
<tr>
<td>5-7</td>
<td>2/-2</td>
</tr>
<tr>
<td>8-9</td>
<td>2/-2</td>
</tr>
<tr>
<td>10 upwards</td>
<td>2/-3</td>
</tr>
</tbody>
</table>

The dose is administered 1½ hours before operation. As nembutal in powder form has a very bitter taste and children under the age of five years cannot easily swallow capsules, the powder from a capsule is often administered sandwiched between layers of honey or jam and washed down with water. Nembutal elixir, though most palatable, has too small a dosage per quantity administered (½ gr. in 1 fl. oz.) for satisfactory usage. Older children are given the capsules with the ends pierced to aid absorption.

Following administration of the sedative, the room is darkened, the child tucked into sleep and no talking is encouraged. A hypodermic injection of atropine sulphate gr. 1/150-1/100 is given half an hour before the operation. This may rouse the child who, however, falls asleep again or relapses into a drowsy state.

This premedication not only ensures that the child leaves the ward in a peaceful condition, with a much less harassed parent in the background, but also provides the benefits of reduced mucus formation or lessened post-operative nausea and vomiting. By contributing to the subsequent ease of anaesthesia there must also obviously be less respiratory and cardiac strain.

Opposition or disinclination to adopt this procedure is based as a rule on two points: One is relatively minor, viz. the fact that the patient falls into a deep sleep on return to the ward and the resumption to full consciousness is delayed. This is no disadvantage if complete haemostasis has been ensured by the surgeon. The other is at present more important. More attention is required from the nursing staff both pre- and post-operatively. This is, however, more than compensated for by the pleasant and peaceful atmosphere that now accompanies our ‘tonsil days’. Any extra effort to ensure that this routine is carried out will be well repaid.

**SUMMARY**

1. ‘Open’ endotracheal anaesthesia without expira­tory valve or re-breathing bag is described and recom­mended as a routine procedure for the removal of tonsils and adenoids in young children.

2. The use of adequate premedication with nembutal is discussed. The importance of high dosage is emphasized.

My sincere thanks are due to Dr. A. J. de Villiers, E.N.T. Specialist, for his interested cooperation and many valuable suggestions.

**REFERENCES**


**MESANTOIN TREATMENT OF EPILEPSY**

G. M. MALAN, M.B., CH.B., AND G. G. HARRISON, M.B., CH.B.

At the Jan Kriel Home for Epileptics at Kuils River, C.P., six cases of grand mal epilepsy were treated with 3-methyl 5, 5 phenylethyl hydantoin (mesantoin). They were chosen as suitable cases because the recognized drugs such as gardenal (phenobarbitone) and epanutin (sodium diphenylhydantoinate) had not brought about much improvement.

Although the literature reports mesantoin as less toxic than other drugs, one of our cases developed a rash and fever, with cessation of symptoms on stopping the drug; one developed a rash which disappeared although the drug was not stopped, and one case proceeded to a fatal ending with aplastic anaemia. Loscalzo first reported successful results in June.
1945. Clein\textsuperscript{2}, Kozol\textsuperscript{3}, Fetterman and Friedman\textsuperscript{4} all agree that a promising drug has been introduced and that approximately 60\% of cases showed improvement. In a total of over 300 patients only one case of fatal aplastic anaemia has been reported (Aird\textsuperscript{5}). The authors all state emphatically that toxic effects were much less in evidence than with other preparations having any appreciable effect.

The case reported here shows the need of careful control of the blood picture when using any anti-convulsant drug. An unmarried female patient aged 27 years had been treated in the Institution for seven years and had been admitted with the history of 14 years' treatment with no improvement. The number of attacks varied from three to 11 per month but the patient was deteriorating physically and mentally. She was dull and non-co-operative and was becoming withdrawn from reality.

In August 1948 she was started on mesantoin gradually, epanutin being withdrawn as mesantoin was increased until by October 1948 she was getting mesantoin gr. 1\(\frac{1}{2}\) q.i.d.

Her general condition improved and although the attacks of epilepsy did not stop, she seemed more rational and cheerful. In January 1949 she developed a sudden high temperature associated with petechial and vaginal haemorrhages. The drug was stopped immediately, but the condition did not improve and the patient was admitted to hospital. It was brought to our notice then that since June 1948 there had been amenorrhoea. Before that time the menstruation had been regular, 3-5 day type.

5 February, 1949. Her temperature on admission was 100.5\(^\circ\) F. She was very pale, obviously anaemic and covered from head to toe with small purpuric spots. The majority were of pinhead size, some a little larger. She was also bleeding per vaginam, a slow, steady ooze. She was extremely apathetic, very drowsy, although easily aroused. At the entrance of the left nostril there was a crusted, septic ulcer, the size of a sixpenny piece. There were petechiae in the conjunctivae of both eyes. There were also petechiae on the posterior pharyngeal wall, but no necrotic lesions. There was no palpable lymph adenopathy. Her pulse was 84 per minute, regular and of a collapsing character. Her blood pressure was 105/60 mm. Hg. The heart was clinically not enlarged. There was a blowing systolic murmur heard maximally at the apex.

**Pulmonary System.** Nothing abnormal was detected.

**Abdomen.** Liver and spleen were not palpable.

**Central Nervous System.** This was not tested adequately as the patient was not co-operative.

**Optic Fundi.** The retinæ were pigmented, but there were no haemorrhages or petechiae.

**Peripheral Nervous System.** This was normal.

There was a firm, warm, indurated red area on either side of the anus, the one area being ulcerated and with a purplish, necrotic centre. They were of the character of bilateral ischiorectal abscesses. Her blood, on examination, showed the following: E.S.R., 135 m.m. in the first hour; Packed cell volume, 18\%; Red Cell count, 2.8 million per c.mm.; White cell count, 2,500 per c.mm.

**Differential White Cell Count.** On two smears of peripheral blood only 60 white cells were seen. These were all lymphocytes; no polymorphs were seen. Red cells showed anisocytosis, some poikilocytosis and polychromasia. A sternal puncture was done and examination of the marrow revealed mature red cells and lymphocytes. No cells of the granulocyte series were seen and no immature cells of any type. Microscopy of urine revealed red cells.

The patient's blood was grouped and found to be group 4\(\frac{1}{0}\) Rh positive. A blood transfusion was put up, preceded by a dextrose-saline drip. The patient was also given penicillin 100,000 units intravenously followed by 100,000 units intramuscularly every three hours. Pyridoxine 100 mg. twice a day and luminal grains 1 t.d.s. The patient had a rigor, the temperature rising to 104.8\(^\circ\) F, one hour after a transfusion was commenced. The blood transfusion was discontinued. Half an hour later the temperature dropped to 102\(^\circ\) F.

Two hours later, after checking her blood group and compatibility again, another transfusion was set up with fresh blood, preceded by a saline-dextrose drip of approximately 100 c.c. An hour later the patient again had a severe rigor, her temperature rising to 106.4\(^\circ\) F. Transfusion was again discontinued. Her temperature dropped within the next hour to 104.8\(^\circ\) F. and after sponging, to 103.8\(^\circ\) F. At this time the patient had an epileptic fit. On 6th February the packed cell volume was 19\%. She had another epileptic fit that afternoon. A transfusion was put up at 2 p.m. preceded by morphine gr. 3, atropine gr. 1/100. Two pints of blood were transfused without reaction. Streptomycin 1 gm. b.d. and pentoxylate 5 c.c. per day were added to the therapy.

7 February. The peripheral blood smear showed as before red cells and a few lymphocytes but no polymorphs. Packed cell volume, 27\%.

8 February. Packed cell volume, 27\%. Peripheral blood again showed no polymorphs. Another pint of blood was given to which the patient reacted with a rigor. Blood was discontinued for an hour and then recontinued, preceded by morphine gr. 3, atropine gr. 1/100, and completed without reaction.

On 9 February the packed cell volume was 30\%. A peripheral blood smear showed red cells, lymphocytes and one degenerate polymorph. On 10 February the lesion on the left nostril was seen to be spreading. An attempt was made to give another pint of blood but after approximately 3 pint the patient had four brief epileptic fits, and her temperature rose rapidly by leaps and bounds to 107\(^\circ\) F. The blood was discontinued, the patient sponged and given an injection of sodium gardenal gr. 1/4. By the following morning the patient's temperature had dropped to 102\(^\circ\) F. and that night was down to 96\(^\circ\) F. Examination of the throat now revealed large sub-mucosal haemorrhages on the post-pharyngeal wall.

On 12 February the packed cell volume was 27\%. The peripheral blood, as before, showed lymphocytes and red cells but no polymorphs. Another pint of blood was given but had to be discontinued because of another rigor. On the night of 13 February the patient commenced bleeding (a constant heavy ooze)
from the nose. Adrenalin swabs failed to stop the bleeding. Various other methods, such as cautery, were considered but abandoned because of the low white cell count and the large area from which haemorrhage was taking place; 2 c.c. of koagamin were given intramuscularly every three hours for the above purpose. That same night, her abdomen became distended and tumid and she complained of colicky abdominal pain and shortly after commenced to pass clots of dark blood per rectum. Her packed cell volume on 14 February was 21%, and the peripheral blood was as before. Another pint of blood was given and completed without reaction. On 15 February the nasal and intestinal haemorrhage continued. She was still bleeding per vaginam as on admission.

On the night of 16 February a cut-down was done and another pint of blood transfused. The packed cell volume at that time was 30%. The pint of blood was followed by a vacoliter of dextrose in 5% saline. The following morning, in spite of all treatment the patient went steadily downhill, developed Cheyne-Stokes breathing and died at 10 a.m. In all the patient received a total of 7½ pints of blood.

SUMMARY

1. Mesantoin is a useful anti-convulsant drug.
2. Cases of grand mal epilepsy which do not improve on any of the recognized drugs have done well on mesantoin.
3. Careful control of the blood picture must be maintained. We suggest fortnightly full and differential blood counts. The proportion of cases which go wrong may be very small but that does not preclude the utmost care being taken.

REFERENCES


BILATERAL MICROPHTHALMIA

REPORT ON A CASE

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Among the rarer causes of congenital blindness is the condition of abnormal smallness of the eyeballs (microphthalmia), ranging to complete absence of the eyeballs (anophthalmia). High degrees of microphthalmia are clinically called anophthalmia, but true anophthalmia with no trace of an eye primordium on either side is extremely rare. Even when the eyeballs appear to be absent, the bony orbit, the muscles of the orbit and the lacrimal apparatus are well developed and the optic nerve is usually present as a fine thread. The above defects are due to an abnormality in embryological formation which manifests itself almost certainly in the earliest stages of the formation of the optic vesicles. There is a tendency to heredity, since microphthalmia and anophthalmia are known to run in families, and these abnormalities are not due to disease processes in the embryo or foetus (Schwalbe, 1906). Further, the study of an anophthalmic strain of mice (Chase, 1941) has proved that the condition is due to genetic constitution.

If the optic vesicles fail to bud out from the forebrain the condition of anophthalmia is produced in which the retina, the ciliary body, the iris and the optic nerve are absent. The lens, which is formed from the surface ectoderm in response to a stimulus received from the neural ectoderm of the optic vesicle, does not develop because its organizer has failed to produce the stimulus at the proper time. The mesodermal elements of the eye—choroid, sclera, extrinsic muscles and orbit—will, however, develop since they are self-determining and it is only their size which is regulated by the presence of the optic vesicle (Mann, 1937). In microphthalmia the optic vesicles have budded out but are retarded or interfered with in their development. An element of the globe of the eye is always present among the orbital contents; but it may be so small that it is not obvious to the finger on palpation. A rudiment of the lens will also be found in cases of microphthalmia, provided that the optic vesicle reached the cup formation stage and was large enough to come into contact with the surface ectoderm.

The present example of microphthalmia was brought to my notice through the kindness of Dr. Bromilow-Downing, Medical Superintendent of the Frere Hospital, East London, who supplied the clinical history and sent the brain of the abnormal child to the Anatomy