employed early and in sufficient dosage. For continuation of treatment, Bull's diet is the basis.

With regard to pentothal sodium in eclampsia: it is my belief that it reduces the number and severity of the fits. Reduction of mortality from eclampsia depends upon its prevention. The nature of the disease is such that once convulsions occur irreparable damage may be inflicted upon important organs in a short period of time. Mortality from eclampsia can be lessened by reducing the incidence of the disease; once the disease occurs, however, fatalities are inevitable. Eclamptics stand transport very badly.

Lastly, a note about toxic accidental hæmorrhage: as I see it, there are two types of case. Toxic hæmorrhage may develop in the woman who has had toxæmia or hypertension and it may occur in the patient who has shown no signs of toxæmia nor of hypertension. Immediate or early complications, such as shock and symmetrical necrosis of the kidneys, may be severe in both cases, but remote sequelæ, such as vascular sclerosis, recurrent toxæmia, chronic hypertension and apoplexy are, of course, more likely to occur in the toxæmic case. Permit me to emphasis some points in treatment, elaborated by the late O'Donel Browne in his last paper to this Academy.

Try to avoid the occurrence of toxic hæmorrhage in hypertensive and toxæmic patients by adequate rest, by induction or by Cæsarean section and by the recognition of the earliest signs of placental separation.

Follow-up defaulters from the Antenatal Clinic.

Provide more antenatal beds.

Reduce the time-lag between the onset of the signs of toxic hæmorrhage in the patient's home and the institution of treatment in hospital.

Appreciate the value of compatible transfusion. The patient suffering from toxic hæmorrhage quickly becomes anæmic; in shock, the peripheral arterioles are dilated and the circulation is slowed down; the heart muscle and brain lack oxygen; the circulation in the kidneys is brought almost to a standstill; a small postpartum hæmorrhage may prove fatal; fibrinogen content may be reduced. Surely, then transfusion is strongly indicated. So far as I know, there is no scientific basis for withholding blood. Don't kill the patient by giving too much so-called normal saline.

Administer oxygen freely.

In the cases which do not respond to restorative measures, think of suprarenal hæmorrhage. Cortisone and *nor*-adrenaline may be indicated.

Dr. MICHAEL SOLOMONS: I was Assistant to the late Professor O'Donel Browne when this, his last Report, was being compiled, and I know how appreciative he would have been to-night about his Report. He was deeply interested in the toxæmias, and two important points of his treatment were the setting up of a special toxæmia clinic and the increased use of Cæsarean section in cases which did not respond to other treatment. At first he had viewed Dr. Falkiner's section incidence of these cases with criticism, but as time went on he changed his views, and if you study his figures for the four years of his Mastership you will see that the incidence for Cæsarean section in the treatment of toxæmia was increasing year by year. There are few questions to answer regarding the Report, nor are there any comments requiring discussion. Dr. Dundon brought up the point regarding the details of postmortem reports in atelectasis. Postmortems were carried out on all cases where such a cause of death was certified. Dr. O'Donel Browne did not consider biochemical tests of much assistance in these cases. We have four consultant anæsthetists on the Rotunda staff, and one of these is always available day or night. My own personal criticism this evening is that I do consider the subject for discussion is very ill-chosen. What have we learnt from to-night's discussion? I do not believe we have learnt anything further about the ætiology or treatment. To conclude, the staffs of the Maternity Hospitals here are all doing their best, but there is always room for improvement.

ERRATUM

The Editor deeply regrets that a serious error in the production of Dr. Shepherd's graphs facing p. 210 in our May issue should have been overlooked. The following legend should have been set at the foot of the page on which the graphs appeared.

FIG. 1.—The heat loss to water at 0·6°C from the injured and normal fingers of E.D., B.T., R.McD. and A.R. Ordinate: Heat loss in cal. per 100 ml. finger per min. The marks indicate the 1,000 and 2,000 cal. levels. Abscissa: Time in minutes. The full width of each frame is 30 minutes. In each case the upper frame shows the responses of the injured finger. The numbers in the top left hand corner of the squares indicate the days after injury.