

Postpartum urinary incontinence

The problem is clear, but there is no simple solution

Papers p 1241

Postpartum urinary incontinence is an important and often overlooked form of maternal morbidity. In this issue Chiarelli and Cockburn (p 1241)¹ highlight and confirm the work of other investigators who have shown that vaginal delivery induces urinary incontinence, especially the first vaginal birth.² Many clinical studies have attempted to discover the particular obstetric event that causes the incontinence. The obvious suspects include large babies and “difficult deliveries” marked by lengthy pushing phases with or without instrumentation. No clear single event has been found to be responsible, suggesting that postpartum urinary incontinence arises from a multifactorial physiological insult. The consequences of this pathophysiology are not limited to urinary incontinence. Pelvic organ prolapse (cystocele, rectocele, and uterine prolapse) and anal incontinence are also troublesome sequelae of vaginal delivery. These prevalent pelvic problems receive even less than the scant attention paid to postpartum urinary incontinence.^{3 4}

While the problem is clear, there is no simple solution. Prevention is rarely discussed among caregivers of urinary incontinence possibly because at this time the price of prevention is major surgery. Several studies suggest even this protection may fade with repeated abdominal deliveries.⁵ It is understandable that this method of prevention has not been met with widespread support. Avoidance or modification of specific obstetric techniques has not been shown to prevent postpartum urinary incontinence. In this vacuum of scientific uncertainty, emotion rapidly fills the void. Patients and physicians alike respond to the scientific uncertainty with preferences based on their personal convictions.⁶

If we cannot prevent the damage that causes postpartum urinary incontinence, it is reasonable to attempt to mitigate the damage. Chiarelli and Cockburn conducted a randomised trial to see if instructions to patients and postpartum pelvic rehabilitation would be beneficial. They provided new mothers with a comprehensive bladder programme, including enhanced information about healthy bladder habits and teaching with reinforcement regarding muscle training. Although the authors report a slight effect, the reader is struck by the high rates of incontinence even with such conscientious rehabilitation efforts: only 7% of incontinent new mothers reduced symptoms, leaving most symptomatic women untreated. Even these excellent efforts were grossly insufficient for the many new mothers who

develop postpartum urinary incontinence, and these young women are likely to continue to experience the indignity of urinary incontinence for many decades to come.⁷

We are left pondering the appropriate individual balance between vaginal birth and pelvic floor health. Large, longitudinal studies with well characterised populations and carefully described outcome measures will be essential to gather information for counselling patients. Recommendations about routes of delivery or the conduct of labour must include maternal pelvic floor morbidity, using appropriate windows of postpartum observation. Surgical trials would not be published if the authors reported surgical morbidity only during the procedure. Obstetric providers must look broadly at pelvic morbidity in order to optimise the care of maternity patients.

Clearly the risk of postpartum urinary incontinence exists, and abdominal delivery without labour markedly reduces the risk. Ongoing research will give us additional information about individual patients who are particularly susceptible to damage, possibly because of their constitutional make up or their particular obstetric situation. Meanwhile, how shall we counsel patients, especially those who particularly abhor the risks of pelvic floor damage?

Each woman should have sufficient information to determine which set of risks she prefers for herself and her baby. In the common situation where there is no additional risk to a baby, obstetric management should focus on reducing maternal morbidity including postpartum urinary incontinence. New mothers are likely to benefit from routine symptom screening and early discussion of healthy bladder habits and proper muscle techniques as part of their postpartum care. Obstetric care should include assessment of the maternal outcome of that birth, including the complete range of pelvic floor injuries known to be associated with childbirth.

Chiarelli and Cockburn are to be commended for bringing high quality science to the neglected problem of postpartum urinary incontinence. This problem cries for attention and a scientific approach to prevention or early intervention. Rather than confining our intellectual discourse to a dichotomous discussion regarding route of delivery, creative approaches are needed to develop a balance that optimises both maternal and child health. Some say that the mark of a civilised nation is the care that the new mother and her child receive. With one of three new mothers

becoming incontinent of urine, how would we be judged?

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Panayiotopoulos syndrome

A common benign but underdiagnosed and unexplored early childhood seizure syndrome

Epilepsy affects 1% of the general population and 4% of children, encompassing heterogeneous seizure syndromes.¹ These are defined by distinct aetiology, age at onset, seizure type, and electroencephalographic features, which taken together provide the key to diagnosis, prognosis, and optimal management. Over the past two decades various distinct paediatric epilepsy syndromes, such as rolandic epilepsy, have been formally recognised.² Panayiotopoulos syndrome is a new idiopathic childhood epilepsy, recently recognised by the International League Against Epilepsy.^{2,3} It is common, benign, and may mimic other common illnesses.

Awareness of this syndrome is important for all professionals who care for children with epileptic seizures, including general practitioners and community nurses, paediatricians and paediatric neurologists and clinical neurophysiologists, for the following reasons. Firstly, it is common. It probably affects about 13% of children of 3-6 years old with one or more non-febrile seizures (peak age 4-5 years), and 6% of the age group 1-15.^{4,5} Secondly, seizures can be prolonged, may mimic non-epileptic disorders, and may vary in severity from trivial to apparently life threatening—implying that the diagnosis may need to be considered in a variety of clinical settings and by medical professionals of different specialties. Thirdly, it is benign—its recognition therefore can provide firm reassurance to families in situations that can be very alarming. Finally, clinical research, necessary in any new syndrome, would require a multidisciplinary approach.

Panayiotopoulos syndrome can be best defined as idiopathic susceptibility to early onset benign childhood seizures with electroencephalographic occipital or extra occipital spikes, and manifests mainly with autonomic seizures.⁴ Large independent studies have accumulated impressively concordant information on the clinical and electroencephalographic features of this syndrome.^{4,9}

Rolandic epilepsy, another common syndrome, affects 15% of children with seizures and has as good a prognosis as febrile convulsions. Seizures usually start between 7-9 years of age, occur mainly during sleep and consist of hemifacial convulsions, speech arrest, oropharyngolaryngeal movements, and hypersalivation. EEG shows centroyral spikes. Gastaut's occipital epilepsy is much less frequent, manifests with mainly

visual seizures, and has less predictable outcome. Electroencephalography shows occipital spikes.²

Autonomic seizures are the hallmark of the Panayiotopoulos syndrome.⁴⁻⁹ Autonomic symptoms and signs (mainly vomiting) occur from the onset in 80% of seizures, with half of them lasting for more than 30 minutes to hours, thus amounting to autonomic status epilepticus. Two thirds of the seizures occur during nocturnal sleep or brief daytime naps. In a typical daytime seizure the child looks pale, complains, "I want to be sick," and vomits. If in sleep, the child wakes up with similar complaints or is found vomiting, confused, or unresponsive. Vomiting occurs in three quarters of seizures. Other autonomic manifestations may occur either concurrently with vomiting or later in the course of the seizure, and include pallor, mydriasis, cardiorespiratory, gastrointestinal and thermoregulatory alterations, incontinence, and hypersalivation. In at least a fifth of the seizures the child becomes unresponsive, pale, and flaccid (ictal syncope)⁴ either before convulsing or in isolation.

Behavioural disturbances, headache, or various non-painful cephalic sensations are common particularly at onset. More conventional manifestations of seizures often ensue: the child becomes confused or unresponsive, eyes may deviate to one side (in 60%) or the patient may stare. Half of the seizures end with hemi or generalised convulsions. Other, less frequent ictal features include speech arrest, hemifacial spasms, visual hallucinations and oropharyngolaryngeal movements, suggesting a maturation related continuum with Rolandic epilepsy.¹⁰

Diagnosis of Panayiotopoulos syndrome may be easily missed—mild and brief ictal autonomic symptoms in the presence of clear consciousness would suggest trivial non-epileptic conditions such as atypical migraine, gastroenteritis or syncope, while prolonged and severe attacks may simulate life threatening insults such as encephalitis, for which many of these children are treated.^{3,4,9}

Characteristically, even after the most severe seizures and status, the child is normal after a few hours of sleep—this is both reassuring and diagnostic. Electroencephalography, which should be done after a first non-febrile seizure, is confirmatory. This usually shows multifocal spikes at various locations.^{3-5,7,11}