Hypoglycemia Associated with Symptoms in the Newborn Period

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ABSTRACT

Hypoglycemia in the neonatal period is a well-recognized phenomenon, but many authors have commented upon the infrequent association of symptoms attributable to it. Six infants were seen who appeared normal at birth but who, between 24 and 72 hours of age, developed apnea, irritability, lethargy, muscular twitchings and convulsions. Blood sugar concentrations of 10 mg./100 ml. or less were found in each case. The mothers of four of the babies had toxemia of pregnancy. Three babies were premature. The hypoglycemia was self-limiting in all cases, and four of the babies recovered completely without sequelae. The other two showed evidence of permanent brain damage, but it is not known whether this was the cause of their symptoms or the result of the hypoglycemia. It is concluded that hypoglycemia may cause neurological symptoms in the newborn period and that treatment by glucose administration is necessary. Whether symptomless hypoglycemia requires treatment remains an open question.

IT HAS been known for more than two decades that blood sugar levels of newborn infants tend to decrease during the first few hours and days of life, and not infrequently reach levels that are considered hypoglycemic by adult standards.[1-10] In premature babies this neonatal hypoglycemia may be especially marked.[11-14] Many workers have also found greater decreases in the blood sugar levels of babies born to diabetic mothers[1, 12, 15-19] but there are also reports that blood sugar levels in babies of diabetic and non-diabetic mothers do not differ.[8, 20]

The blood sugar level in the neonatal period is so variable that it is not easy to define a range of normal values.[21] In addition, the length of the fasting period before the blood sample is taken and the method of blood sugar analysis used make it difficult to compare results reported by different workers. Many earlier authors used methods which estimated total reducing substances in the blood and which obviously did not reflect accurately the level of the blood glucose.

Many of the aforementioned authors reported little correlation between low blood sugar levels and symptoms of hypoglycemia. Blood sugar levels below 10 mg./100 ml. have been recorded without clinical signs.[4] Because of the apparent lack of correlation between low blood sugar levels and symptoms, a number of authors considered that the feeding of glucose to babies of diabetic mothers not only was ineffective but might even be harmful because of the dangers of overhydration and aspiration.[16, 18, 20] Other authors, however, considered that such symptoms as shallow breathing, cyanosis, listlessness, irritability, muscular twitching and convulsions were caused by hypoglycemia, and reported rapid alleviation of symptoms following the administration of glucose. Hartmann and Jaudon[1] reported five infants of diabetic mothers whose symptoms were apparently related to hypoglycemia and two more babies who died, in whom the association was less obvious. They thought that similar symptoms in otherwise normal newborn babies might sometimes be due to hypoglycemia.

Cornblath, Odell and Levin[22] described eight infants, all born to mothers with pre-eclampsia, who developed apnea, cyanosis, coma and convulsions associated with profound hypoglycemia on the second or third day of life. The administration of parenteral glucose rapidly relieved the symptoms in every case. In five of the infants the condition was benign and self-limited, although one baby required the administration of corticosteroids to control the hypoglycemia. Two of the babies were subsequently found to have brain damage and a third died from infection on the seventeenth day of life. It was of interest that after the acute symptoms had been controlled with glucose administration, several of the infants continued to show very low blood sugar levels but remained asymptomatic.

Cornblath, Baens and Lundeen[13] studied blood glucose levels in premature infants. Forty-five infants with signs of respiratory distress, apathy, or cyanotic or apneic spells after 48 hours of age were found to have lower blood glucose levels than 44 asymptomatic infants. In eight infants very low blood glucose levels were found (0-18 mg./100 ml.) and of these, seven had symptoms which were promptly relieved by parenteral glucose.

Within the last 18 months six infants have been seen at our hospital who developed apnea, cyanosis, convulsions and other symptoms on the second or third day of life associated with very low blood sugar levels.

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CASE REPORTS

CASE 1.—This baby boy was born after 38 weeks' gestation. His birth weight was 6 lb. 13 oz., and his length was 20 in. The mother, who had one other healthy child, was healthy and had shown no signs of toxemia during her pregnancy. She had apparently "craved for sugar" during the pregnancy. Her glucose tolerance test was normal.

The baby appeared quite normal at birth. Feedings were started at eight hours of age. He thrived until 60 hours of age when he suddenly became limp, cyanosed and somewhat tremulous, and had slow, shallow, laboured respirations. He refused to suck, and the Moro reflex was depressed. He was placed in an incubator, and the possibilities of intracranial damage or pulmonary infection were considered.

A radiograph of the chest showed a little diffuse cloudiness in the right lung. A lumbar puncture revealed a spinal fluid which was xanthochromic and contained 8 cells/c.mm.; the protein content was 100 mg./100 ml. and the sugar 10 mg./100 ml. A blood sugar estimation at the same time was 7 mg./100 ml.* The serum calcium was 4.3 mEq./l.; potassium 8.8 mEq./l.; the sodium and blood urea nitrogen were normal. No abnormalities were found in the urine.

A slow intravenous infusion with one part normal saline and four parts 5% glucose in water was started. Two hours later he appeared better; he was less cyanosed and his muscular tone had increased. Six hours later he had a generalized convulsion for which he was given paraldehyde and phenobarbital intramuscularly. He was also noticed to be sweating profusely. He remained limp, did not suck, and the Moro reflex was absent. A blood sugar level was 53 mg./100 ml. while the intravenous infusion was running. An electroencephalogram and an electrocardiogram showed no abnormalities. The following day he was still very limp and unresponsive. A random blood sugar estimation revealed a level of less than 10 mg./100 ml. On the fifth day of life the intravenous fluids were stopped and oral feedings were started by gavage every two hours. A mild degree of jaundice was present (serum bilirubin 7.7 mg./100 ml.). He began to improve, and on the sixth day of life he was able to take bottle feedings. By the eighth day a fasting blood sugar level was 44 mg./100 ml. When he was nine days old his general condition appeared very good, and he was discharged from hospital when two weeks of age.

At 4½ months of age he was found to have a severe iron deficiency anemia. A systolic cardiac murmur was also noted at this time. A fasting blood sugar level was 76 mg./100 ml. At six months of age he appeared to be well although some spasticity of the legs was suspected.

He was admitted to hospital when 10 months old for reassessment. He appeared to be severely mentally retarded. He could not sit without support and did not take any interest in his surroundings. He was found to have bilateral central lens opacities. There were no other abnormalities. Fasting blood sugar levels ranged from 47 to 73 mg./100 ml. The administration of L-leucine by mouth resulted in only an insignificant fall in blood sugar.

CASE 2.—This baby was born at full term after a normal delivery. His birth weight was 6 lb. 12 oz. and his length was 20 in. The mother had hypertension and pre-eclampsia during the latter part of the pregnancy. A girl born four years previously was healthy. Two years later a baby was born with hydrocephalus and spina bifida. After the birth of the present baby, the mother had an oral glucose tolerance test which was normal.

The baby appeared normal at birth and was first fed with glucose at 15 hours of age. At 56 hours of age he had a cyanotic spell while feeding and was placed in an incubator. Four hours later another cyanotic attack occurred. When three days old, he was noted to be very irritable and had twitching movements. The hands showed intermittent carpal spasm, and at the same time the feet became plantar flexed. He sucked poorly and had a poor grasp reflex. A blood sugar level at this time was 8 mg./100 ml.; the serum calcium was 4.0 mEq./l., and the urine was normal.

He was given calcium gluconate intravenously and a few hours later appeared to be improved. Glucose and expressed breast milk were given by gavage every two hours. He also received phenobarbital grain 1/12 intramuscularly every eight hours. He remained rather tremulous, and six hours later he developed circulatory failure and required resuscitative measures. The blood sugar level was 17 mg./100 ml. at this time. An intravenous infusion of 10% glucose was started and a blood sugar level of 8 mg./100 ml. was recorded while the intravenous was still running. On the fourth day of life he had a number of convulsions which were controlled by intramuscular paraldehyde. Capillary and venous blood sugar levels were 8 and 23 mg./100 ml., respectively. The serum calcium was 3.5 mEq./l. The following day he seemed much better and no convulsions were recorded. On the seventh day of life he appeared to be quite normal; the blood sugar level was 30 mg./100 ml. and the serum calcium 4.9 mEq./l. He was taking feedings well, and two days later was discharged from hospital.

He continued to make good progress. At five months of age he held his head well and at the age of 10 months was able to stand with support and say single words. The only abnormality was a slight internal strabismus of the left eye.

CASE 3.—This baby girl was born after a 40-week gestation. The birth weight was 9 lb. 2½ oz. and her length was 21 in. The mother, who had three other healthy children, was an obese 28-year-old woman who had had slight toxemia with each pregnancy. During the present pregnancy she had pre-eclampsia and was admitted to hospital for artificial rupture of the membranes. On admission her blood pressure was 150/95 mm. Hg and her urine contained albumin 0.1 g./100 ml.

The baby appeared normal at birth. When 24 hours old she received her first feeding, was noted to be irritable and became quite tremulous on stimulation. She was given chloramphenicol and placed in an incubator with continuous oxygen. A blood sugar level was 10 mg./100 ml. Phenobarbital grain 1/8 was administered every eight hours, and she was fed with milk and glucose every three hours. The following day she continued to be irritable, became cyanotic when taken out of oxygen and sucked poorly. On the fourth and fifth days of life she was less irritable and slightly

*All blood sugar estimations were performed by the Nelson-Sömögyi[29, 30] method or a close modification of it. Precautions were taken to prevent glycolysis after the blood sample had been withdrawn.

icteric. A chest radiograph at this time showed no abnormality. On the sixth day of life she was still tremulous, was noted to have a poor grasp reflex and was 1 lb. 5 oz. below her birth weight. The serum calcium level was 4.1 mEq./l. and the serum inorganic phosphate 6.3 mEq./l. Calcium gluconate 0.5 g. three times a day was given by mouth. The following day she remained quite irritable and jittery. On the ninth day of life, the tone of the limbs seemed improved and she had a good grasp reflex. The fasting blood sugar level was 42 mg./100 ml. For the next 10 days her condition remained generally unsatisfactory, she was floppy and lethargic, sucked poorly, had a weak cry and her reflexes were depressed. However, she improved slowly and was finally discharged from hospital on the 26th day.

At six weeks of age she weighed 10 lb. 1½ oz. and showed much better tone. At two months of age she had an attack of diarrhea and vomiting requiring admission to hospital. Her blood sugar level at this time was 74 mg./100 ml. She was last seen at the age of one year when there were no abnormalities and she was thought to be developing normally.

CASE 4.—A male baby was born after a 38-week pregnancy. The mother, aged 29, had three previous children, all of whom were healthy. She developed severe acute toxemia at 37 weeks and was admitted to hospital. On admission her blood pressure was 170/100 mm. Hg and her urine contained 0.1% protein. She was given hydralazine hydrochloride (Apresoline) intravenously and shortly afterwards went into labour. The baby was extracted after rotation with Kielland's forceps.

The baby's general condition after birth was satisfactory. He weighed 4 lb. 5 oz. and measured 17 in. in length. The head looked rather big and measured 12 3/4 in. in circumference as compared with a crown-rump measurement of 12 inches and a chest circumference of 10 3/7 in. A radiograph of the skull, however, showed no abnormality. The baby was noted to have fine tremors two hours after birth, but was otherwise well. He was first fed with glucose at 24 hours of age.

At 36 hours of age he was much more tremulous and the left side of his mouth was twitching. At 48 hours of age spells of rigidity and circumoral cyanosis were noted. Two hours later he began to have severe generalized convulsions lasting up to four minutes. He was treated with intramuscular sodium phenobarbital. The spinal fluid was xanthochromic and contained two white blood cells/c.mm. and 100 mg. protein/100 ml. The sugar content was not measurable.

A few hours later he had an apneic spell lasting five minutes which required oxygen and intermittent positive pressure breathing. An intravenous infusion with 5% glucose was started at that time and continued for 18 hours. The serum calcium level was 6.0 mEq./l. and the serum inorganic phosphate 2.9 mEq./l. He had no further convulsions but remained irritable and tremulous for the next 12 hours. After the intravenous infusion had been stopped, the blood sugar level was only 2 mg./100 ml. Twenty-two millilitres of 5% glucose was fed by gavage, and blood sugar levels were measured at half-hour intervals for three hours; they were all below 10 mg./100 ml. Three hours after the feeding, he had a generalized convulsion. He was fed with milk every three hours, additional glucose being administered between the feedings. At four days of age some tremors and stiffening of the limbs were noted before feeding. The head circumference at this time measured 13¼ in., an increase of ½ in. since birth. On the fifth day of life he seemed very much better and there were no signs of convulsions. An adrenaline tolerance test was done after a four-hour fast: fasting blood sugar level, 5 mg./100 ml.; 15 minutes, 6 mg./100 ml.; 30 minutes, 9 mg./100 ml.; 60 minutes, 17 mg./100 ml. The baby continued to make satisfactory progress and was discharged home on the twentieth day of life weighing 5 lb. 1½ oz. A fasting blood sugar level three days before discharge was 42 mg./100 ml.

Subsequent progress was slow, although there were no further convulsions of any kind. When last seen at 13 months of age, he was unable to sit unsupported. He had a rather boat-shaped head with a circumference of 19½ in. There appeared to be motor retardation, his performance being that of a 9-month-old child. Part of the retardation was thought to be due to lack of stimulation, since there was evidence of maternal deprivation.

CASE 5.—This baby boy was born by precipitate delivery before arrival at hospital after 38 weeks' gestation. The mother, aged 30, suffered from bronchiectasis and had had a lung resection. She had shown no signs of toxemia of pregnancy. One other child, three years old, is healthy.

The present baby weighed 5 lb. 6½ oz.; his length was 18½ in. and he appeared healthy at birth. He was first fed with glucose when 10 hours old. At 60 hours of age he had two cyanotic attacks, and when examined three hours later his colour was poor, he was hypotonic with depressed reflexes, and the tension of the anterior fontanelle was thought to be increased. The sucking reflex was poor and he had an exaggerated Moro reflex. At about this time he had an apneic spell from which he recovered with artificial respiration. A diagnosis of cerebral irritation was considered.

The cerebrospinal fluid was under normal pressure, was slightly xanthochromic, contained 8 cells/c.mm. and was sterile on culture. The spinal fluid sugar content was not measurable. A blood sugar level was 7 mg./100 ml. The white blood count was not significantly abnormal, and a radiograph of the chest showed no abnormality.

He was treated with penicillin, chloramphenicol and calcium chloride by mouth, and fed every two hours with 5% glucose in water or breast milk. The following day (fourth day of life) there was great improvement in his general condition. His skin was a good colour, although mildly jaundiced, and he was breathing normally. The grasp and Moro reflexes were normal. Three blood sugar levels during the day were 0, 9 and 12 mg./100 ml. The serum bilirubin level was 14.1 mg./100 ml. Two-hourly feedings were continued. The next day, after a four-hour fast, the blood sugar level was 6 mg./100 ml. Half an hour after the injection of glucagon (100 μg./kg.) the blood sugar level was 19 mg./100 ml. Later the same day the fasting blood sugar level was 8 mg./100 ml. Half an hour after feeding 1½ oz. of breast milk it was 26 mg./100 ml. The serum bilirubin level had fallen to 11.6 mg./100 ml. The antibiotics were stopped and the two-hourly

feedings were continued. Good progress was maintained. On the sixth day of life a fasting blood sugar level was 48 mg./100 ml. The next day a fasting blood sugar level was 28 mg./100 ml. Half an hour after the injection of glucagon (200 μg./kg.) intramuscularly, the level was 53 mg./100 ml. On the eighth day of life the baby was feeding normally at the breast and extra glucose feedings were stopped. When nine days old, the fasting blood sugar was 48 mg./100 ml. The baby was last seen at two months of age and appeared normal in all respects.

DISCUSSION

The foregoing six case histories show a number of close similarities (Table I). All babies appeared normal at birth. Cases 4, 5 and 6 were classified as premature because of their birth weight and measurements although their length of gestation was 37 to 38 weeks. The other three babies were mature. The mothers of Cases 2 and 3 had severe toxemia of pregnancy, and those of Cases 4 and 6 had less severe toxemia. The mothers of the other two babies showed no evidence of toxemia. None of the mothers were apparently diabetic, although only two had had glucose tolerance tests and these were both normal. The babies became severely ill between 24 and 72 hours of age, after feedings had been instituted. They developed cyanosis, apnea, irritability, lethargy, muscular twitchings, disappearance of sucking, grasp and other reflexes. Cases 1, 2, 4 and 6 had generalized convulsions. Although Case 4 was noted to have fine tremors two hours after birth, no anxiety was felt until he was 36 hours of age. All six babies were found to have profound hypoglycemia, with blood sugar levels of 10 mg./100 ml. or less.

TABLE I.—SUMMARY OF THE SIX CASES

Case No.	Sex	Birth weight Lb.	Birth weight Oz.	Toxemia in mother	Age at onset of symptoms (hours)	Age in days when asymptomatic	Present age and status
1	M	6	13	—	60	6	Mentally retarded, 10 months.
2	M	6	12	+	56	5	Normal, 10 months.
3	F	9	2	+	24	26	Normal, 12 months.
4	M	4	5	+	2 - 36	5	Mentally retarded, 13 months.
5	M	5	6	—	60	3	Normal, 2 months.
6	M	5	1	+	72	3	Normal, 9 months.

CASE 6.—This baby boy was the first child of a healthy 25-year-old mother and was born after a gestation of 37 weeks. The mother had shown a moderate degree of toxemia during her pregnancy. The delivery was normal and the infant appeared normal at birth. The birth weight was 5 lb. 1 oz. The skull looked slightly large for the size of the baby, although the circumference measured 12¾ in. compared with a crown-rump measurement of 12¾ in.

No anxiety was felt until the infant was 72 hours of age, when it was noticed that the baby was slightly lethargic. Quite suddenly at about 76 hours of age he had a cyanotic attack in the middle of a feeding. He was treated by suction, given oxygen and seemed to improve. However, a little later he began to have a number of cyanotic attacks at about half-hour intervals. During these attacks, which became progressively more severe, there were marked stiffening, arching of the back, upward deviation of the eyes and extension of the arms and hands at the wrists. The cerebrospinal fluid was slightly xanthochromic and contained no cells; a sugar estimation was not performed. At 80 hours of age the blood sugar level was not measurable. The serum calcium was 5.2 mEq./l. At 90 hours of age the blood sugar level was 3 mg./100 ml. At this time the seizures seemed to have stopped and there were no further apneic spells. The baby still had a feeble Moro reflex. An intravenous infusion of 5% glucose resulted in considerable clinical improvement, and the blood sugar rose rapidly over the next 24 hours to 70 and 80 mg./100 ml. At this time the intravenous infusion was discontinued, and the blood sugar was found to be 19 mg./100 ml. Prednisone in a dose of 2.5 mg. every 12 hours was started on the ninth day of life and given for five days. Blood sugar levels ranged between 41 and 188 mg./100 ml. during this period.

By the fourteenth day the child's clinical condition was excellent and the blood sugar levels remained between 50 and 65 mg./100 ml. The baby was discharged home on the seventeenth day of life in good health. He has been seen at frequent intervals since that time, and has remained in excellent health and appears to be developing normally at nine months of age.

The administration of glucose resulted in rapid disappearance of symptoms in Cases 5 and 6. Case 5 showed great clinical improvement within 12 hours after two-hourly carbohydrate feedings were instituted, and no further symptoms developed even though blood sugar levels remained very low for a further two days. This phenomenon was noted in five of the eight babies described by Cornblath, Odell and Levin.[22] These authors reported immediate alleviation of symptoms in their patients after the administration of parenteral glucose. The administration of parenteral glucose to Cases 1, 2 and 4 did not result in any rapid improvement in their clinical condition. In fact, Case 1 continued to have convulsions while an intravenous infusion of 5% glucose was being administered, a blood sugar estimation done at that time was 53 mg./100 ml.; however, a later blood sugar with the infusion still running was only 10 mg./100 ml. In Case 2, hypoglycemia was also recorded during an infusion of 10% glucose. The oral administration of carbohydrate in Cases 4 and 5 resulted in no rise in blood sugar in the former and only a small rise (8-26 mg./100 ml.) in the latter. In retrospect, therefore, the efforts

to raise the blood sugar in some of these babies may not have been sufficiently vigorous. The parenteral administration of more concentrated glucose solutions might have alleviated symptoms sooner.

The hypoglycemia in these six babies was self-limited, as was the condition in the patients described by Cornblath.[22] All of our patients were symptom-free by seven days of age, with the exception of Case 3, who continued to feed poorly and remained hypotonic and listless until she was 26 days of age. Only one blood sugar was obtained from this infant during this period, and this was in the low normal range for her age. Cases 1 and 4 were found to be mentally retarded at 10 and 13 months of age, respectively; Case 5 is not yet old enough for any final opinion in this respect; the other babies appear to be quite normal at the time of writing.

In three of the five babies in whom serum calcium estimations were performed, the level was found to be below the normal range. In Case 2 hypocalcemia was especially marked (3.5 mEq./l.). Many of the signs of neonatal tetany are similar to those of hypoglycemia, and it is possible that the hypocalcemia may have contributed to the symptoms in these three babies. However, the administration of calcium resulted in little permanent improvement in Case 2, and Case 3 continued to show symptoms after the serum calcium level had risen to normal.

Hypoglycemia in the newborn period may be secondary to adrenal or pituitary hypofunction, galactosemia, hepatic disease (particularly glycogen storage disease) or hyperinsulinism (babies of diabetic mothers[1, 23] or those with an islet cell tumour[24]). None of these conditions were present in our cases. Idiopathic spontaneous hypoglycemia with a prolonged course may also begin in the neonatal period,[25, 26] but the self-limited course of the condition in our cases appeared to exclude this, although in Case 1 a few rather low blood sugar levels were found at 10 months of age. A leucine sensitivity test on this baby was negative. Hypoglycemia may also be secondary to disease of the central nervous system.[1, 27] Intracranial abnormalities were considered initially in four of our patients, and lumbar punctures were performed. It was the low level of sugar in the cerebrospinal fluid of three of the cases which led to the diagnosis of hypoglycemia. The subsequent progress of Cases 2, 3, 5 and 6 makes it unlikely that they had any significant intracranial disease at the time of onset of their symptoms. Cases 1 and 4 show evidence of brain damage at the time of writing; whether this was the cause of their symptoms or the result of the hypoglycemia it is not possible to say. Two of the patients described by Cornblath[22] also showed evidence of permanent central nervous system damage.

The etiology of neonatal hypoglycemia has recently been discussed by Schwartz.[28] Commenting upon the enigma of the low blood sugar level often found in the newborn infant without neurological symptoms, he raised the question whether the absence of symptoms necessarily signifies adequate cerebral metabolism from non-glucose metabolites or whether the cerebral cortex may be relatively insensitive to hypoglycemia. In conclusion he left it an open question whether the hypoglycemia is "physiological" and therefore needs no treatment, or "pathological", requiring correction by the administration of glucose.

We believe the hypoglycemia found in our babies was pathological and harmful and agree with Cornblath[22] that hypoglycemia can be responsible for apnea, cyanosis, irritability and convulsions in the newborn period and should be looked for and treated in the presence of these symptoms. Whether symptomless neonatal hypoglycemia requires treatment with glucose we do not know. We have seen blood sugar levels just as low as those observed in the six infants described in this report, in babies who showed no symptoms and no apparent sequelae. However, we believe that some occult harmful effect of symptomless hypoglycemia cannot be excluded at the present time. It is possible that certain cases of mental retardation or other central nervous system damage may be the result of unrecognized hypoglycemia in the neonatal period.

SUMMARY

Six infants are described who appeared normal at birth but who, between 24 and 72 hours of age, became gravely ill with apnea, cyanosis, irritability, lethargy, muscular twitchings and convulsions. Blood sugar levels of 10 mg./100 ml. or less were found in each instance. The mothers of four of the babies had toxemia of pregnancy. Three of the babies were premature.

The hypoglycemia appeared to be self-limited in all cases, and abnormally low blood sugars were not recorded after seven days of age. Four of the babies are apparently normal at the time of writing; the other two show evidence of central nervous system damage.

The relation of neonatal hypoglycemia to neurological symptoms is discussed. It is believed that hypoglycemia should be considered in a newborn baby showing the symptoms described in this report, and if found, should be treated vigorously by the administration of glucose. Whether symptomless hypoglycemia requires treatment remains an open question.

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SPECIAL ARTICLE

A Report on the Evaluation of Exhibits — Mediscope 1961

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ABSTRACT

Thirty-two educational exhibits presented by the Ontario Medical Association at the 1961 Canadian National Exhibition in Toronto in the exhibit known as "Mediscope 1961" were subjected to an evaluative study. Applying the criteria of educational effectiveness to each exhibit, relative ratings for each exhibit as well as the educational value of Mediscope as a whole were obtained. Quantitative data indicated that this venture in health education was a highly successful endeavour, as 80% of the criteria for educational effectiveness were met by all exhibits. In addition, the study emphasized the potential of educational exhibits in the field of public health education as well as education of specific groups.

The desirability of similar studies is stressed. In addition to quantitative assessment of educational exhibits, such studies would disclose the impact of health information on the attitudes and behavioural changes on the part of the public.

CURRENT Canadian public health literature is practically void of any evaluation studies relating to educational exhibits. In spite of this, hundreds of exhibits are being shown annually at meetings, county fairs and exhibitions by official, voluntary and professional health agencies.

Searching for some clues indicating the relative value of exhibits as an educational medium, the Ontario Medical Association requested the author to undertake some form of independent survey to determine "what value Mediscope '61 had in education of the public on matters of health".

Mediscope had been presented initially in 1959 as Canada's first health exposition. It was planned and organized by the Ontario Medical Association in co-operation with allied professions, voluntary health agencies and departments of government. The Ontario Medical Association accepted the invitation of the Canadian National Exhibition to present Mediscope under the auspices of the C.N.E. in 1961. This made it possible to repeat the greater portion of Mediscope through the continuing co-operation of most of the original participants.

The 32 exhibits comprising Mediscope 1961 were located in the Horticultural Building and the former "Sports Hall of Fame" Building on the grounds of the Canadian National Exhibition in Toronto. The exhibits were accessible to the general public from August 18 to September 4, 1961. (For a list of all exhibits, see Table II.)

The primary objective of Mediscope 1961, as stated by the Ontario Medical Association's descriptive brochure, was "to promote public health". Its main purpose was to erase from the public's mind any residual fears of disease and infirmity by bringing to its attention the advances of medical knowledge, techniques and equipment which are used by the medical and allied professions in preventing and combating the effects of injury and disease in man.

To what extent Mediscope 1961 met this objective and fulfilled its purpose was the major concern of this assessment. However, it became apparent during the planning stages of Mediscope 1961 that to assess the educational value of this exposition

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