Canad. M. A. J.
Mar. 25, 1961, vol. 84

PSYCHOSOMATIC INFLUENCES IN THE GENESIS OF TOXEMIA OF PREGNANCY*

C. A. DOUGLAS RINGROSE, M.D.,†
Edmonton, Alta.

DESPITE intensive interest by a long line of thoughtful men in the field of obstetrics, the etiology of pre-eclampsia and eclampsia has remained obscure. At times, down through the years, the fog shrouding this area of our knowledge has appeared to clear, only to billow back more densely than before in the face of critical appraisal.

At present, toxemia of pregnancy can largely be prevented by optimal care and ideal patient cooperation. Moreover, it can be treated, usually with gratifying results, when detected early. It still contributes appreciably, however, to perinatal and maternal morbidity and mortality. For these reasons the search for the cause of toxemia must continue.

This paper, after a review of the literature, submits yet another theory for perusal.

Many organs or organ combinations have been assigned the role of "toxin producer" in toxemia. Sometimes hyperfunction of the organ has been held responsible and at other times hypofunction has been implicated. The "causative" organs have included breast, anterior pituitary, posterior pituitary, adrenal cortex, adrenal medulla, placenta, uterus and kidney.

Sellheim[24] felt that mammary toxins were responsible for eclampsia and likened the disease to parturient paresis of cows. His suggested treatment of bilateral mastectomy did not gain lasting favour.

The role of the placenta and its products has prompted wide interest. "Hyperplacentosis", an overactivity of the Langhans layer with resultant overproduction of chorionic gonadotrophin, has been implicated by some.[16, 33] Others point out that chorionic gonadotrophins frequently are not elevated above normal pregnant levels in toxemia.[38] Also, in choriocarcinoma, when chorionic gonadotrophin titres reach very high levels, toxemia is not a usual part of the picture.[19] Placental ischemia has been said to produce malfunction of enzymes engaged in de-activating pressor and water-retaining substances.[21] These substances can then escape to the circulation, producing hypertension and making vessels more permeable.[1, 3, 9] Kaku[17] and Magara[20] favour a placental substance as the cause of toxemia.

These arguments have been challenged by Eastman,[8] who feels that a prehypertensive period of excess water retention is usual in this disorder. Page,[23] who favours placental ischemia as a cause of toxemia, suggests that hypertension may be compensatory and a result, rather than a cause, of the disease.

Sophian believes that pre-eclampsia is due to renal ischemia. This is caused by the "utero-renal reflex" which results from the myometrium resisting stretch.[35, 36] He considers that Theobold's work[38] in "preventing" toxemia by interrupting the nerve supply to the uterus supports his concept. Schuurmans,[32] however, believes that the structures in the myometrium accommodate well to increased tension or stretch.

The adrenal cortex has frequently been considered the causative organ. An overabundance of corticoid substances, owing to overproduction, decreased de-activation or both, has been proposed.[3, 10, 21] In Venning's opinion,[39] more accurate methods of hormone assay developed recently leave some of these conclusions open to question. Pre-eclampsia has also been reported in Addison's disease[4] and after bilateral adrenalectomy.[22] Cessation of corticoid replacement therapy in the latter case produced adrenal insufficiency but did not cure the toxemia.

Noradrenaline, from the adrenal medulla or other components of the chromaffin system, has been suggested as the cause of hypertension in toxemia.[13, 18] Hofbauer[13, 14] believes that a general imbalance exists in the pituitary-adrenal-pelvic chromaffin system of a patient with toxemia. This would allow the pressor and water-retaining mechanisms of the adrenal, pituitary-hypothalamus and autonomic nervous system to gain dominance over the counteracting, protective mechanisms of the parasympathetic system, placenta, liver and their component enzymes.

Govan *et al.*[11, 12] favour a humoral mediator for toxemic hypertension related to overactivity of the anterior pituitary.

For a time a patient's blood type was considered important in predisposing her to toxemia.[26] Later reports[6, 25] did not support this view.

Rosenbaum and Maltby[29] and others[40] postulate a primary cerebral dysrhythmia in patients who develop eclampsia.

No organic theory for the etiology of toxemia appears to have universal support. It is interesting to note the contemporary pertinence of a comment made 50 years ago by E. Gustav Zinke, who was then chief of obstetrics at the University of Cincinnati. He stated:[42] "We know something of the causes, but precious little of the character of the poison or its origin notwithstanding the extensive and continued investigations by good men the world over."

Many authors have recognized the important role of the emotions and personality in this disorder. Dieckmann[7] has written: "The fact that intelligent prenatal care, even under poor nutritional conditions, has resulted in a decreased incidence of eclampsia and severe pre-eclampsia, together with a marked decrease in the maternal mortality from the toxemias of pregnancy, usually eclampsia,

*Presented in part at the Surgical Forum of the 46th Clinical Congress, American College of Surgeons, San Francisco, October 12, 1960.
†Formerly Resident in Obstetrics and Gynecology, Wayne State University Hospitals, Detroit, Michigan.

Canad. M. A. J.
Mar. 25, 1961, vol. 84

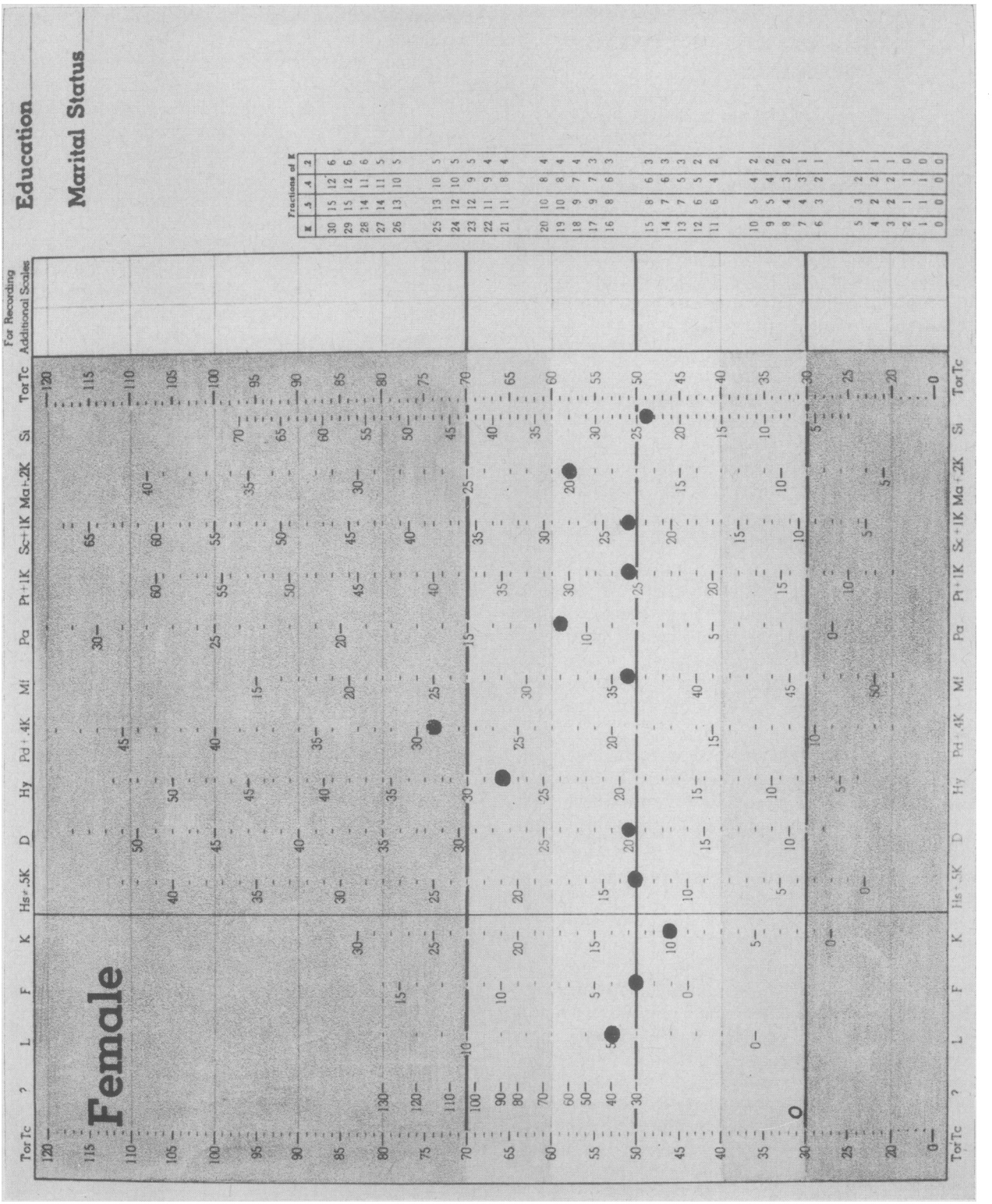

Fig. 1

demonstrates that pre-eclampsia—eclampsia is preventable. The perfect control would be constant 24-hour observation and complete control of the ingestion of food and fluid in the pregnant woman from the first month, together with the elimination of all factors which increase tension." Brown[2] has observed that toxemia occurs frequently in the impoverished, undernourished and uneducated and that more than two prenatal visits virtually prevents eclampsia. This would suggest that even the most superficial doctor-patient relationship has some prophylactic value in this disorder.

Page[23] has noted the development of eclampsia in a Mexican girl seized by the police and placed in jail on suspicion of a felony. A few hours earlier, at a prenatal visit, all was well. The morning after

being apprehended she had edema, a blood pressure of 210/140 mm. Hg, 4+ albuminuria and convulsions.

Soichet[34] states that toxemia is a somatic solution to conflict. The patient feels herself guilty in the eyes of the unborn baby, sensing that the baby is judging her inability. This stimulates destructive forces latent within the woman and toxemia results. He notes that toxemia is unusual in schizophrenics because they employ a different solution in their conflicts. Salerno[31] claims that toxemia occurs frequently in schizoid patients, especially before the first psychotic episode. In his opinion the vasospasm, salt and water retention with resultant hypertension, albuminuria and edema (in toxemia) represent a pathophysiological response to prolonged stress. Holland and Bourne[15] state that "emotional tranquillity and complete physical rest is the ideal at which to aim in the control of pre-eclampsia".

Coppen[5] in an analysis of psychosomatic aspects of toxemia concludes that pre-eclamptics are more masculine than a control group. Rotton, Sachtleben and Friedman[30] note that if no remission of migraine symptoms occurs in susceptible patients during pregnancy, there is an increased incidence of pre-eclampsia and eclampsia.

Thus many authorities would appear to be interested in the role of the psyche in the causation of toxemia.

PATIENTS AND METHODS

In an attempt to assess objectively the psychosomatic component in toxemia, the Minnesota Multiphasic Personality Inventory (M.M.P.I.) test, a reputable personality test, was given to a group of young unmarried mothers living in at a Booth Memorial (Salvation Army) Hospital. This group were predominantly from the middle economic class and usually lived in the hospital during the last half of pregnancy. M.M.P.I. results are available for 41 patients in this group, of whom 19 manifested no pre-eclampsia, eight developed pre-eclampsia, and 14 had "incipient pre-eclampsia". The tests were administered in all cases before the onset of symptoms of pre-eclampsia. All received good prenatal care, and the incipient toxemia category was comprised of subjects who developed abnormal edema and manifested a weight gain of 2 lb. or more in one week. Prompt treatment presumably averted the appearance of a full-blown toxemic picture.

Pre-eclampsia was defined as present when the blood pressure was above 140/90 mm. Hg in the last half of pregnancy, usually in the presence of abnormal edema and/or albuminuria. All blood pressures reverted to normal in the early postpartum period.

The Minnesota Multiphasic Personality Inventory consists of 566 statements which are answered true or false depending on the patient's opinion of herself. The answers are then evaluated and, by comparing the responses to the normal parameters laid down by experience, 10 personality factors can be evaluated. The tests are also evaluated for reliability of response and validity (L, F and K scores). A question score consisting of all of the "cannot say" responses is also obtained. The personality factors evaluated are: hypochondriasis (Hs), depressive (D), hysteria (Hy), psychopathic deviate (Pd), interests (Mf), paranoia (Pa), psychasthenia (Pt), schizophrenia (Sc), hypomania (Ma), and sociability (Si). Liberal variation is permitted, as the answers are considered "normal" if they fall between the 40th and 60th percentile and "abnormal" if they fall outside the 30th or the 70th percentile. Fig. 1 is an example of a personality assessment on a toxemic patient. The hysteria (Hy) score is borderline while the psychopathic deviate (Pd) reading is definitely abnormal.

In the population under study two subjects were between 11 and 15 years, 31 were between 16 and 20 and eight were between 21 and 25. There were 16 gravida I and three gravida II in the "normal" group, seven gravida I and one gravida II in the pre-eclamptic group and 13 gravida I and one gravida II in the incipient pre-eclamptic group. Thirty-seven were white, three Negro and one was Indian.

All 41 infants, 18 girls and 23 boys, were normal healthy newborns.

RESULTS

The results of the M.M.P.I. tests in this group are as follows. Nine of the 14 patients (64%) with incipient pre-eclampsia had abnormal personalities, as evidenced by reliable, valid records with a score or scores above the 70th percentile. Six of the eight patients (75%) with pre-eclampsia had abnormal personalities. In contrast, only six in this group of 19 unmarried mothers (32%) who did not develop pre-eclampsia had abnormal personalities. The incidence of each abnormal factor is outlined in Table I. No scores fell below the 30th percentile.

TABLE I.—INCIDENCE OF ABNORMAL PERSONALITY TRAITS

Category	*Hs*	*D*	*Hy*	*Pd*	*Mf*	*Pa*	*Pt*	*Sc*	*Ma*	*Si*	*Normal*
No pre-eclampsia (19 cases)	0	0	0	5	1	2	1	2	3	1	13
Incipient pre-eclampsia (14 cases)	1	2	0	6	3	3	2	6	2	1	5
Pre-eclampsia (8 cases)	0	0	0	5	1	1	0	1	2	0	2

Multiple abnormal factors were present in some records.

In the pre-eclampsia group, five personalities were abnormal in the psychopathic deviate area, two in the manic category, and one each in interest, paranoia and schizophrenia. Among incipient pre-eclamptics six were abnormal in the schizophrenic category; six in the psychopathic deviate area; three in interest and paranoia; two each in psychasthenia, mania and depressive; and one each in hypochondriasis and sociability. Of the six abnormal

Canad. M. A. J.
Mar. 25, 1961, vol. 84

personalities in the group of 19 who did not develop pre-eclampsia, the incidence of abnormal factors was psychopathic deviate five, mania three, paranoia and schizophrenia two each, and interest, psychasthenia and sociability one each. Statistical analysis of the correlation between emotional instability and toxemia reveals that there is only one possibility in 20 that these results could occur by chance alone.

DISCUSSION

The search for the cause of toxemia has been a challenge to many investigators past and present. "Organic" theories have not stood the test of critical appraisal and it has been extremely difficult to distinguish between cause and effect, with the result that compensatory mechanisms have frequently been assigned an etiological role.

Evidence is presented in favour of personality decompensation during pregnancy as a cause of toxemia. This may not be apparent to the busy clinician but may be revealed by personality tests such as the M.M.P.I. It is possible that a more extensive personality assessment would illustrate the role of the psyche in the etiology of toxemia even more conclusively. The emotional imbalance evidently precedes the onset of toxemia, since this population of unmarried mothers took the M.M.P.I. before the onset of symptoms. The type of personality deviation was not constant and, when average scores were computed for traits in toxemic patients, all fell within the normal range.

The next logical step in implicating the psyche in the cause of toxemia would be to induce the disorder as a result of excessive stress under highly controlled conditions, perhaps with the aid of hypnotic techniques.[28] The time distortion that hypnosis can create should make this practical.

THERAPEUTIC IMPLICATIONS

Prenatal care is potent prophylaxis against the development of toxemia. Leglay, writing in 1812, 46 years before the first prenatal clinic was established, possibly put his finger on the *modus operandi*. He wrote: "Have indulgence for the pregnant woman's complaints, listen to her desires with complaisance, console her, and in place of the severity of the doctor adopt rather the affectionate tone of her friend or her father".[37]

When toxemia occurs, the beneficial effects of rest, sedation and delivery are well established. Supplementary treatment has concentrated on "removing the toxin". In the early years of this century, purgatives[40] were in vogue. Diaphoretics, venesections and the withdrawal of spinal fluid have all enjoyed popularity. Currently, drugs are being sought which will induce more rapid, efficient production of urine with a richer sodium content. Could it be that this diuresis is essentially occupational therapy? Similarly one wonders whether the use of antihypertensive drugs is analogous to employing pancake make-up for treating the "rubor" in inflammation—the patient looks better but the basic disease is not altered and the results are not improved. Brown[2] reported a few years ago that the maternal mortality rate for eclampsia was about 8% whether the treatment consisted of diuretics, antihypertensives, sedatives or "tender loving care".

CONCLUDING REMARKS

Toxemia of pregnancy may be a psychosomatic disorder. In women who develop toxemia, there is frequently a pre-existing occult personality abnormality. Such a personality presumably copes with stress less efficiently, and decompensation may result in toxemia.

Using the ever-popular balance analogy (Table II) the positive constructive influences are listed on the right. These promote a normal, uneventful, gratifying pregnancy and delivery. On the left side are the negative influences which, if dominant in a fragile susceptible personality, may result in decompensation and toxemia.

TABLE II.

"Well-meaning" friends	Prenatal education
Fear of unknown	Self-confidence
Iatrogenic fear	Encouragement by physician
Other stresses	Relaxation
NEGATIVE INFLUENCES ↓ Personality decompensation ↓ Vasospasm, salt and water retention ↓ Clinical toxemia	POSITIVE INFLUENCES ↓ Normal, uneventful, gratifying pregnancy and delivery

Any treatment of toxemia which adds to the right side of this balance, bolstering the patient's personality and alleviating stress, will be helpful. Many of the elaborate therapeutic plans in current use undoubtedly accomplish this purpose. It is possible that it is not the drugs *per se* but the confidence they kindle which produces clinical improvement. This might explain why "paradoxical" treatments, such as administering salt,[27] will aid toxemia, if accompanied by convincing suggestions of benefit to the patient.

The author wishes to acknowledge the invaluable assistance and encouragement of C. S. Stevenson, M.D., professor of obstetrics and gynecology, Wayne State University College of Medicine. Gratitude is also expressed to Brig. Dahlbom and Lt. B. Voeller of the Salvation Army for their aid in this project; they should not be held responsible, however, for any of the ideas expressed herein. The author is also indebted to Dr. A. Bolles, Edmonton psychologist, for advice and Dr. D. Black, Faculty of Education, University of Alberta, for the statistical analysis of results.

11704 - 87 Avenue,
Edmonton, Alta.

REFERENCES

1. VAN BOUWDIJK BASTIAANSE, M. A.: *Am. J. Obst. & Gynec.*, **68**: 151, 1954.
2. BROWN, W. E.: Haas Memorial Lecture, University of Michigan, April 1958.
3. BROWNE, F. J.: *Lancet*, **1**: 115, 1958.
4. COHEN, M.: *Arch. Int. Med.*, **81**: 879, 1948.
5. COPPEN, A. J.: *J. Psychosom. Res.*, **2**: 241, 1958.
6. DICKINS, A. M. *et al.*: *Brit. M. J.*, **1**: 776, 1956.
7. DIECKMANN, W. J.: The toxemias of pregnancy, 2nd ed., C. V. Mosby Company, St. Louis, 1952, p. 364.
8. Editorial Note [EASTMAN, N. J.]: *Obst. & Gynec. Surv.*, **14**: 680, 1959.
9. FEKETE, S.: *Lancet*, **1**: 642, 1958.
10. GORDON, E. S. *et al.*: *Obst. & Gynec.*, **4**: 39, 1954.
11. GOVAN, A. D. T. AND MUKHERJEE, C. L.: *J. Obst. & Gynec. Brit. Emp.*, **57**: 525, 1950.
12. GOVAN, A. D. T. *et al.*: *Ibid.*, **58**: 216, 1951.
13. HOFBAUER, J.: *Am. J. Surg.*, **84**: 394, 1952.
14. *Idem*: *Am. J. Obst. & Gynec.*, **71**: 255, 1956.
15. HOLLAND, E. AND BOURNE, A. W., ed.: British obstetrics and gynaecologic practice, Vol. I, F. A. Davis Company, Philadelphia, 1955, p. 236.
16. JEFFCOATE, T. N. AND SCOTT, J. S.: *Am. J. Obst. & Gynec.*, **77**: 475, 1959.
17. KAKU, M.: *J. Obst. & Gynec. Brit. Emp.*, **60**: 148, 1953.
18. LENNON, C. G. AND GARDINER, J.: *Ibid.*, **65**: 371, 1958.
19. LI, M. C., HERTZ, R. AND BERGENSTAL, D. M.: *New England J. Med.*, **259**: 66, 1958.
20. MAGARA, M.: *J. Internat. Coll. Surgeons*, **14**: 215, 1950.
21. MASTBOOM, J. L.: *Gynaecologia*, **134**: 217, 1952.
22. MOSES, A. M., LOBOTSKY, J. AND LLOYD, C. W.: *J. Clin. Endocrinol.*, **19**: 987, 1959.
23. PAGE, E. W.: The hypertensive disorders of pregnancy, Charles C Thomas, Springfield, Ill., 1953, p. 86.
24. *Idem*: *Ibid.*, p. 83.
25. PEARSON, M. G. AND PINKER, G. D.: *Brit. M. J.*, **1**: 777, 1956.
26. PIKE, L. A. AND DICKINS, A. M.: *Ibid.*, **2**: 321, 1954.
27. ROBINSON, M.: *Lancet*, **1**: 178, 1958.
28. ROSEN, H.: *Chicago M. Soc. Bull.*, **62**: 428, 1959.
29. ROSENBAUM, M. AND MALTBY, G. L.: *Arch. Neurol. & Psychiat.*, **49**: 204, 1943.
30. ROTTON, W. N., SACHTLEBEN, M. R. AND FRIEDMAN, E. A.: *Obst. & Gynec.*, **14**: 322, 1959.
31. SALERNO, L. J.: *Am. J. Obst. & Gynec.*, **76**: 1268, 1958.
32. SCHUURMANS, R.: *Lancet*, **1**: 835, 1957.
33. SCOTT, J. S.: *J. Obst. & Gynec. Brit. Emp.*, **65**: 689, 1958.
34. SOICHET, S.: *Am. J. Obst. & Gynec.*, **77**: 1065, 1959.
35. SOPHIAN, J.: *J. Obst. & Gynec. Brit. Emp.*, **62**: 37, 1955.
36. *Idem*: *Lancet*, **1**: 434, 1958.
37. TAUSSIG, F. J.: *Am. J. Obst. & Gynec.*, **34**: 731, 1937.
38. THEOBALD, G. W.: *Brit. M. J.*, **1**: 422, 1953.
39. VENNING, E. H.: *Clin. Obst. Gynec.*, **1**: 359, 1958.
40. WHITACRE, F. E., LOEB, W. M., JR. AND CHIN, H.: *J. A. M. A.*, **133**: 445, 1947.
41. ZINKE, E. G.: *Am. J. Obst.*, **53**: 226, 1906.
42. *Idem*: *Ibid.*, **63**: 217, 1911.

SPECIAL ARTICLE

AN INTRODUCTION TO THE DIAGNOSIS OF CEREBRAL PALSY AND THE USE OF A PUNCH CARD RECORD*

PRESTON ROBB, M.D.,† *Montreal*

STRICTLY SPEAKING, cerebral palsy can be defined as abnormalities of movement due to disorders of the motor systems of the brain. The causes of the abnormalities may be many and varied, and similarly the disability may vary as to type, degree and location. Although any injury to the brain at any time of life can cause cerebral palsy, here we shall consider only the condition as it occurs in infants or children. It is impossible for any centre with a broad program to limit its facilities to those with lesions of the brain alone. The child with a muscular disorder or spinal cord lesion also needs help which can be provided most practically by the cerebral palsy team. The diagnosis and treatment of cerebral palsy is a team project requiring many experts working in harmony. It is their object to help the child to develop scholastically and socially and to prepare himself for a useful life, a life that is not entirely dependent on the care of others. It is also their desire to help all children and their families who come seeking a "cure". In severe motor and mental retardation, it may mean months of work preparing the parents to accept custodial care of their child. In less severe conditions it may mean convincing the parents that the child does not need a lot of therapy, but should be allowed to develop on his own, naturally, and without overprotection.

The relationship of the child to his environment is evaluated by the social service worker. The intelligence is determined by the psychologist. The motor age is determined by the physiotherapist. His ability to carry out the activities of daily living is evaluated by the occupational therapist. The educational potential is determined by the members of the department of studies. Speech disorders are studied by the speech therapist. Emotional problems are considered by the social service worker, in co-operation with the psychiatrist. The orthopedist determines the need for special shoes, braces, or corrective surgical procedures.

The child with cerebral palsy is often born into a family ill-prepared either emotionally or financially to cope with the innumerable problems that arise. Instead of a normal healthy child, the parents must accept one that falls far short of their ambitions. In this sense the child is unwanted, feelings of rejection and guilt develop, and emotional problems in the child and family are common. It is the desire of the cerebral palsy team to help parents understand these problems, to help them take a relaxed attitude and provide an atmosphere of warmth and affection.

While the roles played by the various therapists will be stressed, it should be emphasized that the most important "therapists" are the parents. It is they who have the greatest influence on the child's development and who carry on treatment at home. At all times they should be considered as part of

*The punch card was prepared with the help and advice of the Medical Advisory Committee of the Cerebral Palsy section of the Canadian Council for Crippled Children and Adults under the chairmanship of Dr. W. Hawke. Punch cards may be obtained from the Canadian Council for Crippled Children and Adults, 31 Alexander St., Suite 115, Toronto.

†From the Cerebral Palsy Division of the Rehabilitation Services of The Montreal Children's Hospital, Montreal.