

MICROGYRIA AND CYTOMEGALIC INCLUSION DISEASE IN INFANCY

BY

L. CROME AND N. E. FRANCE

From the Fountain Hospital and the Mothers' Hospital (Salvation Army), London

(RECEIVED FOR PUBLICATION APRIL 13, 1959)

Many different malformations of the central nervous system can now be produced in the offspring of animals exposed during pregnancy to x rays, drugs, and dietetic deficiency or excess of vitamins, but proof of environmental causation of analogous human conditions is understandably scarce. Occasional neural, somatic, or gonadal maldevelopment has been reported after the administration to pregnant women of aminopterin (Thiersch, 1956), thiouracil (Morris, 1953), methyltestosterone (Nellhaus, 1958), and progesterone (Reilly, Hinman, Pickering, and Crane, 1958; Wilkins and Jones, 1958). Nevertheless, maternal rubella and ionizing radiation remain the only widely known extrinsic causes of human embryopathy. Hence the importance of the suggestion that another, probably infective, condition, cytomegalic inclusion disease, may be also responsible for neural malformation (Diezel, 1954).

It has been known for some time that the brain is one of the organs which may display the characteristic inclusions of cytomegalic inclusion disease. Haymaker, Girdany, Stephens, Lillie, and Fetterman (1954) collected seven such cases in the literature and added one of their own. In addition to the inclusions, some of these brains showed hydrocephalus, periventricular calcification, focal softening, haemorrhages, astrocytic proliferation, perivascular inflammatory infiltration, and ependymal granulations. These are, of course, evidence of neural disease, but not necessarily of malformation. Diezel was the first to report the association of cytomegalic inclusion disease with microgyria, or micropolygyria. Microgyria is a true cerebral malformation characterized by an excess of small abnormally formed gyri (Crome, 1952). Histological and embryological considerations suggest that the cause of this condition operates before the sixth month of foetal development.

The patient described by Diezel was an infant weighing 2,500 g. at birth who developed

neonatal jaundice, ascites with hepatomegaly, and splenomegaly, and died at 24 days. Inclusion bodies were found in the kidneys, liver, and brain. The brain weighed 130 g. and showed microgyria of the neocortical areas. Necrosis was present in the medial basal regions of both temporal lobes and in the cerebellum. The optic nerves were atrophic, there was marked hydrocephalus, and the corpus callosum was thin. On microscopy, the necrotic areas showed infiltration by lymphocytic and plasma cells with many compound granular corpuscles, macrophages, and calcified particles. The olfactory nerves were the seat of marked inflammatory change. Typical inclusions of cytomegalic inclusion disease were seen in the leptomeninges, ependymal, and glial cells, as well as in some of the undifferentiated matrix cells arranged in numerous dense periventricular foci. The only mature neurons to show inclusions were a few Purkinje cells, found after prolonged search. Architectonically, the palaeocortex and archicortex were normal and the relatively older occipital cortex was better developed than the rest of the neocortex. It appeared therefore to the author that the virus of cytomegalic inclusion disease attacked the foetus at a time when migration of nerve cells from the periventricular matrix to the periphery was complete in the older, and still afoot in the newer, parts of the brain, i.e., the beginning of the fourth month of gestation.

Diezel was able to re-examine histological preparations from the brains of the two cases of cytomegalic inclusion disease previously reported by Hartmann (1948) and by Haymaker and his co-workers. He found microgyria in one of these, whilst the other had an anomaly of gyri, possibly of microgyric nature. Microgyria was possibly present in a case of cytomegalic inclusion disease presented by Mercer, Luse, and Guyton (1953): the brain of this child had an irregular, thin-walled cyst, 1.5 cm. in diameter, in the right parietal lobe, the cortical surface around the cyst was slightly

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The Clinical Pathology of Infancy Frederick William Sunderman, 1967 **The Clinical Pathology of Pregnancy and the Newborn Infant** Maurice N. Cauchi, James B. Brown, Gwendolyn L. Gilbert, 1984 **Current Catalog** National Library of Medicine (U.S.), 1992-10 **Sudden Death in Infancy, Childhood and Adolescence** Roger W. Byard, 2004-01-08

This unique comprehensive survey of virtually all aspects of sudden death in infants and childhood will be an essential source of reference for pathologists, clinicians and lawyers who deal with such cases. Individual sections deal in detail with deaths due to inflicted and non inflicted injuries and to natural diseases. This new edition includes 1200 new references, 300 new illustrations and an extensively revised chapter on sudden infant death syndrome. The intentional injury chapter has additional material on head trauma, the biomechanics of injury, neonaticide, suicide and subtle and unusual trauma. The chapter on non intentional injury has also been expanded to more accurately reflect its importance as a cause of death. Deaths in the first week of life are also covered. This new edition also covers the full range of natural causes of death and their pathological investigation undertaken in light of advances in our understanding of genetic susceptibility and pathophysiology.

Potter's Pathology of the Fetus and Infant E-Book Enid Gilbert-Barness, Raj P. Kapur, Luc Laurier Oigny, Joseph R. Siebert, 2007-05-01. This comprehensive reference addresses all aspects of fetal and neonatal pathology including complicated pregnancies, multiple pregnancies, abortion, placental pathology and disorders affecting the full term neonate. A consistent organization allows for quick access to specific guidance and nearly 2 500 illustrations, 2 350 in full color, depict conditions and abnormalities as they present in practice, facilitating diagnosis. An Image Bank on CD ROM, new to this edition, features all of the illustrations from the 2 volume set, downloadable for presentations. Offers comprehensive coverage of all common and rare embryonic, fetal and infant disorders in one source. Correlates clinical, pathologic and genetic findings for each systemic disease. Emphasizes the genetic and molecular basis of birth defects. Features nearly 2 500 illustrations, 2 350 in full color, which depict each abnormality or condition as they present in practice. Presents practical information on autopsy techniques and protocols. Provides the latest guidance on molecular pathology, immunohistochemistry, DNA technology and more. Offers an expanded discussion of developmental biology related to the pathogenesis of birth defects. Features user friendly summary tables and diagnostic flow charts making information quick and easy to find. Includes a CD ROM featuring all of the illustrations from the 2 volume set.

Perinatal Physiology Uwe Stave, 2013-06-29. Living Nature, not dull Art. Shall plan my ways and rule my heart. Cardinal Newman. Nature and Art, 1868. One of the ineluctable consequences of growth in any field of science is that subjects of inquiry, once established, tend to give birth to subsubjects and that the subsubjects, once established, will in time undergo further mitotic division. Not so many years ago, problems surrounding the ietus and newly born infant lay in a realm almost to be described as a no man's land. Obstetricians properly gave major consideration to understanding and learning about processes and disorders concerned with maternal health and

safety The welfare of the infant was regarded as of secondary importance Pediatricians on their part hesitated to invade the nursery a sanctum regarded as belonging to the domain of the accoucheur And the pathologist enveloped in the mysteries of life and death in the adult found scant time for the neonate and the placenta *National Library of Medicine Current Catalog* National Library of Medicine (U.S.), Sudden Death in the Young Roger W. Byard, 2010-09-16 Determining the cause of death in children and young adults can pose considerable challenges Professor Byard provides for the first time a complete overview of pathological aspects of sudden death in the young from before birth to middle adult life Highly illustrated with more than 800 colour figures this third edition contains new sections on sexual abuse pregnancy related deaths and rare natural diseases as well as expanded coverage of unexpected death in young adults up to the age of 30 years Chapters are organised by systems and cover all aspects of natural death as well as accidents suicides and homicides Supported by extensive referencing and numerous tables the book can also be used as a practical autopsy manual An encyclopaedic overview and analysis of sudden death in the young this is a key text for pediatric and forensic pathologists pediatricians and lawyers and physicians involved in medicolegal cases *Brain Damage in the Preterm Infant* Nigel Paneth, Raoul Rudelli, Elias Kazam, William Monte, 1994-01-10 A detailed survey allowing the formulation and testing of hypotheses about the causes and consequences of haemorrhage *Summary of Proceedings, Second International Conference on Causes of Sudden Infant Death, Seattle, 1969* National Institute of Child Health and Human Development (U.S.), 1971 **Medical Record** George Frederick Shrady, Thomas Lathrop Stedman, 1901 *Pathology of Infancy and Childhood* John M. Kissane, 1975 **Handbook of Laboratory Animal Science, Volume III, Third Edition** Jann Hau, Steven J. Schapiro, 2013-12-26 While replacing and reducing the use of laboratory animals are integral parts of the 3Rs replace reduce refine which form the cornerstones of laboratory animal science biomedical research involving animals remains absolutely essential for the advancement of the medical veterinary agricultural and biological sciences Building upon the bestselling previous edition the Handbook of Laboratory Animal Science Volume III Third Edition Animal Models complements volumes I and II of the third edition by completing the task of providing a comprehensive overview of animal models in all biomedical disciplines The three Rs have been integrated throughout this handbook to promote efficient and humane experimental work with animals Written by international experts each chapter focuses on an important subdiscipline of laboratory animal science and can be used as a stand alone text This volume contains new chapters for six additional disease animal models spinal cord injury cardiovascular diseases sudden infant death syndrome developmental disorders eye diseases and human cancer It also presents a new chapter on applying reduction and refinement to animal models This handbook can be used for undergraduate and postgraduate laboratory animal science courses and as a handbook for scientists who work with animals in their research for university veterinarians for regulators and for other specialists in laboratory animal science

Murder, Medicine and Motherhood Emma Cunliffe, 2011-05-27 Since the early 1990s unexplained infant death has

been reformulated as a criminal justice problem within many western societies This shift has produced wrongful convictions in more than one jurisdiction This book uses a detailed case study of the murder trial and appeals of Kathleen Folbigg to examine the pragmatics of proof beyond a reasonable doubt It explores how legal process medical knowledge and expectations of motherhood work together when a mother is charged with killing infants who have died in mysterious circumstances The author argues that Folbigg who remains in prison was wrongly convicted The book also employs Folbigg's trial and appeals to consider what lessons courts have learned from prior wrongful convictions such as those of Sally Clark and Angela Cannings The author's research demonstrates that the Folbigg court was misled about the state of medical knowledge regarding infant death and that the case proceeded on the incorrect assumption that behavioural and scientific evidence provided independent proofs of guilt Individual chapters critically assess the relationships between medical research and expert testimony the operation of unexamined cultural assumptions about good mothering and the manner in which contested cases are reported by the press as overwhelming

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Pathology of Heart Disease in the Fetus, Infant and Child Michael T. Ashworth, 2019-08-22 Clearly presents the pathology of heart disease from fetus to adolescence integrating histology and macroscopy with effects of treatment

Physical Trauma as an Etiological Agent in Mental Retardation, 1970 **Quarterly Bulletin of Northwestern University Medical School** Northwestern University (Evanston, Ill.). Medical School, 1905 **Infant feeding** Clifford Groselle Grulee, 1912 Sudden Infant Death Syndrome, 1973 United States. Congress. Senate. Labor and Public Welfare, 1973

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web recall that the molar mass can be determined from a chemical formula and used as a conversion factor we can add that conversion factor as another step in a calculation to make a mole mass calculation where we start with a given number of moles of a substance and calculate the mass of another substance involved in the chemical equation or vice

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web mass 32 g mass 16 g mass 3 2 g 2 for jars d e f use the number of molecules in the circle to determine the number of moles of O_2 in the jar then calculate the mass of the gas in the jar d e f 12 mol O_2 2 mol O_2 6 mol O_2 mass 384 g mass 64 g mass 192 g 3 jars g h and i contain ammonia gas NH_3

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web learning objective to calculate the quantities of compounds produced or consumed in a chemical reaction a balanced chemical equation gives the identity of the reactants and the products as well as the accurate number of molecules or

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web jul 4 2019 solution from the balanced equation it is known that 1 mol N_2 2 mol NH_3 use the periodic table to look at the atomic weights of the elements and calculate the weights of the reactants and products 1 mol of N_2 28.0 g 1 mol of NH_3 is 17.0 g

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web solution average atomic mass of Li 6.941 amu $\times 6.0151 \text{ amu} = 1 \times 7.0160 \text{ amu}$ 6.941 1.0009 $\times 7.0160$ 1.0009 $\times 0.075 = 0.075$ corresponds to a natural abundance of 6Li of 7.5 percent the natural abundance of 7Li is 92.5 percent 3.7 the unit factor required is 6.022

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web a molar mass Li_2CO_3 73.89 g CO_2 44.01 g H_2O 18.02 g b molar mass CS_2 76.13 g c molar mass CHCl_3 119.37 g d molar mass $\text{C}_6\text{H}_8\text{O}_6$ 176.12 g e molar mass KNO_3 101.10 g

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web it is a small step from mole mass calculations to mass mass calculations if we start with a known mass of one substance in a chemical reaction instead of a known number of moles we can calculate the corresponding masses of other substances in the reaction

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web jul 4 2022 to calculate the mass of titanium metal that you can obtain multiply the number of moles of titanium by the molar mass of titanium 47.867 g/mol moles Ti mass Ti times molar mass Ti 4.12 cancel mol Ti times $\frac{47.867 \text{ g Ti}}{1 \text{ mol Ti}}$ 197 g Ti notag

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web 41.42 chemistry ch 3 mass relationships in chemical reactions molecular mass of NO_2 atomic mass of N 14 atomic mass of

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108 02 amu example 3 2 exercise 3 3