INTERNATIONAL EXPERT PANEL

LUCY LETBY CASE

Summary report – 2 April 2025

Table of Contents

INTRODUCTION *3*
INTERNATIONAL EXPERT PANEL *3*
BIOGRAPHIES *6*
CASE SUMMARIES *22*
SUMMARY OF FINDINGS *34*
CONCLUSIONS *36*

INTRODUCTION

In August 2023, Lucy Letby was convicted of the murder of seven babies and attempted murder of seven other babies at the Countess of Chester Hospital Neonatal Unit. In April 2024, she appealed her conviction but her appeal was rejected. Dr Shoo Lee testified at her appeal and was concerned that there were problems with the medical evidence used in her trial and appeal. As Lucy had exhausted all avenues of appeal, Dr Lee proposed to Lucy's solicitors that he would convene an International Expert Panel to examine all the medical evidence in detail, and to produce an impartial, evidence-based report about the causes of death or injury of all the cases involved in the trial. The report would be released regardless of whether the findings were favorable or unfavorable to Lucy. Lucy and her solicitors agreed.

INTERNATIONAL EXPERT PANEL

The International Expert Panel is an independent panel of some the most experienced and distinguished neonatologists and pediatric specialists in the world.

ACKNOWLEDGEMENT OF FAMILIES

Members of the International Expert Panel would like to acknowledge the families of the infants who have died. We hope they can accept our sympathies and condolences. We will never truly understand their stress and anguish, and our work is not meant to cause more distress. Rather, it is meant to give comfort and assurance in knowing the truth about what really happened to their babies. We value that their families want to know the truth, and that is why the panel is doing this work. To tell the truth and to educate clinicians and leaders. These infants are teachers in their lives and in their deaths.

OBJECTIVE

The objective of the International Expert Panel is to provide an impartial, evidence-based report about the causes of death or injury among patients in the Lucy Letby case, based on the medical evidence. There would be no determination about innocence or guilt of the defendant.

INSTRUCTIONS

Dr Shoo Lee has been instructed to convene an International Expert Panel to examine the medical evidence. The panel is independent and worked under the agreement with Lucy and her solicitors that all findings will be released even if they did not favor the defendant. The defendant and her lawyers provided the panel with access to medical records and witness statements used in the trial. The panel self-determined how to examine the medical evidence, interpret the findings and produce a report.

As Chair, Dr Shoo Lee issued the following instructions to the International Expert Panel:

1. Review the medical records and determine the cause of death or injury
2. Review and comment on the expert witness statements and opinions
3. Issue an independent report about the causes of death or injury

METHODS

Each case was examined by 2 experts independently, who then submitted their reports to the Chair. If their findings were in agreement, their conclusions were accepted as final. If their findings differed, a third member of the panel was asked to review the case and a consensus opinion was developed. Summaries of each case were then developed by the Chair, approved by the panelists who examined the case, and shared with the entire panel. Strict confidentiality was observed.

MEMBERSHIP

Dr Shoo Lee invited members of the panel to participate on a voluntary basis and to work pro bono. Members of the panel did not receive any benefits, in cash or in-kind, for their work.

The panel comprises fourteen very experienced and well-known experts from highly prestigious institutions in 6 countries around the world, including Canada, United States of America, Japan, Germany, Sweden and the United Kingdom. They include ten neonatologists, one pediatric surgeon, one pediatric infectious disease specialist, one pediatric and perinatal pathologist, and one senior neonatal intensive care nurse.

With the exception of the Chair and member from the UK, the other members of the panel knew about the existence of the case but otherwise had little knowledge about it. Panel members participated in their individual capacity, and not on behalf of the institutions they are associated with.

Members of the panel are:

1. Shoo K. Lee, OC, DHC, PhD, FRCPC, MBBS (Chair)
   Professor Emeritus, University of Toronto, Canada Honorary Physician, Mount Sinai Hospital President, Canadian Neonatal Foundation

2. Marta Cohen, OBE, MD, FRCPath, DMJ (Pathol)
   Dip Med Ed Consultant Paediatric Pathologist. Head of Department South Yorkshire and Bassetlaw Pathology
   Histopathology Department. Sheffield Children's NHS FT Vice-President, Royal College of Pathologists Council member, European Society of Pathology

3. Eric Eichenwald, MD, FAAP
   Professor of Pediatrics, Perelman School of Medicine at University of Pennsylvania, USA Chief of the Division of Neonatology at Children's Hospital of Philadelphia Holder of the Thomas Frederick McNair Scott Endowed Chair.

4. Helmut Hummler, MD
   Senior Medical Director, European Foundation for Care of Newborn Infants, Germany

5. Tetsuya Isayama, MD, MSc, PhD
   Head of Division of Neonatology, National Center for Child Health and Development, Tokyo, JapJapan Director, Asian Neonatal Network

6. Joanne Langley, MD. MSc, FRCPC. FSHEA, FIDSA, FPIDS
   Head of Division of Pediatric Infectious Diseases, Dalhousie University, Canada Professor, Departments of Pediatrics and Community Health & Epidemiology, Faculty of Medicine, Dalhousie University Holder of the Canadian Institutes of Health

Research – GlaxoSmithKline Chair in Pediatric Vaccinology, Dalhousie University Active Staff, Pediatric Infectious Diseases, IWK Health Centre

7. Neena Modi, MB ChB; MD; FRCP; FRCPCH; FFPM; FMedSci
   Professor of Neonatal Medicine & Vice-Dean (International), Imperial College London Honorary Consultant, Chelsea and Westminster NHS Foundation Trust President, European Association of Perinatal Medicine

8. Sandra Moore, RN
   Staff Nurse, NICU, Southlake Regional Health Center, Newmarket, Canada Sullivan Medicolegal Experts, Richmond Hill, Ontario

9. Mikael Norman, MD, PhD
   Professor/Senior Physician, Department of Clinical Science, Intervention and Technology, Karolinska Institutet, Sweden Chairman, Swedish Neonatal Quality Register Founder, International Society of Evidence-Based Neonatology (EBNEO)

10. Bruno Piedboeuf, MD, FRCPC
    Professuer Titulaire en Pediatrie, Universitaire Laval, Canada Coordonnateur des Services Cliniques du RUIS de l'Universite Laval Directeur des Affaires Universitaires, Ministere de la Sante et des Services Sociaux du Quebec

11. Prakeshkumar Shah, MSc, MBBS, MD, DCH, MRCP, FRCPC
    Professor of Pediatrics, University of Toronto, Canada Head, Department of Pediatrics & Maternal-Infant Care Research Center, Mount Sinai Hospital Senior Clinician Scientist, Lunenfeld-Tannenbaum Research Institute Director, International Network for Evaluation of Outcomes for Neonates (iNEO) Director, Canadian Preterm Birth Network Scientific Advisor & Past Director, Canadian Neonatal Network

12. Nalini Singhal, MBBS, FRCPC
    Professor Emeritus, University of Calgary, Canada Co-Editor of WHO/AAP Helping Babies Survive Programs

13. Erik Skarsgard, MD, MSc, FRCSC, FACS, FAAP
    Professor, Division of Pediatric Surgery, University of British Columbia, Canada Director, Canadian Pediatric Surgery Network

14. Ann R. Stark, MD, FAAP
    Professor in Residence of Pediatrics, Harvard Medical School, USA Director of Faculty Development, Department of Neonatology, Beth Israel Deaconess Medical Center.

The panel also relied on the reports of external experts in engineering, Professor Geoff Chase and Helen Shannon, for information about insulin and c-peptide testing (Annex). These experts were instructed by those representing Lucy Letby.
Biographies of members of the panel are on the following pages.

BIOGRAPHIES

Professor Emeritus Shoo K. Lee, OC, DHC, PhD, FRCPC, MBBS (Chair)

Dr. Shoo Lee is a neonatologist and health economist. He is Professor Emeritus at the University of Toronto, Honorary Physician at Mount Sinai Hospital, and President of the Canadian Neonatal Foundation. He was formerly Pediatrician-in-Chief at Mount Sinai Hospital, Head of the Division of Neonatology at the University of Toronto and the Hospital for Sick Children, Head of the Department of Newborn and Developmental Pediatrics at Sunnybrook Hospital, Canada Research Chair (Tier 1) and Scientific Director of the Institute of Human Development, Child and Youth Health at the Canadian Institutes of Health Research.

Dr. Lee received his medical degree from the University of Singapore, completed his paediatric training at the Dr Charles A. Janeway Child Health Centre in Canada and neonatal fellowship training at Boston's Children's Hospital, and is a Fellow of the Royal College of Physicians of Canada. He received his PhD in Health Policy (Economics) from Harvard University, and the Doctorate Honoris Causa in Medicine from Laval University.

His research focuses on health policy and healthcare quality improvement. He founded the Canadian Neonatal Network and transformed Canada's neonatal outcomes into one of the best among OECD countries. He created Family Integrated Care to empower parents as NICU care providers, and established training, research and quality improvement programs in Latin America, Africa and Asia. He has published more than 400 scientific papers in peer reviewed journals and received many awards, including the Aventis Pasteur Research Award and the Distinguished Neonatologist Award from the Canadian Pediatric Society, the Knowledge Translation Award from the Canadian Institutes of Health Research, the Douglas K. Richardson Award for Lifetime Achievement in Perinatal Research from the US Society for Pediatric Research, the Premier Member Award from EPIC Latino, and the Magnolia Gold Award from the Shanghai Government. He is an Officer of the Order of Canada, the highest award given to a Canadian.

Dr Marta Cohen, OBE, MD, FRCPath, DMJ (Pathol), Dip Med Ed

Dr Marta Cohen is a Paediatric and Perinatal Pathologist, also trained as Forensic Pathologist. She graduated from La Plata Medical School in Argentina, and was trained in pediatric histopathology and forensic and legal medicine in Buenos Aires and South Africa, and in forensic pathology in Sheffield, UK, and holds the FRCPath (UK).

She is currently a Consultant and Paediatric and Perinatal Pathologist, and Head of the Department of Histopathology at the Sheffield Children's Hospital NHS FT, as well as Honorary Professor of Paediatric Pathology at the University of Sheffield, School of Medicine and Population Health, Division of Clinical Medicine.

Dr Cohen's research focuses on aspects of: Use of post-mortem magnetic resonance and PM CT scan as adjuvant to traditional post-mortem; Investigation of sudden infant death syndrome and sudden death in childhood; Investigation of stillborn and pre-term delivery and Gastrointestinal paediatric pathology. Her publications include: 7 books in print; 36 Chapters in print and 2 in press: and 172 peer reviewed Journals in Print. In addition, I have 28 Peer-refereed conference papers in print/press.

Dr Cohen has received numerous awards, including: ESCMID Fellow in 2025, the Illustrious Visitant, awarded by the City of La Plata, Argentina in 2022, the Bronze Clinical Excellence NHS CEA Award in 2021, the Compassionate Care Award at Sheffield Children's NHS FT in 2021, the Order of the British Empire (OBE) in 2020, The Pathologist's 2019 "Trailblazers" Power List in 2019 (https://thepathologist.com/power-list/2019), recognition in the book "The first fifty years of the European Society of Pathology": Minds, microscopes and molecules. By Andrew Wilson, Springer. ESP in 2016, the Power List of most influential pathologists: (https://thepathologist.com/the-power-list-2015/) in 2015, the BMJ Award: Minimally Invasive Autopsy in 2015, and Sheffield Ambassador since 2015.

Professor Eric Eichenwald, MD, FAAP

Dr. Eichenwald graduated from Harvard Medical School in 1984. He received his training in pediatrics at Boston Children's Hospital, and his training in Neonatal-Perinatal Medicine in the Joint Program in Neonatology, based at Boston Children's, Brigham and Women's and the Beth Israel Hospitals in the United States. He joined the faculty at Harvard Medical School in 1990 and was a staff neonatologist at the Brigham and Women's and Boston Children's Hospitals. He remained on the faculty at Harvard Medical School until 2006, when he was named the Medical Director of the Newborn Center at Texas Children's Hospital and Associate Professor of Pediatrics at Baylor College of Medicine in Houston, Texas. Dr. Eichenwald later joined the faculty of the University of Texas and Children's Memorial Hermann Hospital in 2010, where he was the David R. Park Professor of Pediatric Medicine, Chair of the Department of Pediatrics, Division Director of Neonatal/Perinatal Medicine and Physician-in-Chief at Children's Memorial Herman Hospital.

In 2016, Dr. Eichenwald relocated to the Children's Hospital of Philadelphia, where he is Chief of the Division of Neonatology, and the Thomas Frederick McNair Scott Endowed Chair, Professor of Pediatrics at the Perelman School of Medicine, University of Pennsylvania. He is currently the Alternate PI for the University of Pennsylvania NIH Neonatal Research Network site, and co-PI for an RO1 grant entitled "Intermittent Hypoxia and Caffeine in Infants Born Preterm (iCAF Study)".

Dr. Eichenwald is a member of the Society for Pediatric Research, the American Pediatric Society, and served on the Board of Directors of the American Board of Pediatrics until 2024. He is the current Chair of the American Academy of Pediatrics Committee for Fetus and Newborn and is an Associate Editor of the Archives of Diseases in Children – Fetal and Neonatal Edition.

Professor Helmut Hummler, MD

Dr. Hummler graduated in 1986 from the medical school of the University of Tübingen, Germany. After his residency in Pediatrics in Germany he received his training in Neonatology from 1992 to 1995 by Eduardo Bancalari, M.D. at the Division of Neonatology, Department of Pediatrics, Jackson Memorial Medical Center, University of Miami, Florida, U.S.A. After returning to Germany Dr. Hummler joined the staff of the Division of Neonatology und Pediatric Critical Care, Children's Hospital, University of Ulm, Germany in 1996, where he became Division Chief in 2005. He received his M.B.A. and became Professor of Pediatrics in 2006 and Vice-Chairman of the Dept. of Pediatrics in 2008. He joined the faculty at Sidra Medicine, Doha, Qatar to become Chief of the Division of Neonatology in 2017 and was appointed as Professor of Pediatrics at Weill Cornell Medicine – Qatar in 2018. His team introduced family-centered care the new standard in Sidra Medicine, Doha, Qatar. In 2021 he joined the Dept. of Neonatology, Tübingen University, Germany, and became Chief of the Division of Neonatology and Pediatric Critical Care, Marburg, Germany in 2023. In September 2024 he became the Senior Medical Director of the European foundation for the care of newborn infants (EFCNI). His research interests are related to neonatal lung injury, mechanical ventilation, permissive hypercapnia and to the effects of hypoxemic episodes in VLBWI. He was involved in the development of an automated device for closed-loop FiO2 control. His group was actively participating in many international clinical trials, and he has published more than 150 original articles in peer-reviewed journals and editorials and book chapters. In recent years his activities were focused on research activities related to quality of care as well. In a close collaboration with other stakeholders from obstetrics he led a multidisciplinary team to reduce the rate of IVH and to improve neurodevelopmental outcome substantially. He received the Scientific Award of the German Society of Neonatology and Pediatric Intensive Care and received several awards as a Top Physician in Neonatology in Germany. In 2018 he received the Chief Executive Recognition Award (CEO Sidra Medicine, Doha, Qatar). He has been/is a member of many national and international scientific committees and a frequent peer reviewer for many scientific journals. He is a fellow of the American Academy of Pediatrics and a member of the Society of Pediatric Research and the European Society of Pediatric Research. Dr. Hummler served as an invited speaker at more than 150 national and international meetings and postgraduate courses. Dr. Hummler is a strong advocate of family-centered care, based on "core principles such as dignity and respect for parents/families, information exchange, family participation in care, and cooperation on all levels needed". Furthermore, in his opinion participation in "Quality related clinical research should be a uniform standard in NICUs thriving for excellence".

Dr Tetsuya Isayama, MD, MSc, PhD

Dr. Tetsuya Isayama is a neonatologist and clinical epidemiologist. He is the Head of the Division of Neonatology, National Center for Child Health and Development (NCCHD), Tokyo, Japan. He is an emeritus task force member of the Neonatal Life Support (NLS) of the International Liaison Committee of Resuscitation (ILCOR), the Director of the Asian Neonatal Network Collaboration (AsianNeo), and board member of several neonatology related Japanese academic societies.

He completed neonatology fellowship training at Osaka Women's and Children's Hospital, Osaka, Japan, and then completed the neonatology fellowship of the University of Toronto, Canada. He obtained his PhD in Clinical Epidemiology and Biostatistics at McMaster University, Canada.

He is a recognized expert in the clinical management of extremely preterm infants. His research interests are in neonatal and perinatal epidemiology, international comparative studies on clinical practices and outcomes of preterm infants, neonatal respiratory management & bronchopulmonary dysplasia, circulatory management & patent ductus arteriosus, and systematic reviews & clinical practice guidelines. He has authored more than 100 publications in peer review journals including JAMA, JAMA Pediatrics, Pediatrics, and Circulation.

Joanne M. Langley MD. MSc, FRCPC. FSHEA*, FIDSA*, FPIDS*

Dr. Langley is a pediatric infectious disease physician and vaccine researcher at the Canadian Center for Vaccinology and Professor of Pediatrics and Community Health and Epidemiology at Dalhousie University. She is Head of the Division of Pediatric Infectious Diseases, IWK Health. She is an active investigator in the CIHR-funded Canadian Immunization Research Network (CIRN) and is the co-lead of its Clinical Trials Network.

Dr. Langley's research focuses on the epidemiology, prevention and control of respiratory viruses (particularly influenza and respiratory syncytial virus) and other vaccine-preventable infections. Dr. Langley formerly was Medical Director of Infection Prevention and Control services at IWK Health. Her research also focuses on vaccine policy and evidence-based immunization decision making. During the COVID-19 pandemic she served as co-chair of Canada's COVID-19 Task Force, and as a member of the COVID-19 Expert Panel for the Chief Science Advisor of Canada. She is currently co-chair of the Council of Expert Advisors to the Government of Canada's Ministries of Innovation, Science and Economic Development and of Health, for the development of the Biomanufacturing and Life Sciences Strategy. She is a former member of the Canadian Task Force on Preventive Health Care and served as vice and chair of Canada's National Advisory Committee on Immunization.

https://medicine.dal.ca/departments/department-sites/pediatrics/our-people/our-faculty/joanne-_langley.html

**Fellow, Society of Hospital Epidemiology of America; Fellow, Infectious Disease Society of America; Fellow, Pediatric Infectious Disease Society*

Neena Modi MB ChB; MD; FRCP; FRCPCH; FFPM; FMedSci

Professor of Neonatal Medicine, Imperial College London

Neena is a distinguished clinician scientist and Fellow of the Academy of Medical Sciences. She qualified from the University of Edinburgh and has worked in tertiary neonatal intensive care for over three decades. She heads a multiprofessional neonatal research group and has authored over 400 original research papers, chapters in textbooks, and other publications. She has held a number of professional leadership roles and is the current president of the European Association of Perinatal Medicine, and a past-president of the British Medical Association, and Royal College of Paediatrics and Child Health.

In 2022 she received the US Critical-Path Institute, Pioneer Award for *"contributions to health data research"* and Medical Women International Association award *"to a woman physician who has made outstanding contributions to the cause of women in medicine"*, and in 2023, the Joint European Neonatal Societies "outstanding neonatologist" award.

Neena is a longstanding advocate for fairness, equity, and evidence-informed decisions. Her involvement on the panel is as an individual, and not representative of any organisation or institution.

Sandra Moore, RN

I obtained my Nursing Diploma from Seneca College of Applied Arts and Technology in Toronto, Ontario and have been a dedicated Neonatal Intensive Care Unit (NICU) Registered Nurse for 36 years, including at the Hospital for Sick Children, Mount Sinai Hospital, Cortelucci Vaughan Hospital and Southlake Hospital.

My professional responsibilities have included:

- Neonatal Resuscitation Program Instructor
- George Brown College Clinical Instructor
- Coauthor of the Resuscitation Handbook for RNs
- NICU Team Leader
- Advanced Skills Preceptor & Preceptor and Bereavement Coordinator
- Preceptor for new staff nurses
- Certification as Percutaneously Inserted Central Catheter (PICC) RN
- Member of the IV access team in the NICU

I have received several awards for education and leadership, and was nominated by my peers for the Toronto Star Nightingale Award. I am a member of the Sullivan Medico- Legal Experts, in Richmond Hill, Ontario

Professor Mikael Norman, MD, PhD

Mikael Norman was born 1957 in Stockholm, Sweden. He graduated as M.D. at Karolinska Institutet (KI) in 1982 and finalized his Ph.D. in 1992. In 1995, he was appointed Consultant and in 1998, he became associate professor in pediatrics. In 2001-2004 Mikael Norman worked as senior researcher at the Swedish Research Council. He received a chair as professor in pediatrics & neonatal medicine at KI in 2008. Between 1995 and 2015, he worked - besides being a clinician and researcher - in different administrative positions (head of department) at Karolinska university hospital. Mikael Norman has supervised 26 PhD-students and lists over 270 scientific papers with more than 18,000 citations. He is the editor of 3 Swedish textbooks in pediatrics and neonatology. At present, he works as professor at KI and director for the Swedish Neonatal Quality Register.

Research interests:

- Cardiovascular system in newborn infants and children, structure and function in health and disease
- Developmental origins of health and disease
- Preterm birth: risk factors, interventions and short and long-term outcome
- Perinatal epidemiology
- Patient safety and quality of care

Academic awards:

Swedish Society of Medicine: Regnell´s prize 2010 and 2014 for best scientific manuscripts (all categories), and Hugo Lagercrantz Award 2024 for distinguished international research.

Professor Bruno Piedboeuf, MD, FRCPC

Bruno Piedboeuf, MD, has done his medical school, his pediatric residency and neonatology fellowship at the University of Montreal. After completing an additional three years in Molecular Biology at the University of Rochester, he has joined, in 1993, the Pediatric Department at Université Laval and the CHU de Québec as a clinician researcher where he practiced neonatology for 30 years. From 2003 to 2012, he was the Chair of the Pediatric Department at Université Laval and the pediatric chief at the CHU de Québec, and from 2012 to 2017, he was the executive vice-dean of the Faculty of medicine of Université Laval. Since 2009, he is the president of the Table sectorielle mère enfant of the province of Quebec. In 2018, he was appointed Director of the University affairs for the Quebec Ministère de la Santé et des Services sociaux. His direction his responsible for the planification of the medical resources for the province.

He has maintained research activities over the years, and he is currently director of the Reproductive, maternal and child health research division of the CHU de Québec-Université Laval Research Centre and because of his involvement in different administrative functions in the educational and health-care system, his interest in using evidence based medicine to improve the organization of the health care system growth and has directed his interests in research. He is the new president of the board of governance of the Canadian Neonatal Network.Network in the shared aims of improving the organization of health care for pregnant women and newborns while supporting a new generation of health care researchers.

Professor Prakeshkumar Shah, MSc, MBBS, MD, DCH, MRCP, FRCPC

Dr Prakesh Shah is the Pediatrician-in-Chief at Mount Sinai Hospital, Toronto and Professor in the Department of Pediatrics and Institute of Health Policy, Management and Evaluation at the University of Toronto, Canada. He is the Director of the Canadian Preterm Birth Network (CPTBN) and an International Network for Evaluation of Outcomes of Neonates (iNeo) whereby he oversees a project of benchmarking outcomes of very preterm neonates in 13 countries with population-level neonatal networks with an overarching aim of improving quality of care across 240 NICUs. His areas of interest include Patient and Disease oriented research in Neonatal-Perinatal Medicine, Health Services and Epidemiological Research in Maternal- Newborn care, Knowledge Synthesis and Quality improvement. He has evaluated and produced policy documents on interventions and programs for preterm birth for provincial and national agencies. He is engaged in policy and advocacy work with his role in executive committees and advisory board membership as local, provincial, national, and international levels. He has led the Canadian Neonatal Network as Associate Director between 2010-12 and then was Director from 2012-2024.

Professor Emeritus Nalini Singhal, MBBS, FRCPC

Dr Singhal trained at the All India Institute of Medical Sciences in Delhi in India and did her residency and fellowship in Canada. Her clinical work has been with sick newborn babies both in level 2 and 3 units.

Administratively she developed and implemented resuscitation by respiratory therapists, served on the ILCOR committee for over 10 years, was Chair of the Canadian Neonatal Resuscitation Program committee and was the Regional Division Head of Neonatology in Calgary overseeing both the Level 2 and 3 units.

Her research interests are Resuscitation of the newborn, delayed cord clamping and implementation of newborn programs in resource limited areas and quality improvement. Educationally she has helped develop simple training programs for stabilization of the sick newborn and contributed to the Newborn Resuscitation Program.

In Canada, she has been part of the group for EPIQ (Evidence-based Practice for Improving Quality) that has helped decrease newborn mortality and morbidity across the country. For the past 25 years she has been involved in Global Child Health. Her work has involved contributing to development of 'Helping Babies Breathe,' a program for education in low resource settings, Essential Care for Every Baby and Essential Care of Small Baby. She works as a volunteer with the World Health Organization in developing and implementing newborn programs.

She was part of the team implementing Maternal Newborn and Child program in Tanzania, and Kangaroo Mother Care in Ethiopia. She was the principal investigator of SIM 4Life (A simulation program for Uganda, Tanzania and Nigeria).

Her focus is on implementation of programs that lead to knowledge translation and quality care.

Professor Erik D. Skarsgard MD, MSc, FRCSC, FACS, FAAP

Dr. Skarsgard is the Surgeon in Chief at BC Children's Hospital and professor of surgery at the University of British Columbia, Vancouver, Canada. He is a graduate of the UBC School of Medicine and received training in pediatric surgery at Toronto's Hospital for Sick Children and fetal surgery at the University of California San Francisco. He served on the pediatric surgical faculty at Lucile Packard Children's Hospital at Stanford from 1994-2001, and since 2001 has been at BC Children's Hospital in Vancouver.

Dr. Skarsgard is past-chair of the Advisory Board for the Institute of Human Development, Child and Youth Health (IHDCYH) at Canadian Institutes of Health Research (CIHR), Past-President of the Canadian Association of Pediatric Surgeons and past Governor of the American Pediatric Surgical Association and Advisory Council member for pediatric surgery for the American College of Surgeons. He is the founding Principal Investigator of the Canadian Pediatric Surgery Network (CAPSNet), which targets best practices for the treatment of surgical birth defects. He has published over 170 peer-reviewed articles, 8 textbook chapters and one textbook, and serves on the Editorial boards for the Journal of Pediatric Surgery and World Journal of Pediatric Surgery.

He has appeared as an invited witness to the Standing Committee on Health in the Canadian House of Commons to address children's surgical wait times on behalf of the Pediatric Surgical Chiefs of Canada.

Professor Ann R. Stark, MD, FAAP

Dr. Ann Stark graduated from Mount Holyoke College in 1967 and from Harvard Medical School in 1971, trained in pediatrics at St Louis Children's Hospital and Children's Hospital of Philadelphia, then returned to Boston to complete her fellowship at the new Joint Program in Neonatology, based at Boston Children's, Brigham and Women's, and Beth Israel Hospital. Upon completion of fellowship in 1977, she joined the Harvard Medical School faculty, and practiced in the NICU at Boston Children's, and became Director of the NICU, as well as Clinical Director of the Joint Program in Neonatology. In 2004, she was recruited by Dr. Ralph Feigin to Texas Children's Hospital and Baylor College of Medicine, where she became Director of the Neonatal-Perinatal Medicine Fellowship Program and Head of the Section of Neonatology. After Dr. Feigin's untimely death, she moved to Vanderbilt in 2011, where she directed the Neonatology Fellowship Program and oversaw the Fellowship Programs in the Department of Pediatrics. In 2018, Dr. Stark returned to Boston to join the Department of Neonatology at Beth Israel Deaconess Medical Center (BIDMC) where she is currently Director of Faculty Development and Professor in Residence in Pediatrics at Harvard Medical School in the Department of Neonatology at BIDMC. Working with colleagues, Dr. Stark developed the American Academy of Pediatrics NICU Verification Program and led the development of Standards of Neonatal Care, published in Pediatrics in 2023. Her research interests have focused primarily on respiratory physiology and care. She has held leadership positions including serving as Chair of the AAP Neonatal Perinatal Section, the AAP Committee on Fetus and Newborn, and Chair of the American Board of Pediatrics Sub-board on Neonatal Perinatal Medicine, of which she was also the medical editor.

Distinguished Professor Geoff Chase, FRSNZ, DistFEngNZ

Professor Geoff Chase received his B.S. from CWRU in 1986, with M.S. and PhD from Stanford University (1991, 1996). He spent 6 years working for General Motors and 5 years in Silicon Valley, including Xerox PARC, ReSound, Hughes Space and Communications, and Infineon Technologies, before joining the University of Canterbury, where he is its inaugural Distinguished Professor.

His research focuses on the intersection of engineering and clinical medicine, primarily in intensive care, metabolic disease, and cardio-pulmonary diseases, based on close clinical collaborations and clinical engineering overlap to achieve best outcomes.

These efforts have led to a range of model-based systems to improve care and outcomes, and reduce costs, which are in clinical trials or standard of care use in both adult and neonatal intensive care units (ICU, NICU). Dr. Chase has published over 1800 journal and conference papers and 20 US and European patents. He founded 3 startup companies, and is a Fellow of the Royal Society of NZ (FRSNZ) and Distinguished Fellow of Engineering NZ (DistFEngNZ) among others.

CASE SUMMARIES

BABY 2 SUMMARY

Baby 2 was a 31+2/7 week, 1.69 kg birth weight, twin 1, female infant, who was delivered by emergency caesarean section for maternal hypertension. Mother had anti-phosphilipid syndrome and gestational cholestasis (not charted in medical records). At birth, Baby 2 was pale, floppy and had low heart rate (<60/min). She was resuscitated and intubated with size 2.5 endotracheal tube after 3 attempts. Vocal cords were red, swollen and inflamed. There was a large air leak so the endotracheal tube was removed and continuous positive airway pressure (CPAP) was applied. Chest x'ray was consistent with hyaline membrane disease. She remained on CPAP as her respiratory status was unstable. Two days later, she developed sudden apnoea, bradycardia, and purple discoloration of the skin with white patches. She was resuscitated, recovered and discharged home a month later.

CONVICTION

It was alleged that Baby 2 collapsed from injection of air into the intravenous line, causing air embolism resulting in collapse and patchy purple discolorations of the skin.

PANEL OPINION

On the day of collapse, Baby 2 had a kinked and impacted long venous line, which was not heparinized (treated with anti-coagulants), and which can predispose to thrombosis. Thrombosis of long venous catheters occurs in 20-30% of infants (Cochrane Database of Systematic Reviews 2008, Issue 2. Art. No.: CD002772. DOI: 10.1002/14651858.CD002772.pub3. Accessed 16 January 2025). The risk of thrombosis was further increased because mother had anti-phospholipid syndrome, which is a condition in which the immune system mistakenly creates antibodies that attack tissues in the body, and can trigger blood clots to form in arteries and veins. During pregnancy, antibodies can pass through the placenta to the neonate and lead to thromboembolism. The sibling (Baby A) suffered similar collapse and died one day after birth, and post mortem showed a recent non-occluding thrombus in the liver, indicating a recent thrombotic event. Consequently, Baby 2 was at high risk for thrombosis. On the day of collapse, Baby 2 had high D-dimer of 6219 (normal 220-500 ng/ml) and low platelets of 103, both indicative of thrombosis. Fortunately, Baby 2 survived so we have no post mortem evidence of thrombosis.

Patchy skin discolorations are caused by dilation and contraction of small blood vessels in the skin in response to hypoxia, which can occur in many conditions and are not diagnostic of air embolism. Zhou and Lee reported that non-specific localized patchy skin discolorations have not been reported in infants with venous air embolism, including IV air injection, and that only Lee's sign and Liebermeister's sign are diagnostic of air embolism (Zhou Q, Lee SK. Am J Perinatol 2024 Dec 27. doi: 10.1055/a-2508-2733). If air was deliberately infused through a central venous line to cause air embolism, the line will have to be reinfused with fluid to prevent detection. Collapse from air embolism occurs instantaneously. It is doubtful that this can be achieved quickly enough before other staff in the unit respond to the collapse.

CONCLUSIONS

1. Baby 2 collapsed from thrombotic emboli originating from a central intravenous catheter, which was potentially aggravated by maternal anti-phospholipid syndrome.
2. There was no evidence to support air embolism.

BABY 3 SUMMARY

Baby 3 was born at a gestational age of 30 weeks and 1 day. His intrauterine growth had been extremely poor. His birth weight was 800g (average birth weight of a Baby 2orn at 30 weeks gestation is around 1.4kg) and there had been reversed end-diastolic (abnormal) blood flow. He was hence at high risk of intrauterine demise, stillbirth, and if live-born, multiple immediate, short-term, and long-term complications. The decision to deliver him at a non-tertiary unit was therefore questionable.

He was cold on admission to the neonatal unit. He had low white cell and platelet counts, which are consistent with severe intrauterine growth restriction (IUGR) and not necessarily infection. He developed respiratory distress syndrome and was given surfactant. Chest X-rays showed respiratory distress syndrome, not pneumonia, and that surfactant had been administered mainly into the right lung.

Over the next 3 days Baby 3 showed multiple clinical signs of intermittent bowel obstruction (no bowel opening from birth; dark bilious aspirates; bilious vomit; intermittent stomach and small bowel distension; recurrent crying). No bowel movements and dark bile aspirates and vomiting are clear indication for urgent surgical opinion. This did not occur. Instead despite feeds being contraindicated, he was given a 0.5ml nasogastric feed. He collapsed with a major apnoea 15 minutes later.

Two nurses commenced resuscitation, one of whom was extremely inexperienced and had never done cardiac compressions before. The medical registrar who was called had 3 unsuccessful attempts at intubation before the consultant arrived. Baby 3 therefore would appear to have had ineffective ventilation for at least 20 minutes which would have resulted in marked respiratory and metabolic acidosis and made subsequent resuscitative efforts less likely to be successful. He received seven boluses of adrenaline, three of saline, two of sodium bicarbonate and one of calcium gluconate. Following discussion with his parents a decision was made to stop active resuscitation but to continue ventilation until he was baptized. He lived for 5 hours following cessation of ventilatory support.

Post-mortem showed an aberrant descending colon with persistence of the mesentery (a membrane that normally disappears in fetal life thereby fixing the colon to the back of the peritoneal cavity). With a persisting mesentery the descending colon is mobile and displaced to the right, with the potential for transmesenteric (internal) herniation of small bowel to the left of the descending colon causing obstruction that could be intermittent. Post-mortem also showed widespread hypoxic- ischaemic damage to the heart, lung immaturity, and other changes consistent with severe IUGR.

CONVICTION

It was alleged that air had been deliberately injected into Baby 3 through the nasogastric tube sufficient to cause his collapse.

PANEL OPINION

Baby 3 did not respond adequately to prolonged resuscitation because of inadequate resuscitation for 20 minutes following an acute episode of apnoea. Recovering respiratory distress syndrome and severe growth restriction with myocardial ischaemia, would have added further to the likelihood of a poor response to resuscitation. The apnoeic attack was likely precipitated by severe pain due to a further episode of acute small bowel obstruction

precipitated by feeding. The suggestion that the multiple signs of intestinal obstruction could be caused by injection of air through the nasogastric tube is untenable.

CONCLUSION

1. Baby 3 died because of a decision to discontinue respiratory support and resuscitative effort in the face of a poor response following inadequate resuscitation for at least 20 minutes after an acute episode of apnoea.
2. The clear prior signs of intermittent bowel obstruction that warranted urgent surgical opinion and investigation had gone unrecognized.

BABY 5 SUMMARY

Baby 5 was a 29+5/7 week, 1327 grams birth weight, twin 1, male infant, who was born by semi- elective Caesarean section for twin-twin transfusion syndrome, with oligohydramnios. Antenatal ultrasound showed dilated small bowel loops and absent/reversed end diastolic flow. Baby 5 required bagging at birth and continuous positive airway (CPAP) for apnoea. He showed signs of infection with low white cell and neutrophil counts, and high blood sugar, which were treated with antibiotics and insulin. Chest x'ray was clear. Four days later, he developed respiratory distress (desaturation, chest recession, oxygen need) and bilious aspirates from the nasogastric tube, but the abdomen was soft and not distended. He had two episodes of massive gastrointestinal bleeding with at least 25% of estimated total blood volume aspirated from the nasogastric tube. He was given normal saline but 40 minutes later, he suddenly deteriorated, with desaturation, poor perfusion, low heart rate 80- 90/min, and purple patches of discoloration over the abdomen. An hour later, he collapsed again and died despite resuscitation efforts.

CONVICTION

It was alleged that Baby 5 died from inflicted trauma causing upper GI hemorrhage, and intravenous injection of air, causing air embolism resulting in collapse, patchy discolorations of the skin and death.

PANEL OPINION

Baby 5 was at high risk because he was preterm, had twin-twin transfusion with oligohydramnios, and his antenatal ultrasound showed reversed end diastolic flow and dilated small bowel loops. This meant blood was being sucked out of the fetus at the end of each cardiac cycle, and the intestines were likely damaged before birth. The 2 episodes of massive gastrointestinal haemorrhage were most likely due to in-utero hypoxia causing stomach or small intestinal ulceration, and erosion into an intestinal blood vessel; or to a vascular abnormality like Dieulafoy's lesion, which can cause life- threatening hemorrhage. His 25% blood volume loss was likely an underestimate because more was likely lost in the intestines. Emergency transfusion with group O negative blood should have been immediately given earlier during resuscitation. Since 20% blood loss causes shock, and Baby 5 lost much more, this was fatal. Patchy skin discolorations are caused by dilation and contraction of small blood vessels in the skin in response to hypoxia, which can occur in many conditions and are not diagnostic of air embolism. Zhou and Lee reported that non-specific localized patchy skin discolorations have not been reported in infants with venous air embolism, including IV air injection, and that only Lee's sign and Liebermeister's sign are diagnostic of air embolism (Zhou Q, Lee SK. Am J Perinatol 2024 Dec 27. doi: 10.1055/a-2508-2733). If air was deliberately infused through a central venous line to cause air embolism, the line will have to be reinfused with fluid to prevent detection. Collapse from air embolism occurs instantaneously. It is doubtful that this can be achieved quickly enough before other staff in the unit respond to the collapse.

CONCLUSIONS

1. Baby 5 died from massive gastrointestinal hemorrhage due to either intrauterine hypoxia causing stomach or intestinal ulceration or a congenital vascular lesion.
2. Emergency blood transfusion should have been given much earlier.
3. There was no evidence of air embolism
4. Post-mortem should have been requested

BABY 8 SUMMARY

Baby 8 was a 34+4/7 week, 2.33 kg birth weight, female infant who was born by emergency caesarean section for maternal diabetes type 1 with labile glycemic control. She had respiratory distress syndrome (RDS) with grunting, subcostal retractions, respiratory and metabolic acidosis, and needed oxygen, but continuous positive airway pressure (CPAP) support was not provided until 4 hours later. BIPAP was started the next day as the infant did not improve but chest x'ray was not done. The following day, she was intubated and ventilated for desaturation and gasping. She developed a life threatening tension pneumothorax that was not diagnosed for 2½ hours and was not treated for 1½ hours after diagnosis. She deteriorated and a chest tube was inserted to drain the pneumothorax but a lateral chest x'ray was not performed to check its position. Its malposition led to incomplete evacuation and re-accumulation of the tension pneumothorax. During the next 16 hours, there were 4 episodes of severe desaturations. A second chest tube was inserted but it was not done until 2 hours after a check chest x'ray. The infant was ventilated with high pressures (26/5, rate 40/min, Ti 0.45) and continued to deteriorate over the next day with multiple episodes of desaturations. Despite poor blood gases and re-accumulation of the pneumothorax, it was not drained. Ventilation was further increased to pressure 26/5, rate 60/min, FiO2 100%. A third chest tube was inserted but 3 hours after a check chest x'ray showed re-accumulation of the tension pneumothorax. The infant finally improved and was transferred to Arrowe Hospital.

CONVICTION

It was alleged that Baby 8's clinical deteriorations at around 00.55 hr and 03.30 hr on 27/9/15 were the result of deliberate dislodgement of the endotracheal tube.

PANEL OPINION

This is a straight forward case of a relatively large preterm infant with respiratory distress syndrome who developed a tension pneumothorax. The infant had respiratory distress from birth but was not given CPAP until 4 hours later. Without treatment, the infant grunts to exert chest pressure in an effort to keep the small air spaces in the lung open. This can lead to pneumothorax. Tension pneumothorax is life threatening and should be drained immediately. If done promptly and properly, recovery is quick. Unfortunately, that did not happen. There were repeated and lengthy delays in diagnosis and treatment for the tension pneumothorax, and poor placement of chest tubes with ineffectual removal of air in the chest, resulting in re-accumulation of air in the chest and continuing clinical deterioration. In response, high ventilation pressures were used in an attempt to ventilate the infant but this worsened the situation because excessive pressures compromised venous return to the heart and further impaired circulation and oxygenation. Baby 8 was subjected to multiple invasive procedures (including at least 6 intubations, 7 chest needles, 3 chest drains, and multiple hand bagging episodes), and prolonged period of illness. There was delay in transferring the infant to a higher level facility. We did not find any evidence to support malicious actions like endotracheal tube dislodgement on 27/9/15.

CONCLUSIONS

1. Baby 8's deteriorations were due to medical mismanagement of the tension pneumothorax.
2. There was no evidence of intentional tampering with the endotracheal tube.
3. One prosecution expert witness recognized that care of the pneumothorax was sub optimal

BABY 10 SUMMARY

10 was a 32+2/7 week, 1.709 kg birth weight, twin female infant who was born by elective caesarean section to a mum with twin-twin transfusion/laser treatment, and preterm prelabour rupture of membranes. She had laparotomy with small bowel resection and stoma formation of functioning ileostomy and mucous fistula for malrotation/volvulus and necrotic bowel with perforation and adhesions. She developed stomal excoriation and formation of a mucocutaneous fistula. At 17 days of age, she developed seizures and was treated with antibiotics and Phenobarbitone. At 47 days of age, she was found blue and crying, then became pale and was resuscitated with cardiac compressions and bagging because of low heart rate (40/min). She recovered quickly and was transferred to Alder Hey Hospital, where she was diagnosed with sepsis. She had another similar episode of clinical deterioration at Alder Hey Hospital about 10½ hours after her transfer.

CONVICTION

It was alleged that 10 deteriorated on day 47 of life because of malicious airway obstruction.

PANEL OPINION

10 is a preterm infant with bowel resection and stoma formation with excoriation. She also had seizures that were treated with phenobarbitone. Following transfer to Alder Hey Hospital on day 47 of life, she was diagnosed to have an infection and was treated with antibiotics. She had another similar episode of clinical deterioration at Alder Hey 10½ hours later.

It is most likely that the clinical deterioration on day 47 of life at the Countess of Chester Hospital (CoCH) was an episode of seizure, apnoea or hypoventilation, which was an early manifestation of the sepsis that was subsequently diagnosed after she was transferred to Alder Hey Hospital. Infection does not just happen suddenly. It brews in the body over time, usually 1-2 days before it overwhelms the body's defences and clinical symptoms occur. The episode of deterioration in question is most likely part of the same infection episode and cannot be treated as a separate event. The occurrence of a similar episode of deterioration at Alder Hey Hospital supports the diagnosis of sepsis as the cause of the episode at CoCH.

The resuscitation with chest compressions seems to be an overreaction from the nurses. Dr Soni, who responded immediately to the crash call, recorded that the heart rate was 46/min on the monitor but >100/min on auscultation and the infant was crying under the mask while cardiac massage was ongoing. The nurses reported that the infant was breathing throughout but laboured. Bagging or oxygen would be appropriate for desaturation but not cardiac compressions because it can cause harm to the infant, including trauma to the heart and other organs, rib fractures and disruption of circulation. Of note, the parents were concerned about the care their baby was receiving in CoCH. They requested to have the Baby 14ot transferred back to CoCH and were successful.

CONCLUSIONS

1. 10's deterioration on day 47 of life was due to sepsis.
2. There is no evidence to support malicious airway obstruction.

BABY 12 SUMMARY

Baby 12 was a 33+2/7 week, 1.465 kg birth weight, twin 1, severe intrauterine growth restricted (IUGR) preterm male infant, who was delivered by caesarean section for poor growth and oligohydramnios at 1013 hours. On admission to the NICU, his initial blood glucose was 1.9 and he was started on intravenous 10% dextrose at 100 ml/kg/day. The next day, he was kept on intravenous TPN at 75 ml/kg/day plus 10% dextrose at 1 ml/kg/day plus nasogastric feeds. His blood glucose was 3.6 at 0054 hours but dropped after that and was low throughout the day (1.5 to 1.9). Blood tests showed c-peptide 264, Insulin 1079. At 1920 hours, his dextrose infusion was increased to 12.5% and blood glucose improved to 2.0 to 2.4. The next day, his blood glucose ranged from 2.1 to 2.4. The next day, a long line was inserted and his dextrose infusion was increased to 15% at 0130 hours and fluid volume increased at 0700 hours. His blood glucose stabilized after that from 2.7 to 3.0. Feeds were increased, the intravenous infusion was weaned off, and he was discharged home.

CONVICTION

It was alleged that Baby 12 received exogenous insulin which caused hypoglycemia. The evidence is based on a high insulin to low peptide level ratio (I/C ratio).

PANEL OPINION

It is common for preterm and IUGR infants to have hypoglycemia, due to their limited glycogen and fat stores, inability to generate new glucose using gluconeogenesis pathways, higher metabolic demands due to a relatively larger brain size, and inability to mount a counter-regulatory response to hypoglycemia. Baby 12's blood glucose dropped from 0054 hours on day after admission but his dextrose concentration was not increased until 1920 hours. This is a long interval without adequate sugar and intervention should have been earlier. His blood sugar improved in response to 2.0 to 2.4. However, this is still low and further intervention was necessary. Again, there was delay, and his glucose concentration was not increased to 15% until the next day at 0130 hours and the volume was not increased until 0700 hours. His blood sugars improved to normal range after that. The fact that his blood sugar improved each time the glucose infusion increased indicates that the hypoglycemia persisted because insufficient dextrose was given for this infant's needs. Chase et al reported that premature infants have different normative standards for insulin and c-peptide than adults. The Insulin:C-peptide (I/C) ratio does not prove exogenous insulin was administered because the C-peptide was not low for preterm infants (20-45 percentile), potassium levels were normal (insulin decreases potassium), antibodies can store insulin in the blood, glucose levels should be lower if exogenous insulin was used, the infant's glycaemic profile was inconsistent with insulin administration but consistent with the delivered IV feeding profile, the I/C ratio was within the expected range for preterm infants, and the immunoassay test is unreliable because interference factors can give false positive insulin readings.

CONCLUSIONS

1. Hypoglycemia was due to preterm birth and severe IUGR; it's medical management was inadequate.
2. Baby F's insulin level and I/C ratio do not prove that exogenous insulin was used, and are within the norm for preterm infants. Preterm infants and those will illness have different normative standards compared to healthy adults and older children.

BABY 13 SUMMARY

Baby 13 was a 33+2/7 week, 1.703 kg birth weight, twin 2, preterm male infant, with severe intrauterine growth restriction (IUGR) who was delivered by elective Caesarean section for oligohydramnios and IUGR of the first twin. He was born breech and bagged at birth for being slow to pink. He was plethoric (Hb 190), jaundiced (serum bilirubin 57, above phototherapy threshold), hypoglycemic (blood sugar 1.9) and was treated for possible sepsis with antibiotics and phototherapy. His white cell count and CRP were not raised. At 1215 hours the next day, he developed fever which subsided with change in environmental temperature, increased work of breathing, distended abdomen and bile stained nasogastric (NG) aspirates. The nurse was concerned enough to call the registrar, who stopped feeds and put the nasogastric tube to free drainage. Four hours later, Baby 13 developed apnoea with desaturation and low heart rate. He was resuscitated, given cardiac massage, adrenaline X6 doses, bicarbonate X2 doses, saline X2 doses, intubated and ventilated. Strangely, 2 doses of adrenaline were given before his airway was secured. He stabilized and was extubated the next day. A Eustachian valve was detected on cardiac echo. He improved and was discharged at a month of age.

CONVICTION

It was alleged that air was injected into the intravenous system, causing air embolism and collapse.

PANEL OPINION

Baby 13 had signs of possible sepsis from birth, including jaundice requiring phototherapy and hypoglycemia, and was treated with antibiotics. The next day, he clinically deteriorated for 4 hours prior to collapse, with abdominal distension, increased work of breathing, raised temperature, and bile-stained NG aspirates, and which were of concern to the nurse and registrar. The acute episode of desaturation and bradycardia was likely triggered by apnoea from sepsis, abdominal distension with ileus, and prematurity. It could also be due to the cardiac Eustachian valve, which can cause episodes of desaturation and bradycardia due to intermittent right to left shunting of blood through the foramen ovale. The resuscitation was chaotic, with adrenaline X2 doses given before the airway was secured. Air embolism is sudden and catastrophic and does not present over 4 hours with gradual deterioration, and the infant would not recover quickly. Patchy skin discolorations are caused by dilation and contraction of small blood vessels in the skin in response to hypoxia, which can occur in many conditions and are not diagnostic of air embolism. Non-specific localized patchy skin discolorations have not been reported in infants with venous air embolism, including IV air injection, and only Lee's sign and Liebermeister's sign are diagnostic of air embolism (Zhou Q, Lee SK. Am J Perinatol 2024 Dec 27. doi: 10.1055/a-2508-2733). If air was deliberately infused through a central venous line to cause air embolism, the line will have to be reinfused with fluid to prevent detection. Collapse from air embolism occurs instantaneously. It is doubtful this can be achieved quickly enough before other staff in the unit respond to the collapse.

CONCLUSIONS

1. Baby 13's desaturation and bradycardia was caused by apnoea from sepsis or the Eustachian valve.
2. The resuscitation was suboptimal.

3. There was no evidence of air embolism.

BABY 14 SUMMARY

Baby 14 was a 34+4/7 week, 1.67 kg BW, twin 2, preterm male infant, with severe intrauterine growth restriction (IUGR) who was delivered by emergency caesarean section. Mother was a haemophilia carrier. Baby 14 had mild respiratory distress syndrome, reduced factor VIII 3% (moderate) and raised coagulation times (PT 15.4, APTT 69.7). Twelve hours later, he desaturated, looked dusky, was unsettled, and had increased work of breathing. When seen by a doctor, he was screaming and had subcostal recession, but settled after half an hour. On day 13, he became mottled and had 5 episodes of desaturation, and an episode of profound desaturation. Intubation X3 was attempted but abandoned due to blood in oropharynx and trauma due to repeated intubation attempts. Chest x'ray showed right patchy consolidation consistent with infection and oedema. Intravenous Factor VIII was given. Intubation was re-attempted X5 by registrars, consultants and anesthetist without success. A laryngeal mask was used. A team from Alder Hey Hospital arrived 11 hours after onset of profound desaturation and intubated the infant. He was transferred to Alder Hey Hospital, given Factor VIII and recovered.

CONVICTION

It was alleged that the initial screaming episode was due to inflicted injury or injection of air into his intravenous system causing pain. It was alleged that the desaturation and bleeding on day 17 were caused by trauma inflicted by thrusting a nasogastric tube into the back of Baby 14's throat.

PANEL OPINION

The initial screaming episode is unlikely to be due to inflicted injury because injury of such intensity to cause 30 minutes of screaming would have caused bleeding. It is not due to air embolism because crying due to air embolism is of short duration; it is a response to hypoxia as the infant gasps and cries for air, and quickly resolves or the infant collapses. Crying is usually associated with discomfort from hunger, hypothermia, hypoxia and pain. The desaturation on day 17 was most likely due to bleeding from haemophilia, either spontaneously or from routine cares causing blood and secretions to pool in the oropharynx and obstruct the airway. Moderate haemophilia can cause spontaneous bleeding. It is unclear if a haemophilia nursing protocol was used by the unit to prevent inadvertent trauma from standard nursing care procedures. If it was due to repeated thrusting of a nasogastric tube into the throat, the infant would have cried or screamed, and there would be evidence of lacerations in the pharynx, esophagus and vocal cords. None were reported by the intubating physicians. Trauma was most certainly inflicted during the repeated intubation attempts (8X), which aggravated the bleeding and caused epiglottis swelling. In a haemophilia patient, intubation should be performed by an experienced individual. It is inappropriate to allow junior doctors to try, and then progress up the chain to senior doctors. The patient should not have been subjected to 8 intubation attempts. The referral to Alder Hey Hospital and transfer should have been done much sooner, not 11 hours after initial desaturation.

CONCLUSIONS

1. Crying was not due to injury or air embolism.
2. The subsequent episode of desaturation was due to bleeding, likely spontaneously from haemophilia or routine cares, and exacerbated by repeated traumatic intubation attempts.

BABY 16 SUMMARY

Baby 16 is a 33+2/7 weeks, 2.066 kg birth weight, triplet 1, male infant born by caesarean section for maternal discomfort. Infant had mild respiratory distress and was put on continuous positive airway pressure (CPAP). At 16 hours of age, CPAP was changed to nasal cannula, but 3½ hours later he developed apnoea. Abdomen was full and mildly distended, soft with no tenderness and active bowel sounds, but with mild erythema at the base of the umbilicus. Antibiotics were started. The next day, he suddenly developed desaturation and low heart rate, and was intubated and ventilated with high pressures 26/6 rate, 60/min, FiO2 26% and given adrenaline. Chest x'ray showed a moderately large right pneumothorax. An hour later, he again developed desaturation and low heart rate. Capnography showed the endotracheal tube was dislodged. He was resuscitated, and a new endotracheal tube was inserted. The pneumothorax was needled about 3 hours after the first deterioration, and a chest tube was inserted another 2½ hours later. He had persistent metabolic acidosis despite bicarbonate infusion. One and half hours later, he had cardiorespiratory arrest and died.

CONVICTION

It was alleged that air was injected into Baby 16's stomach by nasogastric tube, which caused gastric distension and splinting of the diaphragm, leading to collapse. The allegation has since been changed to air was later injected into his intravenous line to cause air embolism, collapse and death.

PANEL OPINION

The initial gaseous distension of the abdomen was caused by CPAP, and not by injection of air into the nasogastric tube. It is unclear whether the nasogastric tube was open to drainage. Baby 16's care after the collapse on 24 June was mismanaged and there were unacceptable delays in recognition and treatment of the pneumothorax. Delay in treatment of the pneumothorax led to persistent metabolic acidosis because of persistent poor circulation and gas exchange caused by the pneumothorax. It is unclear whether the pneumothorax preceded or followed the resuscitation. Pneumothorax can occur in preterm infants with mild respiratory distress syndrome, especially with CPAP or other respiratory support, or even without any lung disease or respiratory support. The medical records do not show that a cold light test was performed. He was treated with high pressure ventilation and adrenaline despite having no lung disease and requiring only 26% oxygen. Both are harmful because high ventilation pressures impede venous return to the heart, resulting in worsening circulatory failure, and adrenaline punishes the heart by overworking it unnecessarily. There were no signs of air embolism at final collapse.

CONCLUSIONS

1. Baby 16 died from pneumothorax that was suboptimally managed
2. There was no evidence of air injection into the stomach or into the intravenous line.

BABY 17 SUMMARY

Baby 17 was a 31+3/7 week, 2.076 kg birth weight, male infant who was born by emergency caesarean section for intrapartum haemorrhage from placenta previa accreta. He was blue, gasping and had poor tone and heart rate 60-100/minute at birth. He was given inflation breaths, intubated, ventilated and given surfactant, intravenous fluids and antibiotics. Chest x'ray was consistent with transient trachypnoea of the newborn. Twelve hours later, he was extubated to continuous positive airway pressure (CPAP). The next day, CPAP was discontinued and he was started on phototherapy and trophic feeds. On day 3, he developed frequent episodes of self-limited bradycardia. On day 4, he had moderate amounts of aspirates. At 0900 hours, he "vomited clear fluid nasally and from mouth, desaturation and bradycardia, mottled ++. Neopuff and suction applied. Air ++ aspirated from nasogastric (NG) tube." His gases showed respiratory acidosis and he was put on CPAP. At 1920 hours, he was electively intubated and ventilated and his gases improved. The next day, he had bilious aspirates and palpable loops of bowel in the abdomen. Abdominal x'ray showed a loop of mildly dilated bowel. He was treated as necrotizing enterocolitis (NEC) and transferred to Alder Hey Hospital, where he was treated conservatively for NEC and transferred back to CoCH. He recovered and was discharged home.

CONVICTION

It was alleged that Baby 17 was given an injection of air through the NG tube, causing distension of the abdomen, splinting of the diaphragm and collapse, because air ++ was aspirated from the NG tube during resuscitation.

PANEL OPINION

Baby 17 most likely vomited because he was developing early NEC or sepsis. His symptoms of self- limited bradycardia the day before, plus the subsequent symptoms of bilious aspirates, palpable intestinal loops, acidosis and the dilated bowel seen on abdominal x'ray, are all consistent with NEC or sepsis. Desaturation and mild bradycardia are commonly associated with vomiting, especially when there is infection or NEC, and the episode that Baby 17 exhibited was consistent with this pattern. The nursing notes clearly stated that Neopuff and bagging were applied after his vomiting, and ++ air was aspirated from the NG tube after the bagging. So, the ++ air aspirated from the NG tube were most likely caused by the bagging. The abdomen was subsequently described as soft and not tender and the abdominal x'rays did not show any gaseous distension of the intestines. If air that been pumped into his stomach sufficient to splint the diaphragm and cause collapse, the abdomen would have been very distended, tense and hard, and very noticeable. Yet the nursing notes and medical notes do not mention this. Such a large amount of air would not disappear so quickly and the subsequent abdominal x'rays would show ++air persisting in the intestines. The subsequent course of illness was consistent with NEC or sepsis. The subsequent respiratory management was unsatisfactory, with long delays in escalating ventilation when gases were sub-optimal, which resulted in Baby 17 remaining acidotic and not well ventilated.

CONCLUSIONS

1. Baby 17's deterioration with vomiting was because he was developing early necrotizing enterocolitis or sepsis.
2. There was no evidence to support air injection into the stomach through the nasogastric tube.

SUMMARY OF FINDINGS

COUNTESS OF CHESTER HOSPITAL FINDINGS

To summarize, we found numerous problems in medical care related to the 17 cases, including:

1. Medical histories were incomplete
2. Failure to consider the obstetric history
3. Disregard for surveillance warnings about infectious bacterial colonization
4. Misdiagnosis of diseases
5. Caring for patients that were beyond their designated level of care
6. Unsafe delays in diagnosis and treatment of acutely ill patients
7. Poor skills at resuscitation and intubation
8. Poor supervision of junior doctors in procedures like intubation
9. Poor skills in basic medical procedures like insertion of chest tubes
10. Lack of understanding about respiratory physiology and basics of mechanical ventilation
11. Poor management of common neonatal conditions like hypoglycemia
12. Lack of knowledge about commonly used equipment in the NICU, e.g. Neopuff, capnograph
13. Failure to protect at risk patients (e.g. haemophilia) from trauma during intubation
14. Lack of teamwork and trust between the health professions

GENERAL FINDINGS

Statements given by witnesses point to serious resource and infrastructure deficiencies that impact on general patient care at the Countess of Chester Hospital. Specific concerns expressed in witness statements include:

1. Inadequate numbers of appropriately trained personnel
2. Lack of training for assigned nursing roles
3. Inadequate staffing
4. Work overload
5. Poor plumbing and drainage, resulting in need for intensive cleaning; this was a potential factor in Stenotrophomonas maltophilia colonization and infection
6. Poor environmental temperature control in facility
7. Difficulty in finding a doctor when need arose
8. Congestion at medication cabinet and preparation trolley
9. Lack of appropriate facilities for sterile preparation, e.g. IV drugs prepared in corridor
10. Some high risk infants who should have been born and cared for at higher level institutions were born and cared for in Countess of Chester Hospital because of a shortage of beds at higher level facilities where they should have been admitted
11. There were delays in transfer of sick infants to higher level facilities when the need arose

CONCLUSIONS

CONCLUSIONS

1. There was no medical evidence to support malfeasance causing death or injury in any of the 17 cases in the trial
2. Death or injury of affected infants were due to natural causes or errors in medical care
3. There were problems related to the medical care of patients at the Countess of Chester Hospital neonatal unit
4. There were problems related to teamwork and inter-disciplinary collaboration at the Countess of Chester Hospital neonatal unit